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PLATE I

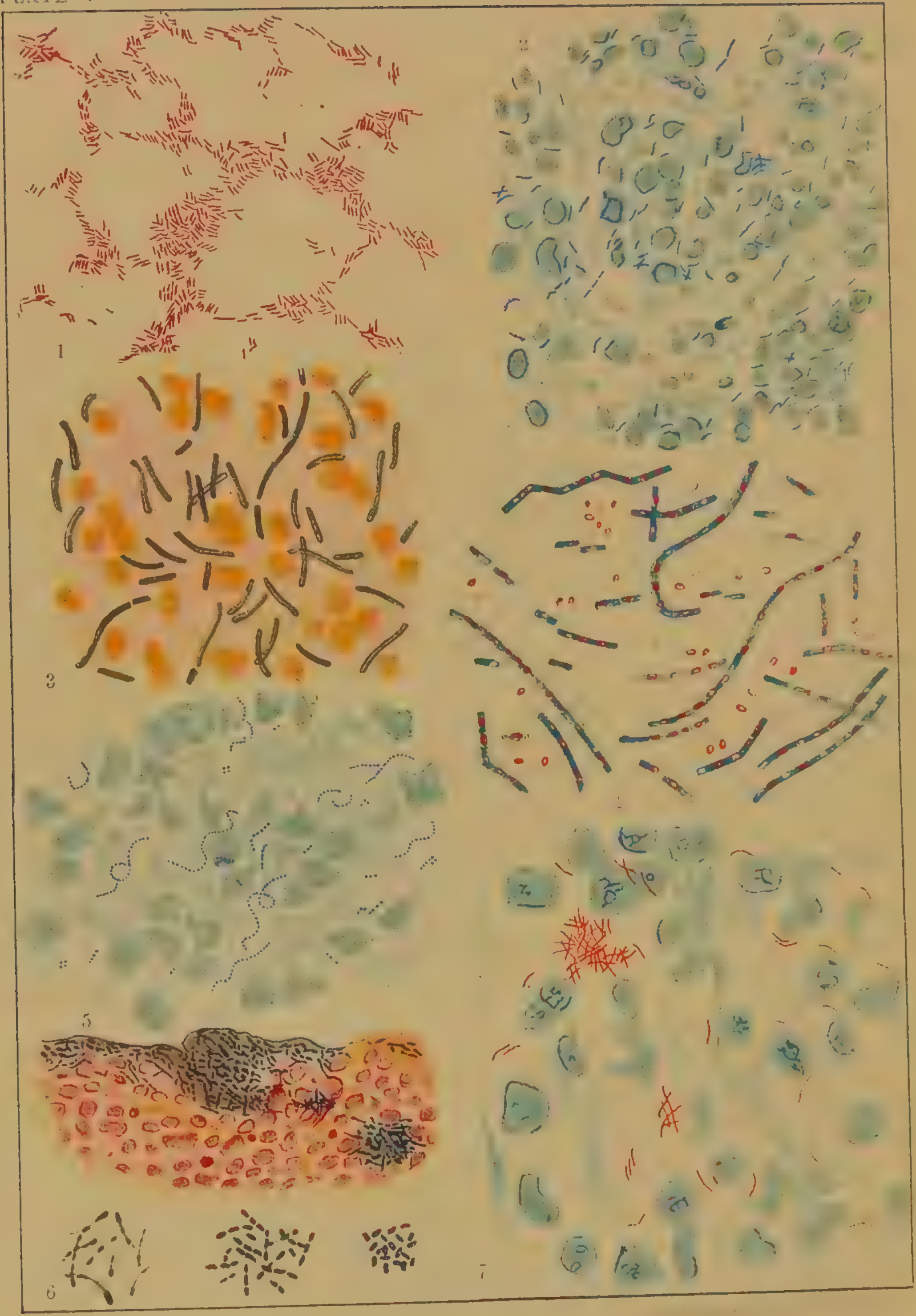

PLATE I.

Fig. 1.—Drawing of Bacillus Mallei in Coverglass Preparation of Pure Culture. Stained with Fuchsine. Magnified about 700 diam.

Fig. 2.—Drawing of Section of young Glanders Nodule in the Lung. Stained by Loeffler's method to bring out the Bacillus Mallei. Magnified 700.

Fig. 3.—Bacillus Anthracis from Spleen of Cow. No spores are to be seen, as the preparation was made as soon as the animal was killed. Magnified 700.

Fig. 4.—Anthrax Bacilli from Pure Culture. Stained by Möller's method. Spore formation well seen. Magnified 700.

Fig. 5.—Streptococcus Pyogenes in the Pus, taken from an acute abscess of the thigh. Stained with Methylene Blue. Magnified 700.

Fig. 6.—Diphtheria Bacilli at and near the surface of a false membrane. Below this are bacilli (*a*) grown on glycerine agar-agar, (*b*) on blood serum, (*c*) as seen on surface of membrane. Magnified 700.

Fig. 7.—Tubercle Bacilli in Coverglass Preparation made from a Tubercular Gland. Stained with Carbol-Fuchsine and Methylene Blue. (Ziehl-Neelsen method.) Magnified 700.

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BY

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Vol. I.

WITH TWO COLOURED PLATES AND 463 ILLUSTRATIONS

SEVENTH THOUSAND

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PREFACE.

The object of this work is to present concisely and with authority an account of the Science of Surgery as it exists at the present day. To deal successfully with a Protean phase of knowledge, which is encumbered by many ancient prejudices and which has been, within recent years, the subject of aggressive activity and the scene of almost revolutionary changes, is a matter of no little difficulty. The names of the Authors who have compiled these volumes will, I believe, afford an assurance that this difficulty has been approached with at least competent equipment, and for their invaluable co-operation it is impossible to express adequate thanks.

There has been no attempt to make the work encyclopædic; but every endeavour has been made to render it useful to the Student and Practitioner of Surgery. Those Subjects which are of the greater importance, are those which have been treated with the greater fulness.

The articles are concerned with the Pathology, Clinical Manifestations and Treatment of the various diseases and injuries that come within the scope of Surgery. The actual details involved in operative procedures are not dwelt upon. The specialisation of Ophthalmic Surgery made it appear desirable to exclude that subject from the present "System."

In a few instances the same subject—or some one aspect of it—is treated by more than one writer, and not always with unanimity of opinion. As the creed of the surgeon is not yet finally formulated, no effort has been made to efface these divergent expressions, or to secure an ideal uniformity.

The woodcuts illustrating the various articles are, with few exceptions, original, and have been for the most part taken from living patients, from photographs, and from actual specimens. The majority of these illustrations have been executed by Mr. Charles Berjeau, to whose care and skill the work is very greatly indebted. In the instances in which figures have been borrowed from other works, the source is in every case acknowledged.

That a book dealing with many subjects, and the work of many hands, must present some omissions is—in a first edition at least—inevitable, but it is hoped that these omissions are not such as to illustrate the statement of Carlyle that "the true value of a man's book is determined by what he does not write."

FREDERICK TREVES.

6, *Wimpole Street, London,*
June, 1895.

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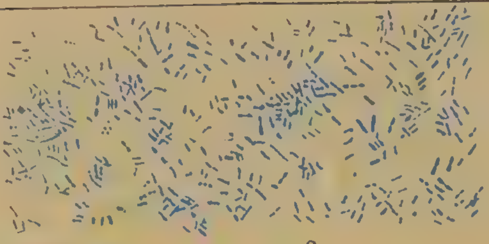
PLATE II.



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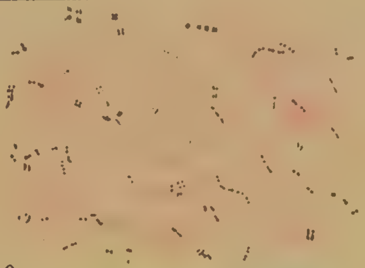

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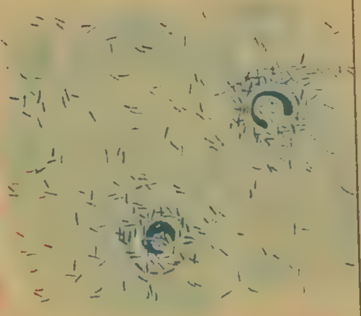

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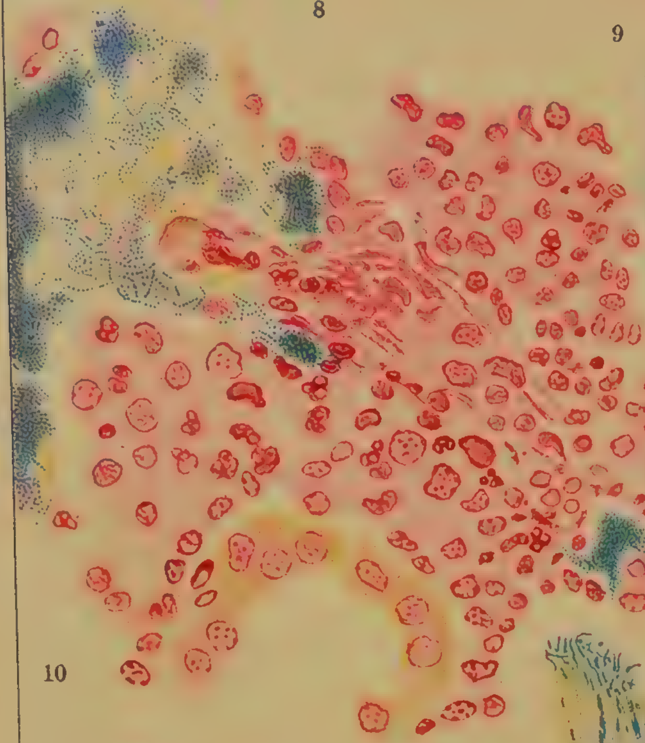

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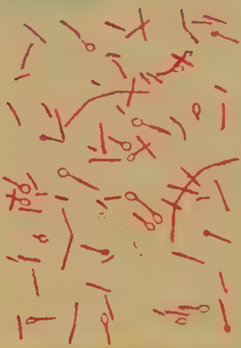

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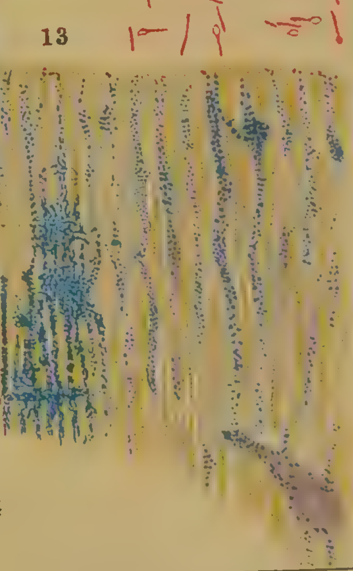

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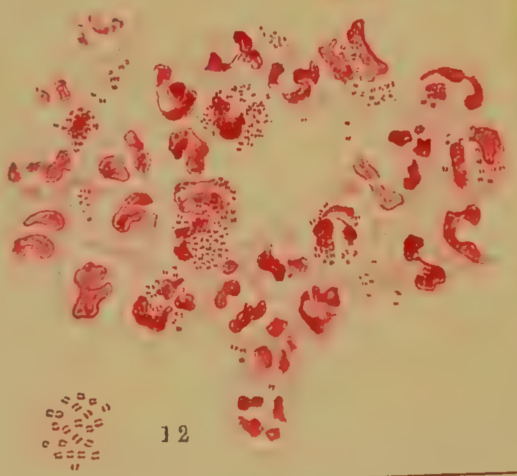

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PLATE II.

Fig. 8.—Coverglass Preparation of Pure Culture of Bacterium Coli Commune (Bacillus Coli Communis). Stained with Methylene Blue. Magnified 700.

Fig. 9.—Coverglass Preparation of Blood containing Diplococcus Pneumoniæ. Stained by Gram's method. Magnified 700.

Fig. 10.—Margin of Abscess in a Bone. In this may be seen numerous cocci in groups or masses and in chains (Staphylococcus and Streptococcus osteomyelitis). Stained by Gram's method. Magnified 700.

Fig. 11.—Bacillus of Mouse Septicæmia in Coverglass Preparation of Blood. Stained with Methylene Blue. Magnified 700.

Fig. 12.—Gonococci in Pus from Case of Acute Gonorrhœa. Note the arrangement of the diplococci in and between the cells. Magnified 700.

Fig. 13.—Tetanus Bacillus in Coverglass Preparation of a pure culture. Stained with Fuchsine. Note the shape of the rods and the size and position of the spores. Magnified 700.

Fig. 14.—Section of Carious Tooth to show Position of Micrococci and Bacilli. Stained with Methylene Blue. Magnified 700.

[Figs. 6, 9, 10, and 14 are drawn from Preparations kindly placed at the author's disposal by Mr. H. G. Plimmer, M.R.C.S., F.L.S.; and Fig. 12 is drawn from a Specimen prepared by F. J. McCann. M.D.]

SYSTEM OF SURGERY.

I. SURGICAL BACTERIOLOGY.

By GERMAN SIMS WOODHEAD, M.D.,

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GENERAL CHARACTERS OF BACTERIA.

Before giving a description of those bacteria that bear a definite relation to surgical diseases, it will be necessary to describe very briefly the general characters of this whole group of micro-organisms which it is now generally agreed are closely allied to lower vegetable forms of life, certain of the more highly organised forms with which we have to deal being classified in well-recognised groups of plants.

The term *bacteria* (Gr. *βακτηρία*; *βάκτρον*, *dim.* *βακτήριον*, a staff), though now well understood and generally recognised as including rounded organisms or cocci, is evidently a misnomer. Bacteria form a group, the members of which have a vegetative growth; they multiply by a process of fission or direct division, each organism as it increases in size dividing into two—hence the name *fission fungi*, *schizomycetes*, or *schizophytes*. In some cases this process of division is complete, the rod or the coccus (Gr. *κόκκος*, a berry) simply dividing into two separate organisms; in other cases the two organisms may remain in contact, and even with a slight bond of union between them, forming what is called a *diplococcus*. The diplococcus may undergo still further division, and if the individual organisms still remain attached, and division is in one plane only, transverse to the longitudinal axis, a chain or *streptococcus* (Gr. *στρεπτός*, a collar; *στρεπτά*, a necklace) may be formed. A *strepto-bacillus* or *streptothrix* is formed in exactly the same way. Where there are several planes of division, so that an irregular or mulberry-shaped mass is formed, a *staphylococcus* (Gr. *σταφυλή*, a bunch of grapes) is the result. Where the rods increase considerably in length, with or without marked subdivision into shorter lengths, the *leptothrix* (Gr. *λεπτός*, slender, thin) is formed. In addition to these more common forms we have spiral or screw-shaped organisms, the long chains of which are known as *spirilla*, these dividing into shorter lengths to form the *comma-shaped* organisms such as are met with in cholera.

1. Classification.—For our purpose the simple classification into micrococci (spheres or granules), bacilli (rods or staves, Lat. *baculus*), and spirilla (screw-shaped organisms or spirals) is sufficient. This does not, of course, take into account the method of development, division, and life-history, but serves merely for purposes of recognition and description in surgical work, for which it is at present amply sufficient.

The micrococci may be classified as diplococci; streptococci (already described); tetrads—in which there are two planes of division at right angles to each other, little groups of four being formed; sarcina or packet or wool-sack cocci—the result of division in three planes, in which the groups are composed of at least eight cells; and ascococci (Gr. ἀσκός, a wine-skin, a leathern bag), a term now not much used, in which groups of cocci are contained within a definite capsule.

The straight and elliptical bacteria are termed bacilli.

2. Structure.—These organisms are made up of a delicate protoplasm or vegetable albumen which takes the stains of aniline dyes so deeply that there is supposed to be interspersed throughout a considerable quantity of nuclear chromatin (page 51). There are, however, more deeply-stained bodies scattered throughout the protoplasm which are probably composed of nuclear substance, though this has not yet been satisfactorily proved. It has been suggested that the rapid vegetative division of the cell is due to the presence of a large amount of this nuclear substance. Unstained, the fungus protein, or myco-protein, as this vegetable albumen was named by Nencki, is colourless or faintly yellow, homogeneous or glassy-looking, granular or vacuolated. Minute granules of chlorophyll, strongly refractile particles which give the reactions of fat, and small granules of sulphur or starch are sometimes seen. In certain cases this protoplasm may assume a yellow, red, violet, magenta, or green colour. Enclosing the protoplasm is a dense resistant sheath or protecting membrane, which resembles the hard covering of the vegetable cells of the higher plants and contains cellulose. Between this dense membrane and the deeply-stained protoplasm a narrow unstained area may be seen; this is, by some, said to be produced artificially by the action of the various reagents used in preparing and staining the organism, whilst others hold that it is a kind of modified protoplasm from which the deeply-staining nuclear material is absent. The inner layers of the outer membrane usually remain firm, but the outer layers may become so modified that they form a gelatinous covering by which, in many cases, the organisms are bound together into little lumps or masses like frogs' spawn, known as *zooglœa masses* or masses of living glue. The colouring matter, so characteristic a feature of many micro-organisms, may be found both in this membrane and in the protoplasm, whilst in other cases it is present in the membrane only and not in the protoplasmic substance. This colouring matter, present in such micro-organisms as the Bacillus pyocyaneus and in the bacillus of blue milk, may be diffused into the surrounding nutrient medium. The presence of this colouring matter in any of the above positions, when grown in sufficiently large quantities to become visible to the naked eye, gives rise to those characteristic beautifully-coloured masses seen in the growths of Bacillus violaceus, (Fig. 1) the magenta micrococcus (both found in water), the Bacillus prodigiosus, and other similar organisms. The limiting or external membrane may be merely altered myco-protein, but in certain bacteria, the Bacillus anthracis for example, it consists of a substance resembling casein, combined with a form of mucin found especially in the embryonic tissues of animals.

Many micro-organisms, especially those which are motile, appear to be provided with *flagella* or *cilia*, developed either directly from the protoplasm of the organism or as a secondary modification of the external membrane. These flagella may be met with singly at the ends of the organism, or in pairs in a similar position, or there may be several pairs arranged at the periphery. They stain (*see* Loeffler's stain for flagella, page 51) like the membranes, and are found especially in those organisms that grow in fluid media and in contact with oxygen (page 8).

Although those bacteria of interest to the surgeon are usually described as single-form growths, it must be remembered that under certain conditions, as pointed out by Zopf, some of them may be poly- or pleo-morphic during certain stages of development. The Beggiatoa, for example, is said to be pleomorphic because it assumes at one time or another the several forms of cocci, rods, and leptothrix filaments, whilst the Actinomyces or Ray-fungus is so termed from the fact that in the lower animals it is made up of a series of club-shaped rays with the rounded ends of the clubs arranged at the periphery of a star. In man, and in young colonies even in the lower animals, it does not exhibit this supposed characteristic arrangement. In place of, or sometimes accompanying the ray-form, may be found cocci, usually arranged in chains or masses; these may be accompanied by leptothrix filaments, which, interlacing freely, form a kind of felted network. These threads vary considerably in length and are sometimes divided; or they may show no sign of division of any kind. Some of the threads are curved, and when they break they form short curved sections almost like spirilla or comma-shaped organisms; the cocci and threads are said to be the active fungus

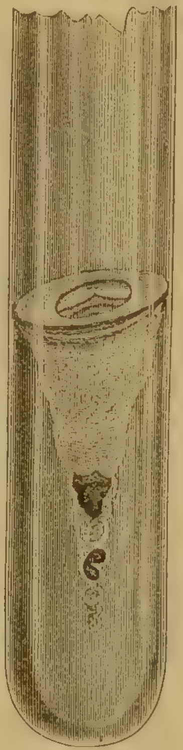

Fig. 1.—Cultivation of Bacillus Violaceus. The dark masses are violet-coloured.

growths, the club forms being merely involution forms, the clubbed extremity being the result of an extraordinary development of the membranous sheath which, though marked when cattle are affected, is comparatively rare in the human subject.

3. **Methods of reproduction.** **Fission.**—Micro-organisms divide very rapidly, the cholera vibrio, for example, under favourable conditions divides into two, once in every twenty minutes; others divide as slowly as once in an hour. Were conditions ideally favourable for the continuance of this growth, all other organic life would therefore soon be crowded out of existence. This vegetative multiplication, however, only takes place when all the conditions for the nutrition of the micro-organism are extremely favourable.

Spores.—If nutrition or the removal of excretory products is retarded, or if there is an excess of oxygen—any of which may interfere with vegetative multiplication or fission—well-marked changes take place in certain bacteria. The protoplasm in the anthrax bacillus (Plate I. Fig. 4), or the Bacillus subtilis (in both of which spore formation is a very marked feature) becomes granular, and a small bright point appears in each bacterium; this point gradually increases in size until its diameter may be greater than that of the original organism. It is then large, strongly refractile, round or ovoid, and is known as a *spore endospore*, or *resting spore* (see Möller's method of staining spores, page 51), which usually consists of two distinct portions—a dark limiting membrane called the exosporium, and the colourless highly refractile speck of protoplasm called the endosporium. It was at one time supposed that the endosporium did not take on staining material readily, but it is now found that the spore does not stain, simply because of the resistance offered by the outer limiting membrane to the passage of colouring matter; so deeply may the endosporium be stained that it is now supposed that it contains a considerable quantity of nuclear substance. The dark outline is usually surrounded by a delicate gelatinous envelope especially well marked near the two poles of the refractile spore. When spore formation is well advanced, degeneration of the protoplasm of the bacteria in which spores are found invariably follows; where the spores are large they readily escape from the degenerating bacillus, but where they are small they may lie for some time embedded in the protoplasm of the cell, which, only as it degenerates or dies, leaves the resting spore free to be carried about by currents of air or water, to be developed when the conditions as regards moisture, temperature and food, again become sufficiently favourable. In the anthrax bacillus (Plate I. Fig. 4) the spore seldom exceeds the diameter of the rod in which it is developed; in other cases, when the diameter of the spore exceeds that of the bacterium, it gives rise to the formation of a spindle-shaped organism when situated in the centre, or to a drum-stick- or pendulum-shaped organism when situated at the end, as in the case of the Bacillus tetani (Plate II. Fig. 13). These spores are important because they are much more resistant to unfavourable conditions and to bactericidal agents than are the vegetative organisms. Most of the vegetative organisms when desiccated rapidly lose their vitality—the cholera bacillus, the typhoid bacillus, and others sometimes succumbing in as short a period as twenty-four hours. Spores, on the other hand, may be dried, treated with chemical reagents, if not too strong, and if, after all, they are again placed in favourable conditions, they develop into this vegetative form, the spore germinating out like a seed, the endosporium making its way through the membrane which becomes ruptured either at the end or the side, as the case may be.

The importance of the study of the conditions under which these spores may be produced is well seen on considering the method of formation of spores by the anthrax bacillus when it has attacked an ox. During the life of the animal, whilst the bacillus is confined to the blood and lymph spaces in the tissues, it will be found that not a single spore is developed, and if the animal be buried deep down, with an unbroken skin, and before the discharges from the nostrils and other orifices of the body have been allowed to come in contact with the air for any length of time, no spores will be developed, and the anthrax bacillus will die out as the carcase becomes decomposed. Should the animal be left exposed for any length of time, however, wounds being made in the skin and discharges flowing from the various orifices, the bacilli as they come into the presence of air soon form spores which resist the action even of putrefactive organisms, and may remain a source of infection on a farm for some time after the animal has been buried. Splashes of blood, or of discharges, all represent foci from which the spores may spread, and gain access to the manure heaps or other organic matter so plentiful about farms and at the margins of ponds, where they in turn may form dangerous infective centres. Similarly the spores of the organisms found in tetanus and malignant œdema are a constant source of danger in any soil to which they have once gained access. Spores seem to be very susceptible to the action of the actinic rays of light.

A second form of spore described, but not yet thoroughly understood, is the *arthrospore*, which consists rather of a modified individual in a group of organisms than of a separate structure in an individual organism. For instance, it is held that a single element in a chain of cocci may become so modified as to be more resistant than its fellows, remaining alive after the others have been destroyed, resisting the action of the various unfavourable surroundings, and then, when again placed in suitable conditions, developing, multiplying, and keeping up the species.

4. Culture diagnosis.—At first sight it would appear to be hardly necessary to draw attention to the appearance of colonies of micro-organisms that have grown to such a size that they may be readily distinguished with the naked eye. But the distinct morphological characters of micro-organisms are so few, that in order to classify bacteria we must have recourse to every fact that has yet been observed, not only as to their structure and functions, but also as to their growth *en masse.* The functions may vary very greatly even in the case of micro-organisms which are apparently morphologically identical, whilst similar functions are sometimes observed in organisms which under the microscope present very marked and characteristic differences.

The most prominent of all the naked-eye features is colour; the Bacillus prodigiosus produces the well-known blood-red appearance which gives rise to what is known as "bloody sweat"; the red bacillus from the harbour water of Kiel has also a characteristic colour; whilst the magenta micrococcus, the Bacillus violaceus, the Bacillus pyocyaneus, the bacillus of blue pus, and the blue milk bacillus, may be instanced as bacteria in which colour formation is a most characteristic feature, often enabling us to distinguish one micro-organism from others which have very similar or even identical microscopic appearances.

Some of these colour-producing organisms, and a number of others, have an additional function, that of digesting, peptonising, and *liquefying a gelatine medium* (Fig. 1) in which they are growing; others, however, do not develop this power of digestion, so that here again is a diagnostic feature which facilitates the classification of certain groups of micro-organisms. This power of peptonisation is a feature of very great importance in the series of pathogenic germs, as it is found that a large number of the parasitic infective bacteria possess it in a most remarkable degree; in fact, in some cases the degree of peptonisation appears to run concurrently with the pathogenic activity of the micro-organism, though there are undoubtedly exceptions to this rule. Two very prominent examples, the anthrax bacillus and the Staphylococcus aureus, may be taken. The virulent anthrax bacillus sets up a rapid liquefaction of the gelatine; this begins at the surface (Fig. 2) and becomes less and less as the surface is left. The Staphylococcus aureus, or, as it was first described by Becker, the staphylococcus of osteomyelitis, when active, also sets up extreme liquefaction, the gelatine becoming slightly turbid and the organism falling down through the liquefied gelatine to form an orange-coloured mass at the bottom of a kind of funnel. Other examples will be found in the descriptions that follow.

Much information may be gathered as to the functions of bacteria, by growing them on milk. The proteids in the milk may be simply precipitated as the result of an acid fermentation, the casein being thrown down in larger or smaller flakes in different cases. Again, the casein may be first precipitated and then digested or peptonised by the action of enzymes, or the enzyme may come into play at once and bring about peptonisation of the casein, without the intervention of any preliminary coagulation. Then, too, the physical characters of the milk—its appearance, taste, and smell, and its chemical reactions—may indicate the formation of different colouring matters, various acids, alcohols, ethers, and other volatile substances, some of which have already been fairly thoroughly worked out, whilst much still remains to be done at others.

Certain organisms, especially those which may be grown anaërobically, have the power of *producing gas*, which, collecting around the organisms in the deeper layers of the nutrient media, appears as small bubbles. This formation of gas is sometimes a point of considerable diagnostic value, as, for example, in distinguishing certain organisms which, though morphologically resembling the tetanus bacillus, have the power of developing gas, a function which does not belong to the tetanus bacillus, so that if gas bubbles are formed in any culture in which the tetanus bacillus is present, it is not a pure culture of that organism.

Certain organisms have also the power of producing a *phosphorescent glow*, especially when they are supplied with special food materials, the energy which they develop from this food material assuming the form of light.

5. Production of toxines.—From the point of view of the pathologist, however, the most important products of these organisms are what may be spoken of as *toxines* and toxic albumoses, peptones, alkaloidal bases, and poisonous or poison-forming acids. At the same time the whole of the ordinary decomposition products, beginning with the ptomaines, leucine, tyrosine, and the aromatic and organic acids, and ending with ammonia, nitrites, nitrates, and ultimately carbon dioxide, hydrogen and nitrogen, are formed. Many micro-organisms have the power of forming hydrolytic ferments, amongst which may be mentioned those having the properties of diastase, of invertine, pepsine, tripsine, by which the ordinary liquefaction of gelatine is brought about, and of rennet. We thus see that the vital manifestations of these organisms are exceedingly complex, and that each organism or group of organisms requires to be separately studied in order that its morphology and functions may be accurately defined.

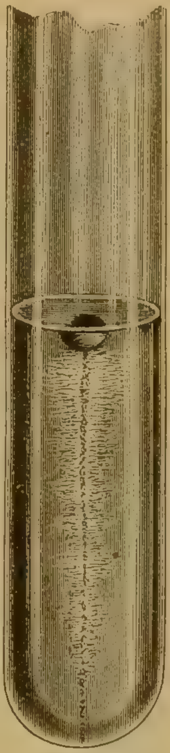
Fig. 2.—Cultivation of Bacillus of Anthrax.

6. Conditions of bacterial life.—In order that bacteria may grow and multiply and give rise to the products above mentioned, it is necessary that they should have suitable food, some of them requiring proteid or albuminous substances for their nutrition, others living on carbohydrates. They all require water, carbon, nitrogen, oxygen, hydrogen, and certain inorganic salts, the most important of which appear to be those of lime and potassium. They differ very greatly as regards their power of obtaining the materials they require from these various substances: they are unlike plants in so far as they are unable to assimilate carbon dioxide. Some are able to obtain their carbon and oxygen from starch and sugars, from certain organic acids, and from the glycerine group, whilst others seem to have the power of wresting the food materials only directly from proteid substances. In all cases the hydrogen and oxygen are separated partly from the breaking-down food molecules, partly from water and partly from the air. Certain organisms seem to have the power of taking up their nitrogen from the atmosphere, though this power is apparently somewhat restricted in its distribution. It may be stated generally, that the more parasitic the organism the less capable is it of taking up food from carbohydrates or sugars.

It may be well at this stage to point out that certain bacteria grow best when they are able to obtain a free supply of oxygen from the air, without which they are unable to carry on their full peptonising, enzyme-forming, or colour-forming function. Such organisms are described as *aërobic;* when cultivated on gelatine they always grow most vigorously near the surface; the peptonised area, when present, is usually funnel-shaped (*see* Fig. 1), and the formation of pigment goes on principally near the surface or in that part of the medium in which the oxygen has become mechanically entangled. Some bacteria, which are known as obligate aërobes, can grow only in the presence of oxygen; others again, which are spoken of as facultative aërobes, may become so modified that they can wrest their oxygen from the nutrient medium in which they are growing. It should be noticed that wherever this occurs, unless the growth of the organism is exceedingly feeble, the breaking-down of food material and the production of secondary products are much more marked than when the organisms have free access to oxygen.

Other organisms, as for example the tetanus bacillus, can grow and produce their special products only when air is rigidly excluded (Fig. 3); even when such organisms do grow feebly in the presence of air, it is found that they do not give rise to the special products by which their presence is characterised in the non-aërobic growths. They are termed *anaërobic.* Such organisms appear to be capable of developing their special functions only when they draw the elements of their food from complex nitrogenous food or carbohydrates, of which there is always a far greater disintegration, accompanied by the production of greater quantities of specific and aromatic substances, than in the case of those organisms which obtain their oxygen from the air. The tetanus bacillus and the bacillus of malignant œdema are perhaps the two most typical anaërobic organisms with which the surgeon has to deal.

Most of the organisms of interest to the surgeon are *parasitic,* living on or in the higher animal body. A few, such as those producing leprosy and relapsing fever, have never yet been cultivated outside the host, so that they must be spoken of as strictly *obligate parasites.* The conditions of growth of the tubercle bacillus outside the animal body are also so restricted that it practically comes under the same heading, although it is possible that under certain conditions it might grow outside the body. *Facultative parasites* are those which, though usually living outside the body, are capable under certain conditions of taking up their lodging in an animal host and of there, usually, giving rise to definite pathogenic conditions.

A *saprophytic organism* is one which is capable of growing on dead organic matter, *i.e.* outside the animal body. The terms obligate and facultative are applied to saprophytes in very much the same way as they are applied to parasites, obligate saprophytes being unable to exist as parasitic organisms, and the facultative saprophyte being a parasitic organism which can continue its growth and multiplication on dead organic matter—anthrax tetanus and malignant œdema bacilli may all, under certain conditions, become facultative parasites and alternately *facultative saprophytes.*

It is important for the surgeon to remember that most organisms are saprophytic, and that even those that now exert a pathogenic influence are apparently derived from a saprophytic stock. Indeed, the conditions favourable to the growth of saprophytic organisms are so widespread and the part they play in the process of putrefaction and conversion of dead material for the use of plants is so definite, whilst the agencies inimical to the vitality of the pathogenic organisms are so numerous and effective, that the keeping down of the number of disease-producing germs should be a comparatively easy matter. The obligate parasites are confined to certain regions where the conditions are such that they can be passed on directly from individual to individual, but the facultative parasites, which are able to assume a saprophytic existence—take the anthrax bacillus, for instance, which can flourish on the refuse of a farmyard—are much more dangerous organisms. In the same way those organisms which, through the formation of spores, are capable of continuing their species under unfavourable conditions are more difficult to get rid of than the more easily killed non-spore-bearing micro-organisms, such as the cholera bacillus.

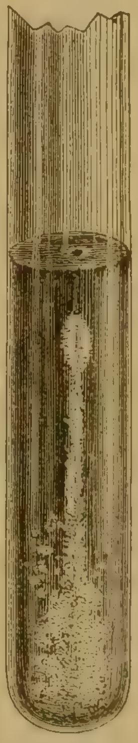

Fig. 3.—Cultivation of Bacillus of Tetanus.

7. Temperature conditions.—The majority of the pathogenic micro-organisms develop most readily and luxuriantly at the temperature of the human body. Some will grow at the temperature of the room—15° or 16° C., but any temperature below 30° C. or above 42° C. usually leads to a diminution of their virulence or of their vital activity. It is scarcely necessary to consider the temperatures (outside the limits) at which organisms retain their vitality or are killed off; but speaking generally, it may be stated that most bacteria cease to grow at a temperature of 5° to 10° C. on the one hand and above 45° to 50° C. on the other, though certain species have been found which are capable of exhibiting vegetative and functional activity even beyond the limit (65° to 70° C.).

8. Distribution and habitat.—Bacteria are so widely distributed that it may be well to consider, briefly, the positions in which they occur, and from what points surgical wounds are usually attacked. It was for long supposed that most of the organisms setting up suppuration, hospital gangrene, and similar conditions, were carried by *air currents*, but it is now generally accepted that comparatively few organisms, and only those that can survive the entire absence of moisture and are adherent to particles of dust, can be carried in this manner. Most of the pathogenic and pyogenic organisms are incapable of continuing their existence in the absence of *moisture*, so that the danger of infection through the air is comparatively slight, though it is one that cannot be neglected, especially where large numbers of organisms are being thrown off from a dry warm surface, or are present in a heated atmosphere where currents are numerous and strong. There are, as a rule, comparatively few pathogenic organisms found floating in the atmosphere except when *dust*, to which groups of micro-organisms will be found to adhere, is being blown about. Pyogenic organisms, however, have been repeatedly found in the air of inhabited rooms, but this is explicable by the fact that these organisms are so frequently met with even on the *surface of healthy bodies.* In some experiments carried on by Dr. Cartwright Wood and myself, we were able to prove that tubercle bacilli were present in considerable numbers in the air of a room in which were a number of tubercular animals. Similar observations have been made by other observers.

The *surface of the soil*, especially that holding a large quantity of organic matter, contains large numbers of micro-organisms, most of which are instrumental in carrying on the processes of putrefaction and in breaking-down highly organised matter into its simpler constituent elements. There may be an entire absence of free micro-organisms from the *subsoil* at a greater depth than five or six feet, except where they have been washed down by water percolating rapidly through the upper layers. Most of these micro-organisms are saprophytic in character, but as already pointed out, the anthrax bacillus may be a source of danger for some time after it has found its way on to surface soil; whilst the bacillus of tetanus and that of malignant œdema, which are very frequently present in cultivated soil, are factors in a danger which cannot be ignored by the surgeon.

Water contains a very large number of bacteria, some washed from the surface of the soil, whilst others, the so-called water organisms, can be found only in water and are most of them non-pathogenic. These water organisms flourish at a comparatively low temperature, but some are not at all easily cultivated artificially except under conditions similar to those maintained in streams. To the surgeon, the presence of water organisms is of comparatively little importance, as even the most resistant of them may be killed if the water be boiled for a few minutes.

The free surfaces and cavities of the human body are all maintained moist and at a temperature suitable for the growth of micro-organisms; all the conditions for the deposition of bacteria on and in these surfaces and cavities are of the most favourable character. The moist *skin* is exposed to the action of currents of air which carry micro-organisms; the hands are constantly coming in contact with all kinds of objects on which bacteria accumulate, whence they may be readily distributed over the surface of the body. The ducts and glands of the skin are all filled with secretions in which bacteria may thrive. The nose and the mouth, with their moist walls and filtering surfaces, keep back an enormous number of micro-organisms taken in with the air-borne dust which we are constantly inhaling; whilst the alimentary canal is frequently, though more intermittently, invaded by numerous species of bacteria, many of which are ingested with our uncooked food. It thus comes about that so many of the organisms at present known have at one time or other been described as present in the mouth.

More than one hundred distinct species have been found on the surface of the skin by Welch, who has made a special study of the subject; he says "all sorts of bacteria are brought . . . into contact with the skin and exposed mucous surfaces, but most of them are entirely harmless, and are sooner or later removed. There are, however, certain bacteria which are found so constantly or with such frequency in these situations that we can properly speak of a definite bacterial flora of the skin, of the mouth, of the nose, of the intestine, and of the vagina. Thus, in the epidermis and the glandular appendages of the *skin* we find regularly a Staphylococcus epidermidis albus; in the *mouth*, leptothrix and spirillar forms which will not grow on our ordinary culture media; in the *intestine*, the Bacillus lactis aërogenes and the Bacillus coli communis; and in the normal acid secretion of the *vagina*, the Bacillus vaginalis. Other species are usually mingled with these regular inhabitants, among the most common and important intruders being the pyogenic cocci, and in the mouth also the Micrococcus lanceolatus. When the vagina contains pyogenic cocci, its secretion is generally altered in reaction and appearance. These ordinary surface bacteria cannot in health, when in contact with the skin and mucous membrane, penetrate into the circulation and the internal organs, or, if now and then they do happen to make their way into the circulation, they are in health quickly destroyed. The internal organs and fluids are normally entirely free from micro-organisms." Some of these bacteria, though usually harmless, may under certain conditions give rise to serious lesions, whilst others, pathogenic in every sense, may be said to be lying in wait to effect an entrance through an abrasion or wound intentionally made.

It must be borne in mind that the skin and mucous membrane—moist rough surfaces—are not only good collecting grounds, but are also surfaces on which many micro-organisms can develop very readily. In fact, we find, in addition to ordinary non-pathogenic organisms, a considerable number of the pus-forming organisms constantly present on the surface of the skin, whilst in the oral and nasal cavities certain organisms seem to flourish, in spite of the fact that sputum exercises a certain deleterious influence on many of the pyogenic organisms. Those found in the nose are specially the pus-forming organisms and the bacillus of Friedländer, whilst under special conditions the bacillus of rhinoscleroma, the Bacillus fœtidus ozænæ and the bacillus of glanders have been met with, in addition to a considerable number of unimportant non-pathogenic organisms. The organisms found in the mouth may be said to include almost every one that can be carried in dust, and in addition to those specially described by dental surgeons, the various forms of pneumonia bacilli, pyogenic cocci, the Streptococcus articulorum, the bacillus of tuberculosis, and the bacillus of hæmorrhagic septicæmia have all been found.

It is interesting to notice that the mucous membrane of the bladder, the uterus, the Fallopian tubes, and the urethra are in healthy individuals entirely free from micro-organisms, although discharges, when they have reached the vagina, may be found to contain pus-forming organisms. In cases of puerperal septicæmia, these pus-forming organisms are comparatively frequently found in the discharge within the uterus.

9. Methods of invasion.—The method of invasion of the body by micro-organisms, and the conditions that are favourable and unfavourable to the development of pathogenic micro-organisms may be briefly indicated. It was first pointed out by Lister that even in the case of wounds that were apparently aseptic, small points of suppuration often made their appearance around the stitches of the suture, and it was for some time supposed that these small points of suppuration were due entirely to the tension set up by the presence of the *tight sutures*. Although this tension, by acting mechanically on the tissues, may, undoubtedly, have something to do with the production of pus, or rather with maintaining the conditions under which pus is formed, it has been demonstrated that the actual pyogenic factors are bacteria, which, present in the skin, are only able to develop and manifest their presence under certain conditions. The presence of the tight suture interferes greatly with the vitality of the tissues; the cells which normally keep the micro-organisms in check are no longer able to exert their inhibitory action, and the great obstacle to the growth of micro-organisms is removed. Beyond this, however, in the slight serous effusion which for some time contains comparatively few cells, the pyogenic micro-organisms are enabled to develop much more luxuriantly than in the less favourable excretory products found in the skin. The spaces between the strands of the suture absorbing nutrient fluids, are also favourable cultivation areas for pyogenic bacteria, which, enabled to develop rapidly and along with their products to get a start of the tissue cells, acting on these cells, set up localised suppuration, the surrounding comparatively healthy tissues ensuring localisation by keeping the bacteria in check.

Wounds of the skin, unless protected, are always liable to be invaded by saprophytic organisms, but it may be laid down as a general rule, that the less the injury to the tissues, and the more the effusion of blood and serum is checked, or, if such effusion does occur, the more carefully it is all removed, the less risk of bacterial invasion, and if bacteria do find their way into the wound, the more readily and completely are they dealt with by the tissues. Although a bruised wound heals readily enough when bacteria are carefully kept out, suppuration almost invariably occurs in a similar wound when bacteria gain access to the injured tissues ; in the one case there is a clean slough or a gradual absorption of the dead material ; in the other there comes away a putrid mass which may leave a suppurating surface. The ultimate result, as regards the lesion itself, may be the same, but the constitutional disturbance—the local pain and inflammation—is vastly greater in the one case than in the other. The surgeon who has had experience of wounds made with dirty infected knives or weapons, recognises only too well the dangerous results following the most insignificant lesions. A punctured dissecting wound, the slight lesion made with a dirty fish-bone in the mouth or in the hands, may be almost as dangerous as one made with a poisoned arrow ; whilst even in those cases in which no general disturbance follows, local suppuration almost inevitably ensues. The wound from a rusty nail, a fragment of broken glass bottle, or a dirty splinter of wood that has been lying on the ground, may suppurate owing to the presence of pyogenic micro-organisms that have remained alive on damp warm soil; or an attack of tetanus or malignant œdema, or even anthrax, may be set up, the particles of rust or the open texture of the wood offering not only a nidus for the organisms but spaces in which serum may accumulate for the nutrition of the contained organisms. The bruising of the tissues which usually occurs when wounds are produced by the above agents, is also a most important factor in the production of disease ; the cells which normally destroy bacteria are so far injured that they are unable to do their proper work. Dirty and blunt instruments, especially those that have been used in the treatment of septic cases, are just as deadly as rusty nails and dirty fish-bones. The coagulated blood from a septic case on a pair of bone forceps, or on a blunt knife, may be a focus of infection quite as dangerous as the carcase of a cow (dead from anthrax) left in an open field. Ordinary cleaning of such instruments is not sufficient to remove the coagulated blood, and even the somewhat prolonged action of such antiseptic agents as have a coagulating effect on albumen, is not sufficient to render the whole of such foreign matter aseptic, the micro-organisms contained in it, protected by the coagulated albumen, remaining alive and capable of doing their deadly work. In connection with all this it is now recognised that a sharp knife should be used in order to avoid unnecessary bruising of tissues, this quite apart from the question of rapidity of operation and the comfort of the operator ; that no soft tissues should be torn with forceps, which are necessarily blunt ; that scissors should be used as little as possible, and that the chisel should be used for bone, only after the periosteum has been carefully divided, and that wherever the chisel is used, bruising of the surrounding soft tissues should, as far as possible, be avoided. It would not be necessary to lay such stress on these points could we be absolutely certain of eliminating the action of bacteria in every surgical operation.

It is more difficult to clean instruments than was at one time supposed, and it is now recognised that if instruments are to be kept scrupulously clean, they should be washed first in *cold* water immediately after they have been used (a cold $\frac{3}{4}$ per cent. saline solution is probably the best for this purpose), then after the removal of the whole of the albuminous material (the fatty matter being removed by means of hot water) the instruments should be soaked in a 5 per cent. watery solution of carbolic acid, or, better still, subjected to moist heat in one of the numerous surgical sterilisers now in the market—that devised by Cathcart being one of the best for this purpose.

It has already been mentioned that special conditions appear to be necessary for the local development of pathogenic and pyogenic organisms; for instance, the diphtheria bacillus and the bacillus of pneumonia, the Bacillus lanceolatus and other bacilli, have been demonstrated in the mouth in perfectly healthy individuals, and it is only reasonable to suppose that these organisms do not produce their pathogenic effect simply because they can find no vulnerable point in the mucous membrane with which they are in contact. The protection afforded by the surface of the skin is so complete that the presence of pyogenic organisms on its surface is of constant occurrence in healthy individuals, without the slightest inconvenience resulting. Becker in his studies on the micrococcus of osteomyelitis, now known as the Staphylococcus pyogenes aureus, pointed out that even when injected into the circulation the micrococcus produced no appreciable result; when, however, the bones of the leg of a rabbit were crushed and fractured before the micrococcus was injected, the animal died in about a fortnight, presenting at the seat of the fracture the usual appearances of osteomyelitis (Plate II. Fig. 10), and the pus removed was found to contain enormous numbers of Staphylococcus pyogenes aureus. It is interesting to note, however, that more recent experimenters have succeeded in setting up a similar condition of osteomyelitis, even without first injuring the bone in any way. Periostitis too, in many cases a perfectly simple condition, is sometimes associated with the most virulent septic inflammation, leading to suppuration, separation of the periosteum, and, often, a general infection. The only explanation of these acute cases (which almost invariably only follow bruising of the periosteum) is that the organisms have been temporarily and accidentally present in the circulation before the bruising occurred, and that it has only been when the tissues have had their vitality lowered by injury that the bacteria have been able to exert their specific powers.

Bacteria are undoubtedly transported from point to point by the *blood* in the blood-vessels; of this we have evidence in the condition of septic endocarditis, where the bacteria must have come directly in contact with the surfaces of injured or roughened valves; whilst septic emboli are known to be of comparatively frequent occurrence. It is not always so easy to follow the spread of the more slowly-growing organisms by this path, though quite recently, considerable evidence has been brought forward in support of the theory that tubercle bacilli may make their way by blood-vessels to the ends of long bones, to which they can only have gained access by the large nutrient arteries of the shaft, or of the smaller nutrient vessels passing in at the ends to nourish the epiphyses.

The erysipelas organism may be taken as a type of the organisms which spread by the *lymphatic* spaces and vessels. Here they set up an acute, partially localised, but gradually spreading lymphangitis. The lymphangitis occurring in the early stages of poisoned wounds has come within the experience of most observers.

Most pathogenic micro-organisms appear to have the power of producing toxic substances, some of which, although they have a stimulating effect on the protoplasm of the cells, gradually cause exhaustion of their powers, so that ultimately these cells become depressed and incapable of resisting the attack of the bacteria. The changes produced in the protoplasm may be so profound that the cells undergo more or less complete degeneration. In the case of pyogenic organisms, and of the tubercle bacillus, the devitalisation of the cells is such a marked feature that the presence of toxic products was early suggested as the cause of the changes set up; but only comparatively recently has the importance of this factor been recognised in connection with such organisms as the tetanus bacillus. When this organism is inoculated alone, that is, as pure culture separated from its products, it appears to be incapable of producing any lesion, either local or general, the active tissue cells being able to destroy the organism before it has time to produce any of its poisons or to exert any markedly deleterious action on the tissues. If, however, even a small quantity of the poisonous products of the organism, or of certain non-pathogenic bacteria, or of such a substance as lactic acid be introduced along with the pathogenic organism, the tissues appear to be so paralysed or pre-occupied that they are no longer able to prevent the vital manifestations of the micro-organisms, which are left free to multiply and elaborate their poisonous products from the proteid nutrient material contained in the tissue fluids.

It has been stated that tetanus bacilli protected in threads, in splinters of wood, in collections of fluids, or in clots to which the tissue cells cannot gain direct access, are specially deadly; and this for two reasons, (1) they are well provided with nutriment and suitable temperature and moisture, and (2) consequently are able to produce large quantities of their products which act upon the surrounding tissues.

As regards the invasion of the various *cavities* and *channels* of the body, it must be recognised that distinct protective barriers are erected at various points, and that it is only when these barriers are removed that bacterial invasion can take place. For example, no bacteria are found in the healthy bladder; the external meatus of the urethra is so small, and the sides of this channel are so closely apposed when urine is not being expelled, that bacteria never make their way beyond the entrance. Then too, owing to the constant contraction of the bladder muscle, the current is always in one direction, so that unless bacteria be introduced by means of a dirty catheter or sound, invasion of the bladder by bacteria is of very rare occurrence. The restrictive action of the walls of the meatus and urethra is due to the direct action either of the cellular protoplasm, or of some secretion of the lining epithelial cells. The moist mucous membrane of the nostrils, of the mouth, and of the upper part of the pharynx is usually sufficient to catch and prevent the further passage of bacteria from the air into the respiratory channel, whilst the ciliated epithelium setting up currents away from the trachea, is constantly bringing about the return of solid particles to the upper part of the larynx. Other cavities appear to be protected by the action of the sphincters as well as by the secretions of the mucous membrane covering their walls.

10. Predisposition.—Although bacteria must now be looked upon as the prime causal agents in the production of certain surgical fevers, the fact is being daily more insisted upon that the old ideas regarding *racial, individual* and *tissue predisposition,* are of very great importance in relation to infection. The whole idea of protective inoculation and acquired immunity rests on the fact that these varying susceptibilities to various diseases are present in different species and under different conditions. Thus the tubercle bacillus finds a much more congenial soil for its growth and development in certain tissues, in certain people, and in certain species, than in others; the extent and duration of the infective process varying with the degree of susceptibility of the tissues or organism attacked. On the other hand, it has been noticed that the tubercle bacillus itself may become so modified, by being placed under different surroundings, that it may set up a modified form of tuberculosis. The importance of these two factors is very great, and should always be borne in mind when the question of infection is under consideration, as it is only by the careful consideration of many of the facts associated with these factors that the true meaning of bacterial infection in different surgical cases can be appreciated.

It is much more easy to trace the *local predisposing causes* in surgical than in medical diseases. In the former, of course, we have a local susceptibility wherever wounds or lesions of the tissues (such as inflammations, ulcers, death of a part, hæmorrhage, accumulation of fluids in serous or other cavities, and the presence of foreign bodies, especially those of a porous or absorbent nature) in which fluid, removed from the influence of the surrounding tissues, may accumulate; in this fluid, organisms carried in along with the foreign bodies, or following them, have a favourable nidus, in which they may multiply and develop their products, so gaining a firm foothold from which to attack the surrounding tissues. Even the diphtheria bacillus, which, for the present, we may look upon as connected with a surgical disease, is not usually able to invade perfectly healthy fauces and tonsils; if, however, these parts become slightly inflamed, and there is slight exudation or ulceration at any point, the bacilli are enabled to gain a foothold; they give rise to their special toxic products and acute inflammation accompanied by exudation of fibrinous lymph, rapid degeneration, and necrosis of the special tissues results. Then too, although the Bacillus diphtheriæ cannot exist either in the blood stream or in the lymph channels, except immediately around the lesion, the poison secreted by the diphtheria bacillus is transported to all parts of the body, and by its action may give rise to marked degeneration of the peripheral nerves, and sometimes of the muscles supplied by them, thus setting up the characteristic peripheral paralysis, whilst at the same time other organisms, taking advantage of the lowered vitality of the tissues and fluids at the seat of the disease, may take the opportunity of making their way by the various channels into the surrounding tissues, and even into distant organs.

One of the best examples of a localised predisposition in medical diseases is the septic ulcerative endocarditis, which results from the deposition of bacteria—Staphylococcus pyogenes aureus and Streptococcus pyogenes aureus—upon roughened or lacerated valves. The predisposition to osteomyelitis, when bones are fractured, before the Staphylococcus pyogenes aureus is injected into the blood system, has already been referred to. Another surgical example is found in an attack of bone tuberculosis, which almost invariably dates in the mind of the patient from a bruise or injury of the affected bone.

It cannot be too strongly insisted upon that everything which lowers the *general condition of the patient*, predisposes to infective surgical diseases—overcrowding, vitiated atmosphere, want of cleanliness, bad hygienic conditions, including bad and insufficient food and exposure to extremes of hot and cold, want of sleep, imperfect or irregular evacuation of the excretions, excitement or emotion—an influence all important to the surgeon now that many of these factors have actually been found by experiments on animals to predispose to certain forms of disease. Hæmorrhage, at one time so constantly associated with surgical operations, has been found to increase the susceptibility of animals to the attacks of the anthrax bacillus and of the micrococcus of osteomyelitis by the anæmia it produces. Glycosuria has such a marked influence in predisposing to tubercular and purulent infection, that most surgeons dread to have anything to do with diabetic patients. The same holds good in cases of albuminuria, although local changes in the kidney may, in part at any rate, account for the special vulnerability of that organ.

It would appear that in all these conditions there is some general factor at work, and Metchnikoff maintains that the factor to be specially taken into account is the *leucocyte*, in which, he contends, we have the first line of defence against the invasion of micro-organisms.

11. The mode of action of bacteria.—Since the more systematic examination of the products of bacteria has been undertaken, much light has been thrown on the question of how these bacteria act. Some investigators have gone so far as to assume that the whole subject could be resolved into one based on the action of organised poisons. When, however, we come to consider that certain disease-producing organisms can produce their poisonous products outside the body, *i.e.* from dead organic matter, whilst others produce theirs locally in some accumulation of fluid or on a devitalised surface; that some organisms are killed off in the blood but retain their vitality when injected into subcutaneous or other tissues, especially if these tissues be bruised, but that others, again, appear to have the power of parasitic growth even within the blood-vessels, either free or on deposits of fibrin, it is evident that the method of attack must vary very greatly in different forms of bacterial disease. Speaking roughly, the *bacterial surgical fevers* may be classified under three headings: (1) Sapræmia or true toxæmia, in which the products only of bacteria find their way into the system; under this heading two sets of conditions may be included: (*a*) in the case of the tetanus bacillus the manufactory may go on entirely outside the body, and the products of the organism, finding their way along with earth into a bruised wound, may set up a distinct tetanic condition before the micro-organism has had time to develop any further products within the body; (*b*) in other cases of tetanus the bacillus is introduced locally into devitalised tissues: here a fresh quantity of poison is developed by the multiplying organisms at the seat of injury, and is absorbed into the body, the bacilli remaining strictly localised. In diphtheria there is a similar toxic condition arising out of the development of poisonous products which are carried into the system, although here again the bacilli are strictly localised to the seat of original invasion. (2) In the case of true septicæmia, not only the bacterial products; but the bacteria themselves, find their way into the distant parts of the body; of this there are many examples among the lower animals; such diseases as anthrax and acute hæmorrhagic septicæmia may be taken as examples in the human subject. In the latter disease it is evident that the specific bacteria or their products are acting directly on the walls of the blood-vessels. (3) In order to create a further class for cases in which there are distinct clinical appearances, the old term pyæmia may be retained to denominate those cases in which we have the presence of abscesses, the result of septic thrombi or emboli, the active agents in these thrombi and emboli being bacteria which have usually found their way into the body by way of some accidental or surgical wound. (*See* Art. VIII.)

12. The nature of the toxic products.—The nature of the toxic products of the more important bacteria has been worked out almost entirely in connection with those which are associated with surgical diseases. That exceedingly soluble poisons are formed by these organisms is evident from the fact that in the cases where the lesions are strictly localised the constitutional disturbances may be strikingly marked, especially in those conditions with which the surgeon has to deal, or which through usage come specially under his notice; thus malignant or septic œdema, symptomatic anthrax, tetanus and diphtheria, are all characterised by this peculiarity, and although the surgeon is called upon to treat the local lesions in order, if possible, to cut out the manufactory of these poisons, the rate of absorption of the formed poisons is so great that the most profound toxic symptoms may continue up to the death of the patient, even after the removal of the local lesion or after the destruction of the bacteria in this lesion. On the other hand, in the septicæmias (accepting this term in the sense in which we have already defined it) the local lesion may be of the most trifling nature; it has simply served as the point of entrance of organisms, which, enabled to multiply in the blood, and especially in the internal organs, are capable of setting up serious constitutional disturbances similar, in many respects, to those that follow the absorption of poison from the local lesions. Such diseases are characterised at some period or other (usually a short time before the death of the patient) by the presence of micro-organisms in the blood, as, for example, in cases of anthrax, but especially in that group of diseases known as the hæmorrhagic septicæmias, which in different animals are associated with bacteria bearing a strong family likeness. For example, we have the bacillus of rabbit septicæmia, of fowl-cholera, of the septicæmia of deer, of mouse septicæmia (Plate II. Fig. 11), of erysipelas in pigs, and the septicæmias set up by the Pneumococcus or Micrococcus lanceolatus.

Most of the earlier attempts to separate the poisonous products from cultures grown in special proteid media (in which only many of these organisms can produce their poisonous products), were directed to obtaining substances corresponding to the vegetable poisonous alkaloids, which could be crystallised out by means of special reagents. Panum's *sepsines* and Selmi's *ptomaines* were looked upon as mixtures of such crystallisable substances. Then Brieger separated from tetanus products distinct substances, one of which he named *tetanin*, another *tetano-toxine*, another *spasmo-toxine*, and a fourth a basic substance (nameless), which when injected was found to bring about a rise of the temperature of the animal into which it was injected. These alkaloids, poisonous in a certain degree, are, however, incapable of producing the extraordinary results observed in the diseases with which they are associated, and it was soon recognised that alkaloids are probably the result of the breaking-down of more complicated and certainly of more virulent substances; especially as these poisonous alkaloids, in turn, under the influence of oxidation, lose much of their virulence. It was then naturally suggested that the actual poisonous agent might be mixed with or entangled amongst the crystals, rather than form an integral part of them. An important crystalline alkaloid, however—*phlogosin*—has been obtained by Leber from the products of the Staphylococcus aureus; which injected gives rise to marked suppuration. Similarly, Charrin has found an alkaloid—*pyocyanin*—in blue pus, the Bacillus pyocyaneus being able to form a similar substance in appropriate nutrient media outside the body; and Sidney Martin has succeeded in isolating a poisonous alkaloid from anthrax blood and even from cultures of the anthrax bacillus in alkali albumen.

Within recent years an impetus has been given to the study of these poisonous products by Roux and Yersin, who, along with Sidney Martin and others, have been able to demonstrate that in the diphtheria poison there is no crystallisable alkaloid. Brieger, giving the name of *toxines* to the poisonous products of bacteria as a whole, divided them into crystallisable and non-crystallisable or amorphous toxines, giving to the latter the name of *tox-albumens* or toxic proteids, as they are found to give certain proteid reactions and are formed most readily from proteid substances. Even when they can be built up from non-proteid materials, they are formed only through the action of the protoplasm of the bacteria. But just as we have now come to look upon the crystallisable alkaloids as the result of the breaking-down of these proteid toxines, it is maintained by many observers, as first suggested by Loeffler, and then by Roux and Yersin, that the real toxic agent is something even more refined than the proteid, which may serve merely to hold or entangle an intensely virulent substance, probably of the nature of an enzyme or organised ferment, which may vary somewhat in character, according as it is locked up in globulines, nucleo-albumins, peptones, albumoses, or like substances. Whatever may be the nature of the poisonous substance locked up in the proteid molecule, there can be little doubt that we have not yet arrived at its exact chemical composition; and for the present we can only extend our knowledge of these poisons by physiological experiment, though the vast amount of clinical work that is being done appears to promise results of great value to the physiologist, the surgeon, and the physician.

It should be pointed out that the amorphous toxic products or proteid poisons may be divided into two classes. The *first* of these classes contains those which may be looked upon as secretions or excretions of bacteria, or the result of the action of bacteria on the surrounding media. These, as a rule, are exceedingly soluble, and, so far as our knowledge at present goes, have a more specific action than the second group—*i.e.* to them appears to be due the specific activity of a micro-organism. The *second group*—one of very great importance, from the surgical point of view—contains those poisons which are more intimately bound up in the protoplasm of the bodies of bacteria, and are set free only in very old cultures, or when the bodies of the bacteria are treated with a strong alkali, or when they begin to undergo degenerative changes. This group of substances may be formed from non-proteid materials, though this is not usually the case. These substances, unlike the enzymes, are, as pointed out by Buchner, extremely resistant to the action of heat, as they remain capable of producing their specific effects even after being boiled. They may be dissolved out by the action of alkalies, and in alkaline solution they possess many of the properties of the original substance. It is in consequence of the presence of this form of proteid poison that the bodies of many bacteria, even when rendered incapable of multiplication by the action of prolonged boiling, have still the power of setting up local suppuration. Koch and Mitchell Prudden and Hodenpyl have drawn attention to this peculiarity of the protoplasm of the tubercle bacillus, and no fewer than seventeen organisms have been found, which, even when dead, have the same properties. They have not only the power of acting, by what is called *chemotaxis*, on the leucocytes, attracting them to the point at which they are placed, but they exert such an action through the poisons they contain, that the leucocytes undergo degenerative changes and death, and as a result suppuration is set up.

Proteids formed by bacteria vary according to the nutrient media in which the cultivations are made. Thus, Hankin only succeeded in producing the proteid poison of anthrax bacillus when he grew it on sterilised fibrin, and Sidney Martin was only successful in producing the anthrax albumose when he cultivated the bacillus on alkali albumen.

The anti-toxines, which may be considered under the heading of Immunity, are also apparently of a proteid nature, but little is known of their chemical composition.

13. Immunity.—Individual insusceptibility to certain diseases is so well marked that many people are said to be incapable of taking any of the exanthematous diseases, whilst in the popular mind an attack of one of these diseases is looked upon as an almost specific protection against a second attack, and such insusceptibility is looked upon as being to all intents and purposes permanent. This popular supposition is based upon well-accredited facts and figures. On the other hand, it is supposed that a certain temporary protection is conferred by a single attack of diphtheria or Asiatic cholera, but that this protection gradually wears off; while there are people who are not only not protected by a single attack, but seem to be even more susceptible to future attacks. Hereditary or racial immunity or susceptibility is also recognised. Koch early pointed out that field mice resist the action of the bacillus of mouse septicæmia—an organism intensely fatal to the house mouse; whilst, on the other hand, as insisted upon by Loeffler and Schütz, the field mouse is susceptible to infection by the glanders bacillus, although white mice and house mice resist the disease in a most remarkable fashion. This resistance may often be readily overcome. If phloridzin be given in their food to white mice, glycosuria, which follows the exhibition of this drug, is set up, and the animals become immediately susceptible to the action of the glanders bacillus. They have, in fact, lost their immunity to glanders. It is sometimes said that anatomists and pathologists gradually acquire a certain immunity even against septic poisoning. There can be no doubt that individuals differ enormously as regards their susceptibility to poisoning by post-mortem dissection wounds. Again, an artificial immunity may be produced in animals against certain of what are known as surgical diseases. Pasteur has carried out this on a large scale in connection with anthrax. The same thing has been done with malignant œdema, diphtheria, and tetanus, and a temporary immunity has been obtained against the action of the erysipelas organism. A similar immunity can be produced against the action of the Pneumococcus, the Bacillus pyocyaneus, and the pyogenic bacteria, amongst which may be reckoned the pyogenic staphylococci and streptococci, certain members of the proteus group, and the Bacillus coli communis. Sometimes protection against pus-forming organisms, of a more or less complete character, may be observed, but at present no very definite results have been obtained, and no attempt has been made to apply the knowledge so obtained to the prevention of suppuration in the human subject. It will be seen that the methods that have been used to produce artificial immunity are, in most cases, of such a character, that their employment would be attended with very considerable risk, unless the exact conditions are more definitely laid down than they possibly can be at present.

It is well known that the number of virulent organisms injected into the tissues, or into the blood, is a most important factor in determining whether a disease shall be mild or acute; and numerous experiments have been made with the object of determining the lethal dose of certain bacteria when exhibited in different ways. It requires a certain number of tubercle bacilli to be introduced subcutaneously in order to set up tuberculosis. Taking a less harmful bacterium, Watson Cheyne showed that it requires 18 millions of the Proteus vulgaris to produce any appreciable pathological effect, whilst to produce an abscess in an animal by the introduction of the same bacterium it requires 250 millions; in this case it may be that the proteids contained in the bodies of these bacteria are the causes of the chemotaxis and of the degeneration of the attracted cells.

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This factor of *dosage* has been utilised in the production of immunity. To obtain such immunity, a dose, small enough not to be fatal, of a living virulent culture of the specific micro-organism against which the immunity is to be set up, is injected. A second method is so far to *attenuate* the virulence of the specific micro-organism, that comparatively large doses may be exhibited for the purpose of setting up a mild attack of the disease; in this method the boundary line between non-lethal and lethal doses is very much broadened.

Temperature

Such attenuation of micro-organisms has been effected in various fashions:—by the action of chemical reagents; by the cultivation of bacteria at slightly higher temperatures than those at which they flourish most luxuriantly, for instance, in the case of the anthrax bacillus, at 42° C., when all stages of attenuation may be obtained, virulence being ultimately lost; by exposure to light and air on an artificial medium; by drying, which gradually kills certain organisms, especially those which do not form spores; by the growth of two organisms side by side—the Bacillus pyocyaneus apparently yields products which interfere with the virulence of the anthrax bacillus; and by the passage of bacteria through certain animals, in order to diminish their virulence.

The third method of producing immunity consists in the introduction into the body to be rendered immune, of the *products of the specific bacterium* against which immunity is to be obtained; in some cases the germ-free filtrate is used, in others the sterilised cultures containing the dead bodies of the bacilli, which have been killed by heat or some chemical reagent. This toxic material may be heated or mixed with chemical agents which have the power of weakening the activity of these bacterial poisons. Lastly, the method now most in vogue is that in which what is known as immunised serum or blood serum (obtained from animals rendered immune by one or other of the methods above described) is used. Even secretions or certain excretions from immunised animals, such as milk or urine, are supposed to have this immunising power.

Whatever method be used, the following points are now almost proved: (1) that the immunising substances are in all probability of a proteid nature; (2) that sterilised cultures or filtrates deprived of their specific poisonous properties may have a marked power of producing immunity; (3) an inoculated animal is for a short time after inoculation more susceptible to an attack of the specific disease, but as soon as this stage of susceptibility has been passed, the resistance is increased; (4) whenever specific bacteria are used, either in the virulent or the attenuated form, the aim is to produce a mild attack of the disease, as a result of which the animal is immune; (5) the period required for the acquisition of immunity is in this case much greater than when anti-toxic blood serum is used, in which last, a protective influence is recognised almost immediately or, at any rate, in the course of a few hours; (6) it is found, however, that the immunity so produced (the passive immunity of Ehrlich) is more transient than that obtained by inoculation with micro-organisms or their products; (7) the blood serum of naturally immune animals has not yet been proved capable of producing immunity when injected into susceptible animals.

The manner in which this immunity is brought about is still a controversial point. Metchnikoff and his disciples maintain that immunity is due to the action of certain living amœboid cells of the body, which, it is held, are capable of ingesting and digesting living micro-organisms which find their way into the blood or tissues. The polynuclear leucocytes are the most important, but the whole of the connective tissue cells may assume the same function. The leucocytes of susceptible individuals appear to be repelled by the pathogenic bacteria which, left unattacked, multiply and form their poison *in situ.* In the case of an immune animal, the leucocytes, instead of being repulsed by the bacteria, attack them, envelope them in their protoplasm and digest them, thus preventing them from carrying on their work, surrounding them and bringing about their localisation. It is supposed that the process of immunisation consists in a kind of acclimatisation of the polynuclear leucocytes and other phagocytic cells to the presence and action of special micro-organisms. Those who do not believe in *phagocytosis*, or who hold that it is only partially responsible for the process, maintain that bacteria are killed or weakened by the blood serum, and that only after such weakening is it possible for them to be taken up by leucocytes; it may be readily understood that where the phagocytic cells have undergone degeneration, as in tuberculous nodules and in abscesses, many of them may succumb in the effort to cope with active bacteria. That these cells can destroy living bacteria, now admits of very little doubt, but that micro-organisms are weakened by the fluid constituents of the blood may also be accepted as almost proved, though this only takes us a step farther back, as the quality of the blood plasma must, in great measure, depend upon the conditions obtaining in the cells which it contains and the tissues through which it passes, and any immunising products which these fluids contain must be derived from the cells, so that ultimately we must look to the cells for the production of the immunising factor, whether the phagocytic or the humoral theory be accepted. Chemotaxis is almost a necessary factor in the theory of phagocytosis, as only when the proteids of degenerated cells attract leucocytes more than the toxic proteids repel them, is it possible to bring the cells into contact with the bacteria, without which there can be no phagocytosis.

The *exhaustion theory*, promulgated by Pasteur, was based on the supposition that specific bacteria, when introduced into the body, are capable of using up certain materials which are necessary for their nutrition, so that these bacteria gradually die out; and even when introduced afresh, cannot exist, as there is nothing left for them on which to feed. Then came what was known as the *retention theory*, in which it was assumed that bacterial products inimical to the existence of the bacteria which form them, gradually accumulate in the blood, there remaining for an indefinite period, so that any new intruders of the same species are at once killed. This theory, however, is not now seriously held in its original form. The third theory is that the serous fluids of the blood and lymph have the power of neutralising or antagonising the specific poison of certain micro-organisms. This undoubtedly holds true, in a certain degree, in the acquired immunity against diphtheria obtained by injecting anti-toxic serum. A fourth theory, now somewhat generally received, is that these serous fluids have the power of exerting a *bactericidal action* on the micro-organisms themselves, quite apart from their power of acting upon the toxines produced by the micro-organisms, though these two latter very frequently run side by side.

It is here unnecessary to enter into the various suggestions made as to the cause of this bactericidal power of serum, but it has lately been put forward by Kossell and Vaughan (quoted by Welch) that the defensive proteids, which are exceedingly unstable, really owe their characteristic action to the presence of nuclein or nuclenic acid, so that we must ultimately look upon all these altered states of the serum as depending upon changes which take place in the tissue cells and leucocytes. One of the chief duties of the surgeon, therefore, is to keep the tissue cells and leucocytes so well nourished by judicious feeding, good hygiene, the prevention of the loss of blood, and the removal of all dead matter from the body, that they may be as little embarrassed, and may carry on their functions as perfectly, as possible.

SPECIAL BACTERIA.

Pyogenic bacteria.—It has already been mentioned that cultures of various species of bacteria, previously sterilised by heat, on being introduced in considerable quantities into animal tissues, have the power of setting up a localised suppuration, just as certain products of these same micro-organisms (sepsin, cadaverin, etc.) appear to exert a definite chemical action on the tissues, by which they are deprived of their vitality, acting in exactly the same fashion as nitrate of silver, strong ammonia, oil of turpentine, and similar substances. In most cases, however, the *products* themselves, apart from the proteids contained in the bodies of these bacteria—*i.e.* when filtered—have little or no power of setting up suppuration. When, however, the bodies of the dead bacteria that are kept back by the filter, are injected subcutaneously, suppuration follows. This occurs not only when the ordinary pus cocci are used, but also when the Pneumococcus, anthrax bacillus, tubercle bacillus, Bacillus prodigiosus, Bacillus coli communis, Bacillus acidi lactici, and other similar organisms are injected. It is now recognised, however, that the most frequent causes of suppuration are a small group of micrococci, of which it is necessary to describe only a few typical examples.

1. **Staphylococcus pyogenes aureus.**—The first of this group—the Staphylococcus pyogenes aureus (Plate II. Fig. 10; *see also* Fig. 29)—was first observed by Ogston in 1881, in the pus obtained from acute abscesses, and a couple of years later was again found and described by Becker in the pus from cases of acute osteomyelitis. This organism is the most common of those found in abscesses as a facultative parasite, but it may be met with everywhere where man and animals congregate, especially on the skin and the mucous membranes of the upper reaches of the alimentary and respiratory tracts. It is about $\cdot 7\mu$ to $\cdot 9\mu$ in diameter, and usually occurs in small irregular groups, in short chains of three or four links, or in tetrads. It stains readily by Gram's method. (*See* page 51.)

It grows on most media, and is a facultative anaërobe, since—though it flourishes most luxuriantly and produces its colour only in the presence of air—it still continues to grow when air is excluded. It liquefies gelatine more rapidly near the surface than away from the air; after a time, when the pigment is fully formed, the organisms fall to the bottom, and accumulate in a beautiful orange-coloured mass, exceedingly characteristic of the growth. On nutrient agar-agar and potato there appears a moist growth—white at first, or pale yellow, with somewhat wavy outlines; as it becomes thicker the growth assumes an orange or ochre-yellow colour. On plates it occurs as small white dots, which gradually become yellower. When grown in milk, it forms lactic and butyric acids, and coagulation occurs. It may be kept alive on agar-agar or in gelatine for a whole year without re-inoculation. When kept moist, it is killed in about ten minutes at a temperature of from 56° to 58° C.

Injected in considerable quantities under the skin of one of the small rodents, it sets up an abscess at the seat of inoculation. These abscesses are usually not fatal, unless very large, though sometimes a general infection may follow. When injected into the circulation, small capillary emboli, multiple abscesses of the kidney, and even abscesses in the joints and other organs result. Garré, who rubbed cultures of this organism freely on the skin of his own arm, obtained a regular crop of cutaneous furuncular abscesses. This staphylococcus is found in many of the more acute skin diseases, especially in those which are accompanied by suppuration, in acute abscesses, in acute osteomyelitis, and in ulcerative endocarditis. There can be little doubt that in cases of heart disease accompanied by a deposition of fibrin, or where there is a rupture of the valves, the introduction of these micrococci into the blood-stream is followed by a specific infective process, the micrococci becoming attached to the roughened surfaces, where they multiply, and are then distributed as small emboli, which, impacted, form the foci around which abscesses are formed.

2. **Staphylococcus pyogenes albus.**—The second species (Staphylococcus pyogenes albus) is identical with the first, except that it produces no colouring matter, and is supposed to be not quite so virulent—and is frequently found in the same positions. Welch describes an almost exactly similar organism, the Staphylococcus epidermidis albus. It is only slightly pyogenic, but is the organism most frequently met with on and in the skin. Its action in liquefying gelatine, in bringing about the coagulation of milk, and in producing pus when injected subcutaneously, or setting up certain lesions when injected into the circulation, is considerably inferior to that of the Staphylococcus aureus; but this organism is said by Welch to be the most usual cause of the small abscesses that occur around tense stitches.

3. **Staphylococcus pyogenes citreus** and **Micrococcus pyogenes tenuis.**—The Staphylococcus pyogenes citreus is identical in appearance with those already described, except that it assumes a delicate lemon-yellow colour, and that it liquefies gelatine still more slowly than either of the other forms. It is sometimes

found in acute abscesses, but as yet there is little evidence that it has any marked pyogenic activity.

The Micrococcus pyogenes tenuis, which seldom takes the staphylococcus form, is somewhat larger than the Staphylococcus albus, and the elements are more distinctly divided into hemispheres than in any of the other forms. On agar-agar it grows as a narrow, thin, transparent, watery layer. Its relation to pus-formation has not yet been determined, although it has been found in the pus in several cases of abscess.

4. **Streptococcus pyogenes.**—At present there appears to be some confusion as to the organisms to be included under this heading, as no fewer than six organisms, to each of which a separate name has been assigned, have been described by different observers; of these, however, three probably belong to one group, two to another, whilst the last may be a separate species. It will be found, however, that they are almost identical as regards their microscopical and naked-eye appearances, and it is only when the products of these organisms and their relative pathogenicity are inquired into, that any distinctive characters are found.

First group.—The first member of this group, the *Streptococcus pyogenes* (Plate I. Fig. 5; *see also* Figs. 26, 27, 28, and 34) may be taken as the type; it is found wherever there is progressive erysipeloid suppuration, in very acute abscesses, and in certain cases of ulcerative endocarditis. (*See* Art. VII.) The cocci, which vary very much in size—from ·4μ to ·8μ in diameter—are arranged in pairs or in chains of considerable length. In gelatine tube cultures there is an exceedingly delicate growth on the surface, whilst in the depth, a number of very minute granules, more discrete and larger as we pass away from the surface, are seen; as the surface growth increases in size it also becomes thicker, the edges become wavy, and there is a peculiar, almost characteristic, terraced appearance. This organism grows slowly at the room temperature, and best at the temperature of the body; it does not liquefy gelatine. It stains well by Gram's method (page 51). It is pathogenic to both mice and rabbits.

Streptococcus pyogenes malignus, which was separated from the necrotic foci of a leucæmic spleen by Fluegge, is similar to the form already described, but it grows more slowly. A suppurating point appears at the seat of inoculation, and a few cocci may be found in the blood and in certain organs. It kills both rabbits and mice.

Streptococcus septicus belongs to the same group. The micrococci form chains which break up into diplococci. It grows comparatively slowly, and is fatal to mice and rabbits, killing them in from two to three days. In the case of rabbits injected through the aural vein there is first a local hyperæmia, and then masses of the organisms collect in the vessels to such an extent that they form thrombi, round which necrotic areas become well marked.

Here, then, is a group of organisms all of which have certain characters in common; they prove fatal to mice as well as to rabbits, in this differing from the next group.

Second group. Streptococcus erysipelatis and *Streptococcus septopyæmicus.*—The Streptococcus erysipelatis and Streptococcus septopyæmicus have been described as separate species, though they are probably the same. Their morphological features are very much those of the organisms already described; they are from ·7μ to ·8μ in diameter, occur as diplococci, or as long chains, and are usually found in the lymph channels of the skin or mucous membranes, in cases of erysipelas (page 164).

Grown in gelatine, they appear as very minute points, with scant surface growth; on plates the colonies are small, rounded or ovoid, finely granular, with somewhat irregular margins. On agar-agar they develop as very delicate, translucent, greyish, slightly raised colonies, which spread somewhat slowly in the case of Streptococcus erysipelatis, but more rapidly in the so-called Streptococcus septopyæmicus. Streptococcus erysipelatis, however, does not grow upon potato, whilst small dirty white colonies, somewhat raised from the surface, appear when the Streptococcus septopyæmicus is sown.

Either of these organisms, when inoculated into the ear of a rabbit, sets up, first of all, an erysipeloid inflammation usually unaccompanied by suppuration; in the human subject the organism is found just beyond the spreading margin of erythema in cases of erysipelas, and appears to be very readily killed, disappearing in any region in which the blush is well marked. In this respect these organisms differ somewhat from Streptococcus pyogenes, which develop, in the human tissues more readily than the erysipelas coccus; this, however, may be partly a question of situation and partly one of oxygen and food supply and removal of the excretory products of the microbes. Nencki and his pupils maintain that the difference in the products of the two organisms, when grown in various media, indicates that the Streptococcus pyogenes and the Streptococcus erysipelatis are distinct species. (*See* Art. VII.)

Third group. Streptococcus articulorum.—The last of the important members of the pyogenic organisms is the Streptococcus articulorum, separated by Loeffler from the mucous membranes in cases of diphtheria. It occurs in long chains, of which each segment consists of two hemispheres showing in stained specimens a delicate, clear, intermediate zone. It grows in the form of small, limpid, light grey drops with slightly irregular outlines. On potato, after eight days, the colonies are like those of Streptococcus erysipelatis, and are only distinguishable under the microscope. On blood serum, alone or mixed with peptonised meat infusion, a thin, pale, glistening or greyish-white layer appears at the end of a couple of days. The organism is pathogenic for mice but not for guineapigs. When injected into the veins of a rabbit, the joints seem to be specially affected, early death resulting.

A large number of other *pyogenic* streptococci have been described, but they are probably similar to those above-mentioned, and it is unnecessary to do more than indicate the types of diseases with which they are associated. They are found, for instance, in puerperal fever, in false membranes along with the diphtheria bacillus, in abscesses and inflammatory processes, especially those connected with the buccal and nasal mucous membranes, in cases of peritonitis, in various forms of otitis, in fact wherever superficial pus-formation is met with; in furuncles, pustular skin eruptions, erysipelas, and phlegmonous inflammations, especially those of badly nourished patients, or in those in whom there is any interference with the removal of excretions, or where there is anæmia, especially following hæmorrhage. These streptococci, then, must be looked upon as the pus-forming organisms *par excellence*, although they can only exert their full pus-forming function when the surgical conditions are favourable.

Micrococcus gonorrhœæ (Plate II. Fig. 12; *see* also Fig. 93).—This organism was first described and separated from gonorrhœal pus by Neisser in 1879, who named it the Gonococcus. In 1885 Bumm succeeded in growing it by spreading pus, containing a large number of gonococci, out on a thin layer of consolidated human blood serum and keeping it at a temperature of 30° to 34° C. in a moist atmosphere. It forms a very thin, smooth, moist layer, almost invisible, but having, when seen by reflected light, a greyish yellow colour. It dies out very rapidly—at the end of two or three days. More recently, it has been found possible to grow this organism in a mixture of equal parts of agar-agar and blood serum. It has also been grown on the white of egg of the peewit and in the fluid removed from the joint in a case of synovitis. Bumm was able to produce typical gonorrhœa by inoculating the human urethra with a third and even with a twentieth culture of the gonococcus, but he failed to produce the disease in the lower animals. When suppuration is well advanced, other organisms, such as the Staphylococcus pyogenes aureus, are usually found along with the gonococcus, so that this latter organism appears to prepare the way for suppuration, rather than to be the actual ætiological factor.

It is found in the cells of the gonorrhœal discharge or on the surface of the detached epithelial cells, and occurs most frequently and in greatest numbers in the early stages of the disease. In sections it is seen to be specially numerous in the cement substance between the epithelial cells. When the discharge from an acute case of gonorrhœa is stained and examined, the micrococci are seen to consist of pairs or fours of organisms, each of which is composed of two biscuit-shaped ends with a zone or belt of unstained material between, an arrangement already noted in the elements of some of the streptococci. In consequence of this arrangement, the organism, or perhaps the pair of organisms, is usually slightly longer than broad—$\cdot 8\mu$ to $1\cdot 6\mu$ long, and $\cdot 6\mu$ to $\cdot 8\mu$ broad. The gonococcus differs from the staphylococci and streptococci already described, in that it is not stained by Gram's method, all colour being removed by the iodine solution. (*See* page 51.) It is best stained with methyl-violet, gentian violet or fuchsin; more slowly but very distinctly with methylene blue. (*See* Art. XXI.)

Bacteria associated with croupous pneumonia.—The organism which is most frequently met with in cases of pneumonia gives rise also to certain forms of septicæmia and meningitis; it has been found also in acute abscesses, in ulcerative endocarditis and in otitis media. It is now recognised that *Friedländer's bacillus* plays a comparatively unimportant part in the production of pneumonia, and no causal relation between it and surgical diseases has yet been traced; but a similar organism (Plate II. Fig. 9), the *Micrococcus pneumoniæ crouposæ*, the *Bacillus salivarius septicus*, or the *Streptococcus lanceolatus Pasteuri* (Sternberg), is of greater surgical importance. This organism has been found both in the rusty sputum of cases of croupous pneumonia, and in the saliva of apparently healthy individuals, sometimes appearing there for a short time only, but in others remaining almost permanently. It has also been found, alone or accompanied by the Streptococcus pyogenes, in many cases of purulent cerebro-spinal meningitis, in acute abscesses in the parotid, and in cases of purulent pleurisy, in abscess of the tonsil, in otitis media, and in cases of arthritis of the wrist and shoulder-joint following pneumonia.

It occurs as a somewhat ovoid or grain-shaped organism surrounded by an unstained area, arranged in pairs or in short chains. It is stained by Gram's method, thus differing from Friedländer's bacillus, from which the colour is discharged by the iodine solution. (*See* page 51.) The Bacillus lanceolatus is said to grow only in alkaline media, whilst the Bacillus salivarius septicus grows most luxuriantly in a medium to which ·05 per cent. hydrochloric acid, or an equivalent quantity of nitric or phosphoric acid, has been added. This latter organism will grow in meat infusions, giving rise to a slight milky opacity; after a time the multiplication stops and the organisms settle at the bottom, to form a dense precipitate, the supernatant fluid becoming clear. It may also be cultivated on gelatine, which it does not liquefy, small white colonies developing along the track of the puncture; at the surface it forms a fine white mass, somewhat transparent at the edges. On nutrient agar-agar or coagulated blood serum the growth appears in the form of small transparent drops, some of which run together to form a thin line along the track of the needle; there is no growth upon potato; in milk, acid is formed, the casein becoming coagulated. The organism is very delicate, and soon dies out unless cultures are made every three or four days; it is very susceptible to the action of most disinfectants.

It is highly pathogenic for mice and rabbits, but guineapigs are not so uniformly affected. The two former animals succumb during the first or second day; there is local pus-formation accompanied by a diffused cellulitis with marked œdema, the fluid from which and the blood both contain numerous micrococci. The passage of this organism through a series of animals invariably increases its virulence. The experiments on the production of immunity with filtered cultures of the micrococcus have given very conflicting results, and must be repeated before they can be accepted.

Other micrococci of surgical interest.—Other micrococci to which surgical interest attaches are the *Diplococcus intracellularis meningitidis* of Weichselbaum, the *Staphylococcus salivarius pyogenes*, the *Micrococcus salivarius septicus*—already referred to as Bacillus salivarius septicus, the *Micrococcus tetragenes*—described by Gaffky as occurring in a cavity in the lung, also by Greenfield and others as occurring in septic conditions. *Micrococcus gingivæ pyogenes*, described by Miller, of Berlin, the *Streptococcus septicus liquefaciens*, described by Babes in the organs of a child which succumbed to septicæmia following an attack of scarlatina, and the *Streptococcus coryzæ contagiosæ equorum* which occurs in the pus of the swollen tissues, and in the abscesses that are found in cases of strangles.

Bacillus anthracis.—The Bacillus anthracis was first described by Pollender in Germany in 1849, then by Davaine in France in 1850; it was more fully investigated by Davaine in 1863, and by Pasteur in 1879. (Plate I. Figs. 3, 4.) It is a rod-shaped bacterium from 1μ to $1{\cdot}25\mu$ in breadth, and from 5μ to 20μ or 50μ or more in length. In the blood it occurs as short, simple or jointed segments about 5μ to 8μ in length, with flattened or cup-shaped ends, in the latter case a small clear disc being usually seen at the point of division; the bacilli are rigid non-motile organisms, which never contain spores when in the blood of the living animal. When seen in cultivations or in fluid removed from the thorax, they may appear as long filaments, sometimes grouped in bundles, at others forming a kind of felted mass. In old cultures or in organisms exposed to the air under somewhat unfavourable conditions, spores (*see* page 4) are formed. This organism may be easily seen under the microscope even in unstained preparations, but it is more readily studied when stained by any of the ordinary methods. It grows readily in peptone-gelatine at the ordinary temperature of the room, and in neutral or even slightly alkaline media. In a test-tube gelatine culture it first throws out a series of delicate threads around the central core formed along the track of the needle, the growth having then the appearance of an inverted fir tree (Fig. 2). Then liquefaction commences at the surface and shortly becomes fairly well marked; the fluid, at first opaque, gradually becomes clear as the non-motile organisms fall to the bottom, to form a greyish mass. In agar-agar plate cultures, incubated at the body temperature, greyish-white points develop; these, under the microscope, are most lace-like in their delicacy; on gelatine plates the colonies have the characteristic and often described "Gorgon locks" appearance. The Bacillus anthracis grows upon the surface of a dry potato as a dirty white layer with irregularly roughened surface and margins. As already mentioned, spores never occur in the bacilli found in the blood of living animals, but they soon develop at a temperature of 20° to 35° C. or even at the temperature of the atmosphere, in the discharges coming from a dead animal.

The virulence of these organisms can be modified by heating them

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at a temperature of 42·5° C. for a lengthened period (two or three weeks, according to Koch). Sunlight, and chemical and other agents also attenuate its virulence. The spores are exceedingly resistant, and remain alive and virulent for years if kept dry. They will also withstand a prolonged exposure to a *dry* heat of 140° C., three hours at least being necessary to destroy them. They are destroyed by boiling for three or four minutes.

The products of the anthrax bacillus are various proteid substances nearly allied to the albumoses and peptones, and an organic alkaloid. Toxalbumens are said to be formed, especially by the virulent bacillus when it is grown in fibrin or alkali albumen. An immunising substance is also formed, but the injection of serum from an immunised animal appears to confer only a very temporary immunity. Malignant pustule and pulmonary anthrax in woolsorter's disease, are the diseases set up by this organism, which are of interest to the surgeon.

The anthrax bacillus attacks cattle, sheep, horses, rabbits, guinea-pigs, and mice, especially the young of these animals; whilst white rats, dogs, frogs, and the Algerian sheep are immune. The organism is found in the blood-vessels, especially in the small capillaries of the various organs. In pulmonary anthrax almost pure cultures of long threads of the anthrax bacillus may be found in the serum thrown out into the thoracic cavity. Anthrax inoculation is usually marked by œdema of the connective tissue near the seat of inoculation, small hæmorrhages, some emphysema, pallor, flabbiness and moistness of the neighbouring muscles, with some cloudy swelling and hyaline degeneration. In animals that succumb to the disease the spleen is enlarged, soft, and pulpy, containing a very large quantity of blood, which has a somewhat tarry look. The liver has a parboiled appearance, and contains a large quantity of blood; the lungs are congested, the cavities of the heart are distended, and there may be fluid in the serous cavities. The blood and lymph from the tissues contain the bacilli in very considerable numbers. (*See also* Art. XV. page 312.)

Diphtheria bacillus (Plate I. Fig. 6).—The diphtheria bacillus, described by Klebs, Loeffler, and Klein, is a small straight or curved organism, with pointed or rounded ends. It often contains in its substance small clear spaces, or bright, glistening points. The bacilli are usually from 3μ to 6μ in length, are straight or slightly bent, sometimes swollen at one or both ends, or they may be elongated into club- or skittle-shaped organisms, into threads, and other elongated forms. These latter, however, are only met with in artificial cultures.*

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* To obtain specimens for examination and diagnosis, remove a small fragment of the false membrane by means of a piece of absorbent cotton-wool, tied firmly to a pair of forceps or a penholder; transfer the fragment to a scrap of blotting-paper, and thence to a cover-glass, on which break it down as finely as possible, dry and fix over a Bunsen flame, and stain with Loeffler's alkaline methylene blue (page 51), or by Gram's method (page 51), or by the method described by Roux and Yersin, who use a mixture of equal parts of an aqueous solution of violet dahlia and methyl green, to which water is added until a clear but not too deep blue is obtained. A drop of this is placed on the organisms on a slide, the cover-glass is

When suppuration has set in, these bacilli can be seen only in the deeper layers of the fibrinous lymph of the membrane, because of the presence of numerous other organisms.*

This bacillus, apparently powerless to live in the human body, except at the seat of inoculation, has the power of secreting a virulent poison, which is most active when alkaline, the presence of free acid limiting its action very considerably. This poison is now usually considered to be a toxic albumen (toxic albumose and organic acid which, according to Sidney Martin, are the result of the digestive action of Roux and Yersin's enzymes on the proteids of the body, though Roux and Yersin seem to indicate that this is not the case). It is so virulent that $\frac{1}{20}$ of a milligramme is sufficient to kill a guineapig, $\frac{1}{5}$ of a milligramme to kill a rabbit, and $\frac{2}{5}$ of a milligramme a medium-sized dog. When injected into the veins or into the subcutaneous tissues, this poison appears to act on the walls of the blood-vessels, giving rise to vascular dilatations, hæmorrhages, and the peculiar œdematous areas so characteristic of certain forms of the disease. From the behaviour of this substance it has been suggested that the noxious agent is a poison-forming substance rather than a toxic substance itself, and that it is only after it has been introduced into the body that the actual poisons are formed. This toxine has been separated from cultures or media containing proteids and from media in which there are none, and from animals and human beings who have died of diphtheria. The diphtheria bacillus retains its vitality for some time. It is capable of growing at comparatively low temperatures, and on most kinds of organic matter, especially in milk and on potatoes, although it is a somewhat difficult matter to determine its presence in and on these media, without having recourse to microscopic or cultivation examination. The importance of this point in connection with the spread of diphtheria is very obvious. (The anti-toxic serum treatment is considered in Art. XLIII., on INJURIES AND DISEASES OF THE NECK, Vol. II.)

Bacillus of influenza.—The bacillus of influenza is the smallest bacterium yet described, being about the same breadth ($0{\cdot}1\mu$ to $0{\cdot}2\mu$), but only about half as long ($0{\cdot}4\mu$ to $0{\cdot}5\mu$) as the bacillus of mouse septicæmia. Pfeiffer first, and then Canon, found it in the purulent

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lowered on to it, the superficial fluid removed with a piece of blotting-paper, and the preparation is examined at once. The diphtheria bacilli, seen in small groups, are more readily stained than any of the other organisms present.

* In order to obtain cultures, a particle of the false membrane is taken on a platinum needle, or on a cotton-wool pad as mentioned in the previous foot-note. This needle is then drawn eight or ten times (or the pad once) over the surface of a glycerine agar-agar tube or a tube containing a mixture of three parts of blood serum and one part of neutralised broth, to which have been added 1 per cent. of peptone, 0·5 per cent. of common salt, and 1 per cent. of grape sugar. The strokes are made parallel to one another, like ploughed furrows. Incubated at a little below the body temperature, colonies of bacilli rapidly make their appearance, and are visible to the naked eye in twenty hours as small rounded greyish-white points, with an opaque centre; these spread into greyish rounded discs of considerable size, before any other organisms with which they may be confounded have had time to form colonies at all visible to the naked eye.

discharge from the bronchi of patients suffering from epidemic influenza. It grows on the surface of glycerine agar-agar to which hæmoglobin has been added, in the form of minute pellucid colonies which appear at the end of twenty-four hours (at 38° or 39° C.). These colonies remain perfectly distinct. They grow best when a small quantity of the albuminoid secretion from the lungs or a drop of blood, is smeared over the surface; hence it is always much easier to obtain cultures from the original secretions than to obtain secondary cultures, unless hæmoglobin be added to the nutrient medium. The bacillus is destroyed by drying for twenty hours, or by exposure to a temperature of 60° C. for five minutes.*

The two ends of the short bacillus are more deeply stained than the centre; so that the organism very frequently resembles a diplococcus. This organism has a special interest for surgeons, from the fact that lesions so frequently follow cases of epidemic influenza; but the relation of the bacillus to these disorganisations and suppurations has not yet been clearly traced.

Bacillus of tuberculosis (Plate I. Fig. 7).—The tubercle bacillus is found in tubercular glands, abscesses, and sinuses in joints, bones (especially in tubercular caries), and in various skin affections, *e.g.* lupus, and has thus great interest for the surgeon, by whom the application of surgical treatment to cases of tubercular laryngitis and cavities in the lungs must now be looked upon as part of his work. It is a rod-shaped organism from 2μ to 5μ long—*i.e.* $\frac{1}{4}$ to $\frac{2}{3}$ the diameter of a red blood corpuscle—and $\cdot 2\mu$ to $\cdot 3\mu$ broad, with rounded ends. It is sometimes homogeneous, but usually has a peculiar beaded appearance. It is non-motile.

It is grown most readily on glycerine agar-agar, on the surface of which it forms a luxuriant growth, in the form of folded or wrinkled greyish or yellowish-white scales. It will also grow on potatoes, in the form of smooth colonies, which appear at the end of a fortnight or three weeks. The appearance on consolidated blood serum is that most frequently described. The colonies on this medium are in the form of small, dry scales, greyish-white, sometimes coalescing and "floating on the surface of the water of condensation"; they grow exceedingly slowly, but the maximum development is reached at the end of four weeks. They grow best at the body temperature.

The bacilli are to be found in the scrapings from tubercular sinuses, especially in the soft tissues. It is very difficult to demonstrate their presence in bones and even in glands; in other tubercular lesions this is a comparatively easy matter. Tubercle bacilli are best stained by the Ziehl-Neelsen method.†

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* This organism is stained as follows (*Chenzinsky's method, as modified by Plehn*): A concentrated aqueous solution of methylene blue, 40 grammes; ½ per cent. solution of eosin dissolved in 70 per cent. alcohol, 20 grammes; distilled water, 40 grammes. Cover-glasses immersed in this staining solution are kept at the body temperature from three to six hours, then washed with water and mounted in balsam.

† *Ziehl-Neelsen method for staining tubercle bacilli.*—This is a modification of the Ehrlich-Weigert method. The sections or cover-glasses are stained in Ziehl's carbolic fuchsin solution, made as follows: Fuchsin 1 part, dissolved in 10 parts of

The poisonous material contained in cultures of tubercle bacilli is proteid in nature; it withstands boiling, and appears to be derived directly from the bodies of the bacilli; when these latter are first subjected to boiling for some time, and then injected subcutaneously, they give rise to a circumscribed abscess, or when carried into the circulation, to the formation of giant cells similar to those seen in cases of tuberculosis. They do not, however, undergo caseation, and are strictly localised. These proteins, as they are called, have a marked power of attracting leucocytes. Other poisons which are probably rendered inert by the action of a boiling temperature may be the exciting cause of the caseation which is so characteristic of tubercle. (*See* Art. XVI. page 351.)

Bacillus lepræ.—The Bacillus lepræ was first described by Hansen in 1879, and then by Neisser in the same year. This bacillus is very similar in appearance to the tubercle bacillus; it is, however, more rigid and the ends are pointed. It is usually about 5μ in length and $\cdot 4\mu$ in breadth; otherwise it is like this organism, even as to its staining reactions, except that it is much more readily decolorised. As yet, any attempt to cultivate it has been unattended with success, though it is stated that positive results have followed inoculations into the anterior chamber of the eye in animals. This organism is found specially in the lymph channels of the skin, but sometimes evidently embedded in the protoplasm of cells. It has been found in all forms of leprosy at some stage or other of the disease.

Bacillus mallei (Plate I. Figs. 1, 2).—This bacillus was, in 1882, proved by Loeffler and Schütz to be the cause of glanders. It was first examined in the nodules occurring in that disease and then in cases of farcy. The organism is about the same length as the tubercle bacillus, but is considerably broader, being described as from $\cdot 5\mu$ to $1\cdot 4\mu$ broad. It is doubtful whether spores are formed. The bacillus is motile, and grows in gelatine tube cultures, but only luxuriantly at the temperature of the body, when it is seen as a whitish growth composed of chains and threads coiled irregularly and penetrating the gelatine in

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alcohol, to which solution 100 parts of a 5 per cent. watery solution of carbolic acid are added; this is heated until steam rises pretty freely. The cover-glass preparations are stained in three or four minutes, or even less; sections are usually sufficiently deeply stained in seven or eight minutes. In the cold they may be left for twelve or even twenty-four hours. The superfluous fluid is drained off, and the preparations are placed for a second or two in alcohol (90 per cent.), then in a 25 per cent. solution of sulphuric acid, when the pink tinge should be immediately replaced by a yellowish-brown. The preparations are then washed in 70 per cent. alcohol, and if they are sufficiently decolorised, are transferred to a solution of lithium carbonate. They may afterwards be stained, for a half to two minutes, with a watery solution of methylene blue, cleared up with clove or aniline oil, turpentine, and xylol, and mounted in Canada balsam. In place of sulphuric acid, nitric or hydrochloric acid may be used, and for clinical work, in order to shorten the process, the methylene blue may with advantage be mixed with the acid, the decolorising and contrast-staining being carried on in one process.

Gabbet's method for staining tubercle bacilli.—Stain for two minutes, without heating, in Ziehl's carbolic fuchsin solution, and after washing in water place for a minute in a solution of 2 grammes of methylene blue in 100 cc. of 25 per cent. sulphuric acid, wash again in water, dry, and mount in Canada balsam. This is an excellent practical method, being simple, easily carried out, and very certain in its result.

various directions. On plate cultures it is seen as bright yellow, shining, coalescing colonies; on agar-agar it has a somewhat greyer look; on potato, at the temperature of the body, it appears first as a bright yellow honey-like surface-growth, which gradually takes on a peculiar fawn colour, ultimately becoming almost chocolate-coloured (Fig. 4). It grows well on blood serum, but most luxuriantly of all on glycerine agar-agar, even at the room temperature, and is seen in the form of a white transparent line of considerable breadth along the track of the needle. The organism is best stained by a concentrated alcoholic solution of methylene blue, after which the cover-glass or section is rinsed in a mixture of distilled water 10 cc., concentrated sulphuric acid 2 drops, 5 per cent. oxalic acid 1 drop, for 5 seconds, dehydrated in absolute alcohol, then cleared with cedar oil and mounted in balsam.*

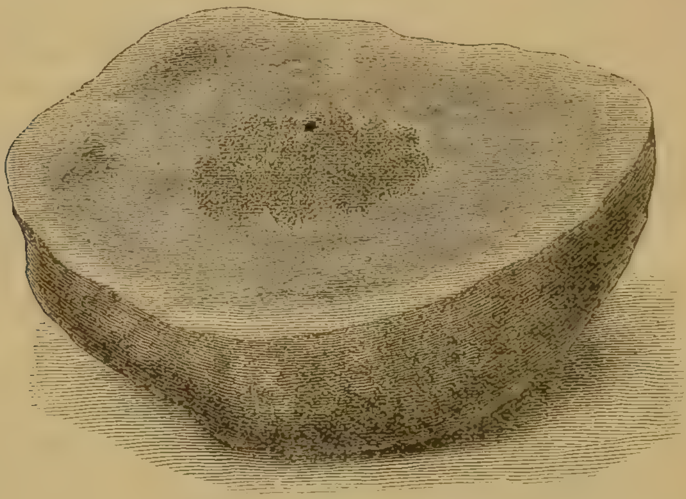

Fig. 4.—Cultivation of Bacillus Mallei on Potato.

Pure cultures of the Bacillus mallei injected into horses, rabbits, guineapigs, and field mice produce typical glanders, often associated with inflammation of the nasal submucosal lymphatics and with orchitis. The whole of the equine species and man are affected. A case of direct infection in man, due to the use of an imperfectly sterilised hypodermic syringe with which glanders cultures had been injected, is recorded. Lions and tigers have also been known to contract the disease. The discharge from the nostrils in cases of glanders contains few bacilli, but quite sufficient to make test inoculations of value. A guineapig inoculated subcutaneously in the back with discharge from a suspected animal, develops tumefaction at the seat of inoculation; the skin over this ulcerates, and there is discharged first a mass of purulent material, and a chronic ulcer with irregular indurated margins is formed, from which then comes a sero-purulent discharge; subsequently this ulcer may heal. The lymphatic glands in the neighbourhood are also affected, and may also ulcerate, and at the end of four or five weeks the animal becomes generally infected. The testicles become enlarged, and later there is enlargement of the joints, due apparently to an acute inflammatory process. Field mice die in three or four days. The inoculation test is far easier of application and more satisfactory than microscopic examination, as it is often an exceedingly difficult matter to determine the presence of these bacilli, mixed as they are with an enormous number of other bacteria which develop in the discharge as it accumulates on the surface of the ulcers. Intraperitoneal injection of the suspected fluid from an ulcerated mucous membrane or gland, induces, in the guineapig, inflammation of the testicles in three or four days; pus is rapidly formed, and in this pus glanders bacilli may be comparatively easily demonstrated, as early as the third or fourth day, especially in the purulent exudate between the two layers of the tunica vaginalis testis. Mallein, the toxic product of the glanders bacillus, is very similar in its character to tuberculin, and has the same specific properties in relation to glanders that tuberculin has to tuberculosis. Mallein, when injected, appears to have comparatively little effect on the healthy tissues, but when injected into a glandered animal it sets up not only great local reaction at the seat of the infection, but also very marked constitutional disturbances, especially those associated with an increase of temperature. So marked is the local reaction, and so invariably does it occur in glandered horses, that it is now almost universally accepted as an unerring diagnostic feature—as pathognomic of the disease in horses. (*See also* Art. XV. page 308.)

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* *Noniewicz's method* for staining glanders bacillus: Stain for two to five minutes in Loeffler's methylene blue (concentrated alcoholic solution of methylene blue 30 cc.; caustic potash 0·01 per cent. solution 100 cc.), wash in distilled water, and then decolorise for one to five seconds, according to the thickness of the sections, in a mixture of 75 parts of a 0·5 per cent. solution of acetic acid and 25 parts of 0·5 per cent. watery solution of tropæolin; wash again in distilled water, and after spreading the section on a slide, dry it first with blotting paper and then in the air or over a spirit lamp. Clear by dropping xylol upon it (oil of cloves, origanum and aniline oil must be avoided), and mount in Canada balsam. The bacilli stain dark blue to nearly black, and the tissue is stained light blue.

Bacillus of syphilis.—In 1884 Lustgarten described, in certain cases of syphilis, a straight or curved bacillus very similar in appearance to the tubercle bacillus, but not stained by the Ziehl-Neelsen method. This organism is curved, may be arranged in pairs set at a comparatively acute angle, or it may be somewhat spiral or S-shaped. The ends are slightly enlarged, the outline is slightly wavy, and there are small bright points from two to four in number in each bacillus, which is about $\cdot 3\mu$ to $\cdot 5\mu$ in length and $\cdot 2\mu$ to $\cdot 3\mu$ in breadth. The bacilli of syphilis are contained in large oval or polygonal cells, each cell containing only one or two at the most. The ætiological relation of this organism to syphilis has not been established, as it has never yet been cultivated, and all inoculation experiments have been unsuccessful.

This organism is stained in the Ehrlich-Weigert gentian violet solution.* The tubercle bacillus and leprosy bacillus stain in much the same way, and it has been suggested that this organism is the leprosy bacillus which appears in syphilitic cases as the result of a mixed infection.

Bacillus of rhinoscleroma.—Another organism of interest to the surgeon is the bacillus of rhinoscleroma, first observed by Von Frisch in 1882, in the newly-formed tubercular thickenings of the skin and nasal mucous membranes, sometimes followed by ulceration, occurring in rhinoscleroma. It is found in the large hyaline cells characteristic of this disease, and also in the lymphatic vessels, especially near the surface of the tumour. The bacillus may be seen in the juice expressed from one of the tubercles. It is short, two or three times as long as broad, and has rounded ends. It may grow as a coccus, singly or in pairs, and appears to develop into bacilli or long threads.

It may be cultivated on gelatine plates, giving rise to whitish-yellow round colonies which, under the microscope, have a distinctly granular appearance. In a gelatine tube the organisms form a peculiar nail-culture-like growth, like that described for Friedländer's pneumonia bacillus, which is white and porcellanous-looking, and is very viscid and sticky. There is on potatoes a slightly yellowish mass, which grows rapidly and develops gas.

It is readily stained with the aniline colours, and is seen to be surrounded by a kind of capsule. Although this organism has been separated from the tissues of rhinoscleroma, it is so like the Friedländer's bacillus, which has been described in the nasal secretions of healthy persons, and also of those suffering from chronic nasal catarrh, that it is by no means certain that this organism has any special relation to rhinoscleroma. The main differences are that it retains its capsule, even when cultivated on artificial media, and that it resists the decolorising action of Gram's iodine solution (page 51).

Bacillus septicæmiæ hæmorrhagicæ.—An organism, or group of organisms, important to the surgeon is the Bacillus septicæmiæ hæmorrhagicæ, described under different names by different observers—the bacillus of fowl cholera of Pasteur, the bacillus of rabbit septicæmia of Koch, the bacillus of swine plague of Loeffler and Schütz and of Salmon and Smith, and the bacillus of game or deer plague described by Hueppe. The bacillus of septicæmia described by Davaine in 1872, and separated from putrid blood, was also probably identical with this organism. The organism found in the blood and œdematous fluid of animals affected, is invariably a short bacillus with rounded ends, about 1·4μ in length and 0·7μ in breadth, *i.e.* about one-fifth the diameter of a red blood corpuscle, sometimes arranged in pairs or in short chains. The extremities of these rods are usually stained with the aniline colours, giving them up again on being treated with Gram's iodine solution, the central zone or band always remaining unstained. In cultures it grows best at the body temperature, and also when the supply of oxygen is somewhat restricted, hence its growth is always more luxuriant in the deeper layers of the culture media than near the surface. In plate cultures the organisms give rise to white isolated colonies of the size of a pin-head, which, under the microscope, appear granular. In tube cultures they occur as small granulated colonies which gradually coalesce; there may be no surface growth at all, but if there is it appears in the form of a thin whitish layer, sometimes smooth, sometimes slightly irregular. On agar-agar the growth is more transparent and greyer than on gelatine. On blood serum it forms a very delicate, yellowish, slightly iridescent layer. If cultivated at the ordinary room temperature it soon loses its virulence. Some observers describe a growth on potatoes, others do not; some affirm that milk is coagulated, others that it is not. All the members of this group of bacilli produce indol when grown in peptone solution, and cause a uniform turbidity of broth. They retain their vitality for a considerable length of time if kept moist, but drying kills them in less than a fortnight. A culture attenuated by growth at a low temperature, may recover its virulence when inoculated into a series of susceptible animals, such as the sparrow. The most characteristic results of the action of this bacillus are swelling of the spleen and lymphatic glands, swelling and ecchymoses of the mucous membranes, acute œdema at the point of inoculation, and hæmorrhages and degenerations of small areas of muscles. The bacilli continue to increase in the body after death. In less susceptible animals local abscesses are formed.

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* Place in the solution (page 51) for from twelve to twenty-four hours at the room temperature, and two hours in the incubating oven at 40° C. The sections are then washed in alcohol and placed for ten seconds in a 1·5 per cent. alcoholic solution of permanganate of potash, afterwards washed in a dilute aqueous solution of pure sulphuric acid, and then in water. It may be necessary to repeat this operation (*i.e.*, returning the sections to the permanganate) three or four times until complete decolorisation takes place. The sections are mounted in the usual way. Cover-glass preparations are washed in water instead of in alcohol.

The bacillus of the bubonic plague.—Quite recently Kitasato has described a bacillus, found in the blood and tissues of patients suffering from the bubonic plague, very much resembling the bacilli of the hæmorrhage septicæmias. This, he maintains, has an ætiological relation to the disease. These observations have been corroborated by Yersin and others. The bacilli are described as "rods with rounded ends, which are readily stained by the ordinary aniline dyes, the poles being stained darker than the middle part, especially in the blood preparations, and presenting a capsule sometimes well marked, sometimes indistinct." Like the Bacillus septicæmiæ hæmorrhagicæ, this bacillus is about one-fifth the diameter of a red blood corpuscle in length, and half as broad as long. It is sometimes single, sometimes arranged in pairs, or it may be in short chains. It is only slightly motile, even when grown in beef broth, which medium it renders somewhat cloudy. It grows best on blood serum at the body temperature, as a moist yellowish-grey growth, which does not peptonise the medium on which it is growing. It also grows freely on glycerine-agar-agar, as whitish-grey colonies, which by reflected light have a bluish appearance. Under the microscope they are rounded patches with uneven edges; "at first they appear everywhere as if piled up with 'glass-wool,' later as if having dense, large centres." Grown in gelatine there is little surface growth, but along the track of the needle a number of minute, white, cocoon-like bodies are seen, which ultimately run together and form an irregularly crenated line; the gelatine is not liquefied. The bacillus as cultivated grows in the form of long threads of bacilli, which, though they might perhaps be mistaken for streptococci, are distinctly made up of the organisms above described. This is due to the fact that the bacillus so frequently resembles a diplococcus with a capsule.

This organism is found in the blood, in the buboes, and in the spleen—in fact, in all the internal organs of patients suffering from the plague.

Mice, rats, guineapigs, and rabbits are all susceptible to inoculation of the disease. Animals fed with pure cultivations of the bacillus, and with pieces of affected organs, die with symptoms of the plague, and with pathological appearances similar to those found in cases of anthrax, or of malignant œdema. The organism is rendered innocuous by the action of direct sunlight continued for three or four hours; when dried in ordinary daylight for a week they are also killed; as also by the action of moist heat at 80° C. for thirty minutes, or at 100° C. for a few minutes. A one per cent. solution of carbolic acid, or of quicklime, destroys the organism in less than an hour.

It is evident then, from the post-mortem appearances, that we have in this disease to deal with a hæmorrhagic septicæmic process, so that the organism naturally comes to be classified in the above group.

Toxic saprophytic bacteria.—In consequence of the toxic effects of the products of certain saprophytic bacteria, it is necessary to give a short description of some of those which have been found, especially in abscesses or, under certain conditions, even in the blood of animals affected.

1. **Bacillus coli communis** (Plate II. Fig. 8).—The best example of this group is the Bacillus coli communis, or Bacterium coli commune, described as Emmerich's bacillus, or Bacillus Neapolitanus, from the fact that it was found by Emmerich in cases of diarrhœa in Naples. It is also probably identical with Brieger's bacillus, or the Bacillus cavicida.

This organism, which is constantly present in the fæces of the human subject, and of many of the lower animals, occurs in the form of short rods, about 2μ in length and $\cdot 5\mu$ in breadth, with rounded ends. It sometimes appears almost like a micrococcus or short oval bacillus, sometimes arranged in pairs. It is not stained by Gram's method (page 51), but is readily brought out by the ordinary aniline staining methods.

It grows rapidly at the room temperature, but better at about 37° to 38° C. It has a somewhat characteristic appearance when grown on gelatine, forming flattened colonies, which are semi-transparent and slightly thicker at the centre, where there is a kind of dimpling; the growth has the appearance of an irregular leaf, with lobes and crenated margins. Along the puncture track of the needle in gelatine cultures there is a growth of white globules or points (discrete or running together to form a streak), some of which may be surrounded by a feathery growth (Fig. 5). On potato this organism grows luxuriantly in the form of moist shining colonies. When grown anaërobically gas is formed.

A large dose, injected into the circulation of a guineapig, kills the animal within twenty-four hours, and the bacilli may be found in the blood in considerable numbers; but the condition is then of a toxic nature rather than of the nature of a true infection. If a small dose only is injected, the duration of the process is prolonged, and we have an affection much more like an ordinary septicæmia. When injected subcutaneously, or into the peritoneal cavity of rabbits or guineapigs, the quantity is a most important factor. With a small dose a local abscess may be produced, but the animal recovers; or peritonitis may be set up, from which the animal may also recover, even after a few organisms have been found in the blood. A large dose, however, gives rise to severe illness, accompanied by fever, diarrhœa, and great collapse; there is hyperæmia of the spleen and of the upper part of the intestine, evidenced by the reddening of the mucous and peritoneal surfaces. Large quantities of this organism injected subcutaneously into dogs, give rise merely to abscess formation.

The Bacillus coli communis is of special importance to the human subject from the fact that it is so frequently associated with peritonitis and suppuration in any region near the intestinal tract, down to the ischio-rectal fossa. Indeed, Fraenkel and others maintain that in cases of peritonitis, especially that form following wounds of the intestine, the inflammatory process is due to the presence of this organism in the abdominal cavity. That it is not the sole cause, however, is made clear by the fact that in certain cases the ordinary pus-forming organisms, or the Bacillus lactis aërogenes, and the Bacillus pneumoniæ have been found.

Fig. 5.—Cultivation of the Bacillus Coli Communis.

2. **Bacillus lactis aërogenes.**—This organism, of importance only from the pathological point of view because it has been found in peritonitis, is usually met with in the fæces of children and animals fed upon milk. It consists of short thick rods 1μ to 2μ long and 5μ to 8μ broad, with rounded ends, and is grouped in pairs or irregular masses.

In gelatine tube cultures it grows luxuriantly in a characteristic nail-shape. It forms soft, prominent, porcelain-like colonies on the surface of a plate culture, and round yellowish colonies in the depth of the gelatine. On potatoes it grows readily at the room temperature, but luxuriantly at that of the body, giving rise to the formation of gas—hence the name—and is usually white or creamy in colour. In milk, in which it sets up an energetic lactic acid fermentation, and in grape sugar solutions, it requires air, and gives rise to the formation of considerable quantities of carbonic acid gas and water. It does not form spores. It is associated with the summer diarrhœa of infants, and bears much the same relation to peritonitis, though in a modified degree, that the Bacillus coli communis does.

3. **Bacillus pyocyaneus.** — Bacillus pyocyaneus, or the bacillus of blue pus, was first found by Gessard in the blue pus from wounds. It is a slender bacillus, with rounded ends, about the same size as the bacillus of mouse septicæmia, but slightly thicker, and is usually so small that it may be taken for a coccus.

It grows rapidly at the room temperature. In a tube culture a funnel-shaped area of the gelatine rapidly becomes liquefied, and a bluish-green fluorescence gradually spreads through the remaining solid gelatine. This coloration of the medium is even better seen in plate cultures, the whole of the gelatine not liquefied assuming a light green colour. On agar-agar there is a moist greenish-white layer, the agar-agar becoming green and fluorescent (Fig. 6). On potatoes rusty-looking colonies are formed; these, when moistened with dilute ammonia, become green, or with dilute acids, red. A bluish colouring matter—pyocyanin—and a fluorescent greenish pigment have been separated.

These organisms, when injected in considerable quantity, either subcutaneously or intraperitoneally, are pathogenic for both guineapigs and rabbits, setting up an acute œdema, purulent infiltration, or purulent peritonitis, as the case may be, the bacillus being found in the blood and in the fluids drawn off from the affected areas. Given in smaller quantities, it sets up local suppuration; but the animal usually recovers, and remains protected against the action of considerably larger doses.

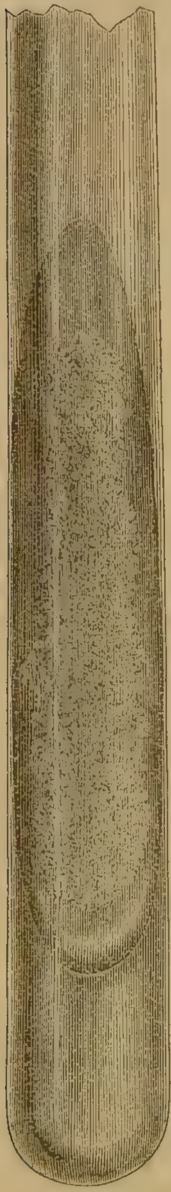

Fig. 6. — Cultivation of Bacillus Pyocyaneus.

4. **Proteus vulgaris.**—Another organism of some surgical interest is the Proteus vulgaris, one of the commonest of the putrefactive bacteria. It is a bacillus with rounded ends from $1{\cdot}25\mu$ to $3{\cdot}75\mu$ in length and $\cdot6\mu$ in breadth, occurring either as oval forms or as

more distinct bacteria, usually in pairs, or in longer straight or spiral filaments. There are also numerous involution forms. This organism is very motile, and possesses long cilia, and is probably the most important of all those organisms at one time included under the term *Bacterium termo.*

On weak gelatine the colonies have usually a yellowish-brown colour, and from the margins of the main mass of the colony branches forming a network shoot off in a characteristic manner, until the whole surface of the gelatine may be covered with zooglœa masses of considerable size. In tube cultures the liquefied gelatine is cloudy along the puncture, although a great part of the culture sinks to the bottom; eventually the whole of the gelatine becomes liquefied. On agar-agar, at the room temperature, a rapidly-growing, moist, greyish-white coating is formed, and a similar moist but much dirtier-looking layer on blood serum. Although spores are never found, the bacillus resists desiccation. It may also grow anaërobically.

A dose of $\frac{1}{20}$ cc. of a liquefied culture injected into the muscles is rapidly fatal to rabbits and guineapigs, half that quantity giving rise to an extensive local abscess, followed at a late period by the death of the animal, whilst $\frac{1}{500}$ cc. produces no effect. Watson Cheyne, who carried on these experiments, calculated that nine millions of these organisms produced no effect when injected into the muscular tissues of rabbits. Injection into the subcutaneous connective tissue—of a dose twice as large as that which proved fatal when injected into a muscle—gave rise to abscess formation, but did not kill the animal; and on recovering, this animal was found to be insusceptible to a similar dose injected into the muscles. Immunity has also been obtained by injecting filtered cultures of this organism. Cheyne points out, however, that if a large but non-lethal dose of the sterilised products of the bacteria be mixed with a small number of living bacteria an unprotected animal will succumb; from this he argues that the poisons devitalise the tissues, and thus enable the small number of bacteria to set up characteristic pathological reactions. This is a point of very great importance as bearing on the necessity of removing the effete matter by the various emunctory channels at regular intervals, wherever surgical operations have been performed. The *Proteus mirabilis* and *Proteus zenkeri* must be considered as having a similar action to the Proteus vulgaris.

5. **Bacillus œdematis aërobicus.**—Klein has obtained from garden earth a bacillus named by him the Bacillus œdematis aërobicus. It is about 1μ to 2μ in length and 7μ broad; it sometimes forms long filaments. It stains with aniline colours, but is decolorised again by Gram's iodine solution (page 51). It is comparatively unimportant, but must be mentioned, as when inoculated into guineapigs, rabbits, and white mice, the animals die very rapidly, often on the first day, suffering from a condition of subcutaneous œdema, the fluid from which is fatal when injected in quantities of a single drop.

Bacillus fœtidus ozænæ.—Bacillus fœtidus ozænæ is of interest principally from the fact that it is found in the nasal secretions of patients suffering from the disagreeable disease of ozæna, though it has not yet been proved that it has anything to do with that condition beyond mere association. The organism, separated by Hajek in 1888, consists of a short bacillus a little longer than broad, the elements of which are usually arranged in pairs or in short chains.

It grows on the ordinary media at the temperature of the room, the colonies becoming visible at the end of 36 hours, and giving rise to liquefaction and the formation of gas. On agar-agar plates the colonies have a granular appearance and the margins are fringed, the surface of the growth being moist and slimy-looking when in contact with the air. Grown on blood serum, at the temperature of the body, they form a whitish layer, from which is emitted a very disagreeable putrefactive odour. Its injection subcutaneously into rabbits and mice is followed by intense local inflammation, which is succeeded by spreading gangrene of the connective tissues. In the same year, 1888, Lumnitzer also separated an organism from a case of putrid bronchitis, which emits a disagreeable odour, and sets up intense local inflammation and a spreading gangrenous process.

The anaërobic bacilli.—The anaërobic bacilli, unable to live in the blood on account of the large quantity of oxygen therein contained, are nevertheless capable of multiplying locally in the subcutaneous tissues or in masses of fibrin; in fact, wherever oxygen is cut off from a localised area by disease processes. When these organisms have gained a foothold in any localised position, they are, like the diphtheria bacillus, able to manufacture and distribute their soluble poisonous products to all parts of the body.

1. **Bacillus tetani** (Plate II. Fig. 13).—The most important of these anaërobic bacilli is the Bacillus tetani, first demonstrated by Nicolaier in 1884, although it was not until 1889 that Kitasato obtained pure cultures by heating for one hour at 80° C. the pus taken from a wound. By this method all other organisms and spores, except those of the tetanus bacillus, were killed off. The spores of the tetanus bacillus were then sown in media placed in vessels from which all the air could be exhausted or driven out by means of hydrogen.

The organism is a thin straight bacillus with rounded ends, sometimes growing into long filaments; these divide into shorter rods, at the extremities of which spores are developed. These spores are usually rounded, but they sometimes assume an oval or oblate shape. They are almost invariably considerably larger than the diameter of the rod in which they are developed, and, as the end becomes distended by the large spore, the bacillus assumes a peculiar pin-shaped or drum-stick form, from which the organism derives its name "drumstick" bacillus. This bacillus is stained by Gram's method (page 51); the spores, however, require to be stained for a much longer time than the protoplasm of the rod, but when the stain is once taken up it is much more firmly retained, so that it is a comparatively easy matter to obtain a good double staining by first treating with fuchsin for a considerable length of time and then decolorising everything but the spore, in an acid, afterwards giving a contrast stain with methylene blue. (*See* Fig. 35.)

This organism only grows anaërobically, best at the body temperature in pure cultures, forming spores at the end of thirty hours; it grows much more slowly in gelatine, at a temperature of from 20° to 25° C., forming spores at the end of a week. The addition of 2 to 3 per cent. of grape sugar to almost any of the ordinary media enables the organism to develop fairly readily along the line of puncture, only below the surface, where a feathery growth is formed, which is always more luxuriant as the bottom of the tube is approached (Fig. 3). No gas is formed if the culture is pure. In peptone bouillon rendered slightly alkaline and kept in contact with an atmosphere of hydrogen, there is an abundant growth of the organism.

This organism retains its virulence under cultivation only so long as it is grown under anaërobic conditions; especially is this the case where there has been no time for spores to develop. Experiments made by inoculating pus from wounds from which patients have acquired tetanus may give entirely negative results in consequence of the fact that the bacilli have continued their existence under conditions unfavourable to the retention of their specific virulence. Tetanus may be produced in the smaller animals by inoculation of very small particles of nearly all cultivated garden earth, by inoculation of pure anaërobic cultures of the tetanus bacillus, by the inoculation of a pure culture *plus* the tetanus poison or *plus* lactic acid or Bacillus prodigiosus, or by inoculation into a bruised wound. It is difficult to produce tetanus with a young culture, and sometimes even with an old culture from which the poison has been washed away. This is due, apparently, to the fact that the tetanus organisms form their poisons slowly, and may be rapidly destroyed by the tissue cells before they have time to form sufficient poison to produce the nervous symptoms associated with the disease. In the cases above mentioned, the tissue cells are so occupied in removing the foreign matter introduced with the organisms, or are so paralysed by the action of the lactic acid, or of the tetanus poison, that they are not able to contend on equal terms with the tetanus bacilli, which, under the favourable conditions so presented, grow rapidly, and eventually the patient succumbs.

The tetanus organism being a facultative saphrophyte, can grow outside the body; it is found in the stable and in the field, so that tetanus is most common amongst those who work with horses, or on the land, and the people most susceptible are those in whom bruised wounds with which dirt may come in contact are most common—gardeners, agricultural labourers, soldiers on campaign, and young children.

The specific tetanus poison, which has been found in the blood and spleen of patients who have died from tetanus, and has also been obtained in considerable quantities from pure cultures of the tetanus bacillus, when inoculated into rabbits, guineapigs, or mice, or even into horses, produces a distinct intoxication setting up all the symptoms of tetanus. It resembles the enzymes in being destroyed at the comparatively low temperature of 65° C. in about 5 minutes, in 20 minutes at 60° C. and 1½ hour at 55° C. It is not affected by drying at ordinary temperatures, but in consequence of its instability in the presence of chemical agents, it is exceedingly difficult to remove it as a pure substance. Brieger and Kitasato and Weyl, however, have succeeded in obtaining a most virulent poison, in the form of yellow transparent flakes readily soluble in water, which does not give the reaction for albumens. It is not destroyed by drying, nor in the dried state by absolute alcohol, chloroform or anhydrous ether, but is injuriously affected by acids and alkalis, sulphuretted hydrogen and high temperature, thus resembling the tetanus poison in its original solution. This poison is so powerful that its toxicity may be represented as being 500 times as great as that of atropin and 120 to 400 times as great as that of strychnine. The tetanus bacillus has never been found at a distance from the seat of inoculation, whence then the absorption of the poison must go on, but when the extreme virulence of the tetano-toxine is remembered the deadliness of the process can be readily understood. The first experiments on the production of immunity against tetanus were futile, but Kitasato was able to produce a temporary immunity in rabbits by inoculating with the filtrate from a culture of the tetanus bacillus, and then injecting at the same point a small quantity—3 cc. of a 1 per cent. solution of terchloride of iodine—at intervals of 24 hours for five days. He then found that mice, in which he had previously failed to obtain any immunity, were rendered immune by the injection into the abdominal cavity of 0·2 cc. of the blood of the immune rabbit so obtained; this treatment proved efficacious, even after symptoms had become developed in the affected mice. The blood of an immune animal was also found to have the power of neutralising the tetanus poison; to such an extent has this been carried that 300 times the lethal dose of a virulent culture, after being mixed with serum from an immunised animal, produced no pathogenic results. Upon this rests the whole method of the preparation of immunising serum and the antitoxic treatment of tetanus. (*See* Art. IX. page 200.)

2. **Bacillus œdematis maligni** (bacillus of malignant œdema, vibrion septique).—This organism is of importance as being one of those so carefully studied by Pasteur (1877) and by Koch four years later. Like the tetanus bacillus it is found in the superficial layers of the soil, especially those soils containing a large quantity of organic matter, and in water that has been allowed to stand in pools near such soil. It is usually obtained by introducing a small quantity of garden earth into a pocket beneath the skin of a rabbit or guinea-pig, when there is developed in the course of 14 to 24 hours, according to the virulence of the organism, some swelling around the seat of

inoculation, in which a peculiar crackling associated with a condition of emphysema may be felt. On the death of the animal, which usually takes place within 24 or 48 hours, bloody serum is found infiltrating the subcutaneous and intermuscular tissue. In this serum the bacillus is found, but so frequently mixed with other organisms that the best method of obtaining a pure culture is to remove a drop of blood from the heart, under strict antiseptic precautions, and incubate it in a sealed tube for 24 hours, at the end of which time the blood has gone much darker in colour, bubbles of gas have developed at points along the tube, and when the tip of the tube is broken, drops of blood are forced out violently, indicating that owing to the formation of gas the pressure within the tube is very considerable.

These bacilli taken from the tissues are from 3μ to $3{\cdot}5\mu$ long and 1μ to $1{\cdot}1\mu$ broad, occurring either as single rods, in chains of two or three, or in long filaments containing twelve to fifteen of these rods or segments, the transverse divisions not being well marked. The bacilli differ from those of anthrax, in that they are not quite so broad, the segmentation in the longer chains is not nearly so distinct, and the ends of the rods are invariably convex. These organisms are not stained by Gram's method. The short segments often contain spores, which are oval and sometimes of greater diameter than the bacillus itself, so that they cause swelling either at the end or in the middle, according to the position of the spore. These spores may be stained in the same way as those of the tetanus bacillus. Flagella may also be demonstrated. This organism cannot grow in the presence of oxygen, and is therefore an obligate anaërobe; it grows best and forms spores most abundantly at a temperature of 37° C. Like the anthrax bacillus it does not form spores in the body of the living animal in which it is developed, but multiplies rapidly and forms spores readily after the death of the host, if the body is kept warm.

This organism is best grown in nutrient gelatine, to which has been added 2 per cent. of grape sugar. Like the tetanus bacillus it does not grow near the surface, but fairly luxuriantly near the bottom of the tube. As a result of this growth, the deeper part of the gelatine first becomes liquefied and cloudy in appearance, and gas bubbles form in considerable numbers, liquefaction being most marked where most gas is formed; ultimately the whole of the gelatine may become liquefied. When the gelatine is liquefied before the bacilli are added, small liquefying points are formed in the course of two or three days in the lower portion of the gelatine. These points resemble the liquefied gelatine already described, and when examined with a magnifying glass they are found to have running across them a fine network, whilst the periphery has somewhat the appearance of a chestnut "burr." The organism also grows in the depth of nutrient agar-agar and in blood serum, the latter medium being rapidly liquefied. This organism may also be grown in meat, to which a small quantity of water has been added, in a flask containing hydrogen. The odour of a culture of the malignant œdema bacillus is extremely characteristic, and once observed can never be forgotten.

It is pathogenic for nearly all the domestic animals, with the exception of cattle, but it is met with only as an accidental disease in horses and in man, developing in the latter in the form of what the French call "gangrène gazeuse." It is noticeable that pure cultures inoculated into a susceptible animal do not set up the peculiar formation of gas to the same extent as when earth, which no doubt contains a number of other bacteria, is injected. This organism, when thrown directly into the veins, is killed off, and the animal does not succumb. The most sure way of producing the disease is to inject a pure culture subcutaneously, or into the muscular tissues, especially if this is accompanied by an injection of lactic acid, or if there is any previous injury to the tissues. An animal that recovers from one attack of this disease is said to be immune; filtered cultures of the organism, or the filtered serum from immune animals, are said to produce a similar immunity; in both these cases, however, the inoculation must be repeated daily, for from three to eight days, 100 cc. of filtered culture being injected in three days, or 8 cc. of the filtered serum in eight days.

3. **Bacillus of black quarter.**—The bacillus of symptomatic anthrax, Rauschbrand bacillus, bacille du charbon symptomatique, bacillus of "blackleg" or "quarter evil."

This bacillus is not pathogenic to the human subject, but is important as being one of the anaërobic bacilli, and is usually studied in England as the cause of "black quarter" in cattle. Like the preceding organism, it sets up emphysematous swellings of the subcutaneous tissue, a process which often spreads into the intermuscular septa; it is found especially on the quarters, particularly where the subcutaneous tissue or muscle has been injured. On cutting into the darker-coloured affected area, gas and dark red serum exude in considerable quantities. In this serum is found a bacillus either single or in pairs, seldom in threads of any length. The individual bacilli have rounded ends, and are from 3μ to 5μ long and ·5μ to ·6μ broad. As in the other organisms of this group, flagella and spores may be demonstrated, the former by Loeffler's method (page 51), the latter by means of a prolonged staining with fuchsin at a high temperature. The oval spore is generally within the bacillus, and is placed near the middle of the rod, in some cases forming a considerable swelling, so that the bacillus becomes more or less spindle-shaped. This bacillus is not stained by Gram's method.

Like the other anaërobic organisms this bacillus grows best in nutrient materials to which a certain proportion of glucose—2 per cent.—or glycerine—5 per cent.—has been added; it will also grow on the surface of gelatine in an atmosphere of hydrogen, the colonies appearing as small rounded growths with irregular margins and surface; after a time the gelatine becomes liquefied, and rays shoot out from the central mass into the surrounding liquefied gelatine.

When grown in the depth of gelatine or agar-agar, gas may be formed in considerable quantities. The cultures have then a peculiar acid penetrating odour.

These organisms develop best at the body temperature, and the disease in cattle is therefore usually met with in summer, when the temperature is high. Spores are never found in the living host, but only at the end of ten to forty-eight hours after death. This disease, though occurring amongst cattle specially, may give rise to a slight local reaction in the horse.

The small experimental animals are not readily infected, but by the addition of a 20 per cent. solution of lactic acid to the inoculating fluid, animals which ordinarily exhibit little susceptibility to the disease succumb comparatively readily, and, as in the case of tetanus bacillus, by the addition of sterilised or non-sterilised cultures of the Bacillus prodigiosus or of Proteus vulgaris, the disease may be induced even in rabbits and mice. The guineapig is easily affected, and presents the same lesions as are found in cattle. The organisms here are found principally in the dark-coloured serum at and around the seat of inoculation, though cultures can usually be made from blood taken from the heart immediately after death, in which they multiply with great rapidity. This organism is readily attenuated by heat, and Arloing and Kitt have used powdered dried muscles from affected cases—these muscles having been dried at from 32° to 35° C.—as vaccine material. This powder, according to Kitt's method, is exposed to a temperature of 85° to 90° C. for six hours for the second vaccine, and the first vaccine is made by exposing for the same period to a temperature of 100° to 104° C., care being taken to prevent any moisture from gaining access to the powder. Inoculation in succession with these two vaccines sets up a local reaction, the animal recovers, and may then be safely injected with the most virulent material. Other methods of obtaining the same end have been used, but none are more convenient or more efficacious than the above.

Actinomycosis.—The actinomyces or ray fungus (Figs. 55 and 56) is found in the pus obtained from certain forms of abscesses of the lungs, the liver or other organs, and bones, especially those of the spinal column. The most commonly described form is found, especially in cattle, in the centre of firm fibrous nodules, this centre being usually softened or reticulated, the meshes surrounding softened caseous points which, on examination under the microscope, are found to be made up of a mass of cells in which is embedded a yellow or brownish point, composed of wedge-shaped rays, the bases of which are somewhat rounded, the whole mass having a peculiar mulberry appearance when seen in profile, and a conventional star-like appearance when seen in section. This ray-like fungus, however, is stated to be merely an involution form found in older tumours, the active fungus appearing in the form of cocci, arranged in chains, or leptothrix threads which interlace freely so as to form a kind of felted network in the centre of the colony, with a somewhat radiate arrangement near the periphery. These threads, somewhat larger in diameter than the cocci (the cocci are 0·5 μ), vary very considerably in length, being sometimes divided into short bacilli, or even cocci, whilst in other cases they are seen as long threads without any sign of division. Near the margin the threads may become branched, and very frequently, near the extremity, there is some thickening of the sheath of the coccus without, however, the club form being well developed. In other cases, again, the development of the club form is well marked. That these filaments are the active forms of the fungus was proved by Boström, who found that although the club-shaped peripheral portion of one of these masses gave rise to no growth on any of the culture media, those parts which contained numerous filaments were capable of being cultivated on ox blood serum and agar-agar, appearing first as fine granules along the line of inoculation; the growth as it becomes more marked develops into small yellowish-red nodules, around which delicate branched processes spread out. These yellowish masses soon run together, and at the end of seven or eight days are covered with a delicate fluffy white layer. This organism grows especially well under anaërobic conditions and at the body temperature. On microscopic examination, cocci, segmented threads, the longer threads and the clubs were all found, and when the threads or cocci were inoculated into the peritoneal cavity of rabbits or subcutaneously in calves, a typical actinomycosis was set up. The actinomyces lives outside the body, on barley and other cereals, from which it is supposed to make its way by the tonsils, where it has been found in the crypts, both in the human subject and in the pig. It may also be introduced through wounds, especially in horses and cattle that are cast and operated upon on a straw bed; even accidental scratches of the skin, mouth and pharynx may allow of the entrance of the organism, whilst it is thought that in certain cases it may make its way along the mammary ducts. The leptothrix form is that which is most frequently met with in the human subject. (*See* Art. XV. page 316.)

Mycetoma or Madura foot.—Closely allied to the actinomyces is the fungus found in mycetoma or Madura foot, which has recently been described by several writers in Great Britain, and has been cultivated by Surveyor and Boyce. It appears to be a streptothrix growing slowly on glycerine-agar at the body temperature; the form cultivated was from the white variety of the disease, and did not form pigment. There is true dichotomous branching of the filaments. To the naked eye the growth is a prominent, somewhat tough growth, which is divided almost like the ovum of a frog, first into four and then into eight or even more segments. Experimental inoculation gives rise to local irritation only. (*See* Art. XV.)

The Staining of Bacteria.

Summary of the various stages in staining cover-glass preparations.—Spread out the fluid on the cover-glass, and allow it to dry; draw the cover-glass three times through a flame; stain from one to three minutes in aqueous solutions of fuchsin, gentian violet, methylene blue, or Bismarck brown. Gentian violet gives good results, especially if carbolic acid, aniline water, or other reagents are employed to intensify its action. Loeffler's methylene blue and Gram's method are also very suitable.

Staining bacteria in sections.—Sections are transferred directly from absolute alcohol to the staining solution. Gentian violet is very suitable for most cases. As a rule, the same methods may be employed here as for cover-glass preparations, with certain modifications: (1) Sections should be left for a longer time in the stain; (2) heat should be applied while the staining process is being carried on, either in an incubator or by warming over the flame of a spirit lamp until vapour begins to rise; (3) when the nuclei have lost their colour, or become indistinct during the decolorising process, they can be brought out again by means of a contrast stain, such as lithium carmine, picrocarmine, or Bismarck brown; (4) excessive decolorisation during dehydration may be prevented by adding a small quantity of the stain employed to the alcohol—as a rule, however, this precaution is unnecessary, for if the sections have been well stained, a slight loss of colour is rather an advantage than otherwise; (5) clove oil should never be used for clearing the sections, as it dissolves out aniline dyes, and, to a certain extent, spoils the colour. Origanum oil is better, but xylol will be found best in most cases.

Gram's method.—Sections or cover-glasses should be kept in absolute alcohol, from which they are transferred to Ehrlich-Weigert gentian violet solution (aniline water, 100 cc.; concentrated alcoholic solution of gentian violet, 11 cc.; and absolute alcohol, 10 cc.), and left for one to three minutes (tubercle bacilli, twelve to fourteen hours). Wash for two to three minutes in alcohol, and then put into a solution of ten parts iodine and twenty parts iodide of potassium in three thousand parts water, until the dark blue violet is replaced by a dark purple red. Wash once or twice in alcohol, until most of the colour has disappeared; then clear up in oil of cloves, until the whole of the colour is washed out from the sections, and mount in balsam. Nuclei and tissues are stained yellow, and the micro-organisms, if present, deep blue or almost black. After the bleaching process the nuclei may be stained with eosin, vesuvin or Bismarck brown; then wash in alcohol, and mount in balsam, glycerine, or glycerine jelly. Aniline water is prepared by adding five parts of aniline oil to one hundred parts of water; shake well every half hour for three or four hours, and decant the water, as the oil settles to the bottom, or take the milky fluid and filter it, the filter being thoroughly moistened with water, so that the undissolved drops of aniline oil may be kept back; the fluid should come through quite clear and transparent. The commercial aniline is about half the price, but it is said not to answer the purpose so well. Aniline water soon spoils, and therefore should be used fresh.

Möller's method of staining spores.—The dried cover-glass film is passed three times through the flame, or placed for two minutes in absolute alcohol; then it is rinsed in chloroform for two minutes, washed in water, and put into a 5 per cent. solution of chromic acid for half to two minutes, after which it must be thoroughly washed. Now place a drop of Ziehl's carbolic fuchsin on the cover-glass, and heat for one minute over the flame, until it just begins to boil; then wash off the solution, and decolorise in 5 per cent. sulphuric acid. Wash thoroughly in water, and stain for thirty seconds in a watery solution of methylene blue or malachite green; wash again, dry, and mount in Canada balsam. The spores are stained dark red, and the protoplasm of the bacilli blue or green.

Loeffler's stain for flagella.—Make a potato broth, composed of two parts cooked potato, mashed and boiled in ten parts of distilled water. Carefully sterilise, and on this make a cultivation of the required organism. A drop of the culture is then diluted with from five to ten times its volume of distilled water. If the organisms will

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not grow on this potato broth, they may be cultivated in meat bouillon, which must be diluted forty or fifty times before it is used for microscopic examination; or on gelatine plates, the cultivations in this case being diluted about one hundred times. A drop of the diluted fluid is spread on a cover-glass; on this a drop of 10 per cent. alcohol is allowed to fall, and the whole is dried in the open air or in a warm room, at a temperature of 40° C. The bacilli are then mordanted in a solution made up as follows:—20 per cent. tannin solution, ten parts; cold saturated solution of sulphate of iron, five parts; aqueous or alcoholic solution of fuchsin, or alcoholic solution of methyl-violet, one part. To this mixture add a drop of hydrochloric, acetic, or sulphuric acid (in the case of those bacteria which give an alkaline reaction), or of an alkaline solution, say, 1 per cent. NaOH (in the case of bacteria which have an acid reaction). Put a few drops of this mixture on a cover-glass, heat until steam rises, wash in distilled water, and then in absolute alcohol, until all colour is removed. Repeat the process, and stain in an aniline water or carbolic acid solution of fuchsin, which has been neutralised by the addition, drop by drop, of a 1 per cent. solution of NaOH, until a layer of the previously transparent fluid, several centimetres thick, begins to become opaque. This will bring out flagella most beautifully. Success depends on adding just the right quantity of acid or alkali to the mordant.

II. INFLAMMATION.

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Definition.—Inflammation may be defined as the immediate series of changes which occur in the tissues as the result of an injury, provided always that the injury is not of sufficient violence to destroy the tissues at once. That is to say, whenever an injury is done to a part, whether the noxious agent be a chemical or a mechanical one, a certain series of changes commences in that part, and that series of changes, up to a certain point (so long as they are of an exudative or destructive character), forms what we know as the inflammatory process. The *degree* of the inflammatory change and destruction of the tissue will depend on the length of time that the inflammation goes on, and its severity and after-effects on the duration of the action of the irritant, and on the intensity with which it acts. I have especially said that it is the early series of changes occurring after an injury which are inflammatory, and if the injury be slight, or if the irritant only act for a very short time, the changes which deserve the name of inflammation soon cease, and the further processes which occur are those of *repair*. Inflammation precedes repair; but repair is not inflammation, and must not be confounded with it. Inflammation is essentially a destructive process, and when it ceases repair commences.

The phenomena of inflammation.—We see very well what happens in the early stage of inflammation if we watch the effect of a mustard poultice applied to the skin. If the poultice be removed after, say, ten minutes, the skin will be seen to be reddened over the whole area to which the irritant was applied; but if the finger be placed on any part of the red area, the redness at once disappears beneath it. If, on the other hand, instead of removing the mustard poultice in ten minutes it be left on, say for an hour, it will again be found that the skin has become reddened; but it will also be seen that on passing the finger over the reddened area there are points here and there from which the redness cannot be pressed away. Thirdly, if the poultice be left on for a longer time, it will be found that not only is the redness of the skin a redness which cannot be dispelled by pressure, but that blisters have formed on the surface.

In this observation we see the whole series of changes which occur in the early stage of inflammation. The first effect, the *redness*, shows that there is an increased amount of blood in the part, and that the blood-vessels are dilated; and the fact that the redness can be dispelled implies that this blood is circulating freely. The second effect, where *the blood cannot be entirely dispelled by pressure*, shows that in these parts the circulation is no longer going on, but has been arrested, this condition being known as *inflammatory stasis.* The third stage, where blisters have formed, implies that certain constituents of the blood have passed out and accumulated under the epidermis, thus forming a *blister.* Hence we see that in the early stages of inflammation changes take place, some of which have reference to the vessel walls and some to the vessel contents.

By studying the changes which follow irritation in a transparent living membrane, such as the frog's foot, one can see exactly what takes place. In the first instance, on the application of the irritant, one usually finds that the arteries commence to dilate, till, by-and-by, they may be as large, or even larger, than their accompanying veins. It must be remarked, however, that in some cases, especially after the application of certain chemical irritants, a preliminary period of contraction precedes this dilatation. In addition to this dilatation of the arteries—which affects more especially the small arteries—it will be seen that the capillaries, and also to some extent the veins, become distended, while at the same time the blood is circulating through the part with increased rapidity. If, however, the condition be watched, it will be found that presently the circulation in these distended vessels becomes slower and slower, so that after a time one can see the individual corpuscles passing along the channels; and ultimately, at the part where the irritant has acted most severely, the flow of blood becomes entirely arrested, and we have the condition spoken of as *inflammatory stasis* (Fig. 7).

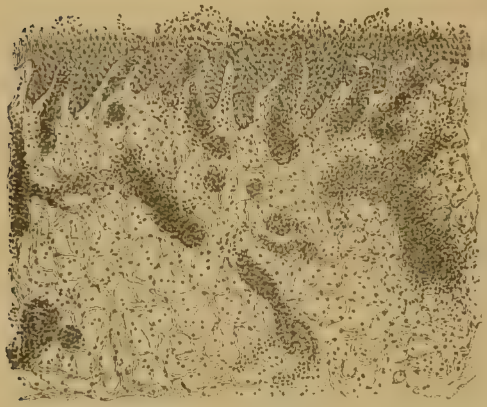
Fig. 7.—Section of Skin from Acute Traumatic Gangrene, showing stasis in all the blood-vessels which were gorged with blood, and in some places ruptured. There has also been a quantity of fluid poured out raising the epidermis (bullæ), which has at this part been lost. There has been no time for cellular infiltration of the tissues.

The **explanation of these phenomena** has been furnished by Sir Joseph Lister as the result of observations made a long time ago on the behaviour of the pigment cells in a frog's foot under the action of an irritant. On looking at the frog's web, one sees not only blood-vessels, but also pigment cells, which are numerous in the skin, and are present also in the deeper tissues, more especially along the blood-vessels. On watching these pigment cells, it will be seen that their protoplasm is in constant motion. At one time the protoplasm containing pigment granules has retreated towards the centre of the cell and formed a ball; in this condition the frog is light in colour. At another time the protoplasm will be seen rushing out from the centre into numerous ramifications of the cells, which were previously invisible, till the whole tissue becomes a network of this cell protoplasm containing the pigment granules; in this state the frog becomes dark. This action of the protoplasm of the cells seems to be a reflex phenomenon in connection with the access of light to the frog's eye. Bring the frog into a bright room, and at once the protoplasm of the pigment cells begins to retreat towards the centre of the cell and the frog becomes light. Shut out the light from the frog's eyes, and again the pigment-bearing protoplasm commences to hurry out from the centre of the cell into the various branches, and the frog becomes dark in tint. If the frog be killed the pigment always becomes concentrated towards the centre of the cells.

Now, if we take the frog into a light room and apply mustard to the frog's web while the pigment is in a state of concentration, and then either put it into a dark room or shut out the light from its eyes, it will be found that the pigment has become diffused over the whole of the body with the exception of the inflamed area, over which it remains in the same condition as it was when the irritant was applied. If, on the other hand, the irritant be applied while the frog is of dark colour, and the animal be afterwards brought into a light room, the pigment throughout the body becomes concentrated, while it remains diffused in the irritated area. If the irritant be removed recovery gradually takes place; the pigment begins to move in the cells in the affected area, more sluggishly it is true than elsewhere, but ultimately recovery becomes complete. Hence the condition as regards the pigment cells is that of a temporary suspension of their function—in other words, a *temporary paralysis;* and no doubt this condition of temporary paralysis is not limited to the pigment cells, but affects all the other fixed cells in the same area. Hence the most probable theory as regards the dilatation of the vessels in the early stage of inflammation is that at the focus of the inflammation it results from the direct paralysing action of the irritant. This, however, does not completely explain the phenomenon, because this dilatation of the blood-vessels is not limited to the focus of the inflammation; there is a similar condition extending for a considerable area around, an area greater than that directly affected by the irritant itself. This secondary dilatation of the blood-vessels is no doubt a *reflex phenomenon*, and hence the explanation of the dilatation of the vessels is two-fold, namely, in part direct paralysis as the result of the action of the irritant on the vessel wall, and, in part, a reflex paralysis through the nervous system. I need not go into the various other theories which have been put forward.

The *stasis*, also, may be explained in the same manner. It certainly is not due to any action of the irritant on the blood contained in the vessels, because stasis does not necessarily occur immediately after the lesion, and the blood, which was acted on by the irritant, has passed through the vessels of the part long before the occurrence of the stasis. Besides, substances such as chloroform, which interfere with the coagulation of the blood, nevertheless produce stasis when applied to the surface of the tissues. Further, it will be seen, on watching the inflamed area, that the corpuscles flow freely in the vessels up to the focus of the inflammation, and it is only when they arrive at that part that they acquire this tendency to stick to each other and to the walls of the vessels; and further, if one watches the vessels leading from the inflamed area, it will be seen that as the corpuscles escape from that part, they lose their adhesive tendency, and flow on perfectly freely in the circulating blood. Hence the cause which leads to the stasis is something which is permanently present in the part, and this stasis has been looked on by Sir Joseph Lister as another manifestation of the paralysis of the functions of the vessel wall: that is to say, blood when in contact with the walls of healthy vessels does not coagulate, either because the vessel wall has the power of actively preventing coagulation, or because it is absolutely neutral in the matter as compared with dead material, whereas in inflammation the walls of the blood-vessels being in a state of suspended function coagulation tends to take place within them.

It matters not which of these explanations is the correct one; the fact remains that when injured the vessel wall loses its normal characteristics, and comes to resemble dead material in so far that the blood tends to coagulate when it comes in contact with it, this loss of its function being due to the direct action of the irritant on the vessel wall, and not merely to the dilatation of the blood-vessels. As we can readily see, there is no tendency to stasis in those blood-vessels which are dilated in the vicinity of the inflammation in which the cause of the dilatation is not a direct paralysis of the vessel wall, but a reflex phenomenon. On the other hand, the stasis and the dilatation in the centre of the inflammation are probably due to the same cause, namely, to direct action on the vessel wall, this action not resulting, in the first instance, at any rate, in the death of the tissues, but simply in a suspension of function, which is recoverable again if the irritant do not act too long or too severely.

While these changes are going on in connection with the circulation of the blood, another set of changes are also taking place which have reference to the *contents of the vessels*, namely, certain constituents of the blood tend to pass out through the vessel walls and accumulate in the tissues around. This is the phenomenon of *exudation*, evidenced clinically by the occurrence of the blister when mustard has been applied to the skin, and the constituents which pass out of the blood-vessels are the blood corpuscles, both red and white, but more especially the white, and certain elements contained in the blood plasma. The passage of the blood corpuscles—more especially the white corpuscles—out of the blood-vessels during the early stage of inflammation, was demonstrated experimentally and attributed to their power of amœboid movement even before their migration had been actually observed.

I may mention one or two of these observations. For example, if the centre of a frog's cornea be touched with a fine point of nitrate of silver, it will be noticed that some hours afterwards a delicate, cloudy ring has formed around the margin of the cornea, and if the injury done have been considerable, this ring—which on microscopical examination is found to consist of cells resembling leucocytes—gradually approaches the injured part, and the whole cornea becomes cloudy. The conclusion, therefore, is not that these cells have been derived from multiplication of the corneal corpuscles—in which case they would not necessarily appear in the first instance quite at the margin — but that they are actually leucocytes which have passed out of the conjunctival blood-vessels at the margin of the cornea, and, therefore, accumulate in the first instance in largest numbers at that part. Further evidence of this is obtained by injecting finely-powdered cinnabar, in the first instance, into the dorsal lymph sac of the frog, and then, after some hours have elapsed, cauterising the cornea in the manner just mentioned. When cinnabar is injected into the lymph sac, the lymph corpuscles, which are present there in large numbers, take up the particles into their interior by virtue of their amœboid power, and can then be afterwards recognised by the presence of these particles in them. Now in this experiment it has been found that among the cells which appear around the corneal margin are some which contain particles of cinnabar, and which therefore must have come from the lymph sac, and consequently have passed through the walls of the blood-vessels. Lastly, if the cinnabar be injected into the blood stream instead of into the lymph sac, and the experiment be performed as before, it is found that the

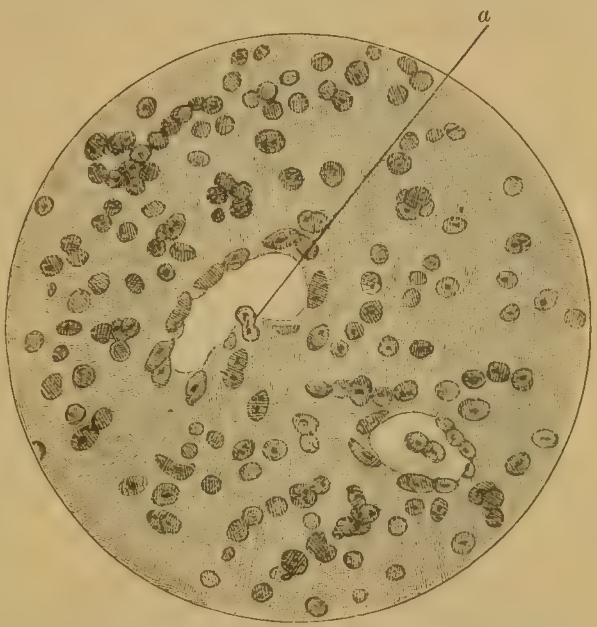

Fig. 8.—Section of Granulation Tissue from a suppurating Ulcer. At *a*, a white blood corpuscle is seen in the act of passing through the wall of a blood-vessel.

number of corpuscles in the cornea, which contain cinnabar, is much greater, and this is in accordance with what one would expect, seeing that a larger number of the leucocytes in the blood have had the opportunity of taking up these particles.

It having been demonstrated in this way that a considerable number, at any rate, of the cells which appear in the inflamed area during the early stage of inflammation, are actual leucocytes which have passed out of the blood-vessels, Cohnheim was the first to observe the actual migration of these corpuscles. This is done by spreading out the mesentery of a frog on a glass plate. As the result of the action of the air, it will be found that the early stages of inflammation are set up in the exposed part of the mesentery, and we have the various phenomena of dilatation of the vessels, and slowing of the blood current, which have been already mentioned. Now if, while the circulation is becoming slow, a small vein be carefully watched, it will be found that two currents can be distinguished in the vessel—a central current containing the red blood corpuscles, and a marginal current, in which are the white blood corpuscles. If these white corpuscles be watched, it will be found that they pass along much more slowly than the red, tend to stick to the walls of the vessels and to each other, and ultimately come to a standstill altogether, and thus the inner wall of the blood-vessel may after a time be seen to be lined with a layer of leucocytes (Fig. 8).

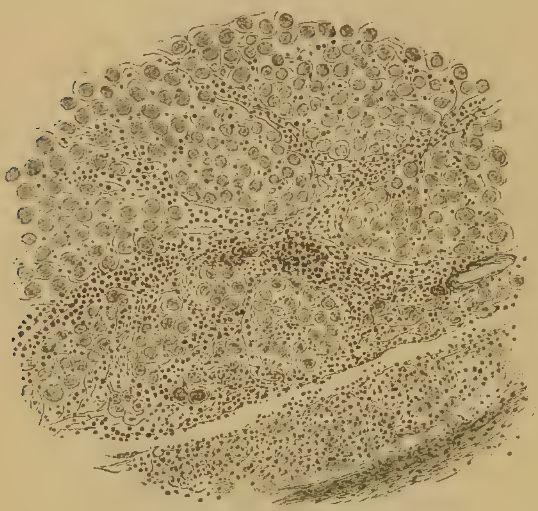

Fig. 9.—Early Stage of Inflammation of Muscle. The muscular fibres are separated by serous exudation (œdematous) and leucocytes are collecting around the blood-vessels. At one part the muscular fibres are being destroyed, and granulation tissue is being formed.

If the specimen be left for some hours, it will be seen on examining it again that not only is there a layer of white corpuscles inside the vessel, but that there are large numbers of similar cells outside the vessel; and one may find appearances showing corpuscles which have only partially passed through the vessel, and, as a matter of fact, the actual migration of the corpuscle has been observed after hours of careful watching. Once these corpuscles have passed through the vessel, they proceed by virtue of their amœboid action to wander away through the tissues, and thus, after a time, the whole surrounding district is seen to be infiltrated with leucocytes, although for a considerable time the largest numbers of these cells are found immediately surrounding the blood-vessels (Fig. 9). Not only do the white corpuscles migrate through the walls of the blood-vessels, but a certain number of red corpuscles also pass out, in some cases in considerable numbers. This migration of white corpuscles seems to go on most actively through the smaller veins, but it also occurs through the capillaries, and perhaps also through the small arteries. The exudation of the red corpuscles apparently occurs mainly through the walls of the capillaries. As to the explanation of the migration of these corpuscles, it is most usually assumed that the white corpuscles pass out by virtue of their amœboid movement, being to some extent helped by the blood pressure, and that the red corpuscles pass out solely as the result of that pressure. It may be remarked that this migration ceases when complete stasis takes place.

At the same time that this migration of blood corpuscles is going on, certain of the fluid elements of the blood pass out—sometimes in large quantities—giving rise to marked swelling of the tissues. This blood plasma coagulates outside the vessels, and consequently the resulting swelling is of a firm, hard character at the focus of the inflammation. Where the vessels are dilated reflexly at the margin of the inflammation, there is also a certain amount of exudation, more especially of fluid, from the blood-vessels, but this fluid does not coagulate, and consequently the parts around, instead of being hard and brawny, are œdematous, and pit on pressure.

The essential feature of this stage, therefore, is exudation, but the character and the amount of the exuded material vary much in different cases, and in some instances give a definite type to the disease. As I have said, the exudation in typical cases coagulates in the part, but in certain forms of inflammation this coagulation is only imperfect, and there is what is spoken of as a *sero-fibrinous* exudation; while in some cases coagulation does not take place at all, and we have a *serous* exudation. Where the inflammation affects a mucous surface, the exudation contains not only fibrin, but mucin and some dead epithelial cells (superficial layer), and is spoken of as a *croupous* exudation. In other cases, again, there is a large admixture of red blood corpuscles with the exudation, for example, where the inflammation is very intense, as in some forms of bacteric disease, or where the walls of the blood-vessels are very friable, as in cancerous pleurisy and so on, these exudations being spoken of as *hæmorrhagic*.

The terminations of inflammation.—So far, I have explained the phenomena of the early stage of inflammation, and when this stage has been completed, several things may happen. In the first place, the inflammation may come to a standstill, and, at once, changes leading to restoration of the part to a state of health take place, in other words, *resolution* occurs. By resolution, we mean the complete restoration of the part to its normal condition; the walls of the blood-vessels gradually recover, the stasis passes off, and the circulation becomes re-established. The exuded materials become re-absorbed, passing either into the blood-vessels, or most usually into the lymphatic vessels—those which have coagulated undergoing, in the first instance, a certain amount of fatty degeneration: the corpuscles either re-enter the blood-vessels or get into the lymphatic vessels, or undergo fatty degeneration, and are absorbed by the lymphatics in the form of *débris*, and in this way the part recovers its normal condition. In some cases, however, this resolution is not quite complete, as, for example, where the inflammation has attacked the surface of a serous membrane, such as the pleura or the synovial membrane of joints. There, the two serous surfaces are apt to become stuck together by the lymph, which acts as a mould into which plasma cells penetrate, these cells subsequently organising into fibrous tissue, and giving rise to *adhesions* between the serous surfaces, a well-known result for example in the pleura as pleural adhesions, and in the joints as a cause of stiffness (Fig. 10).

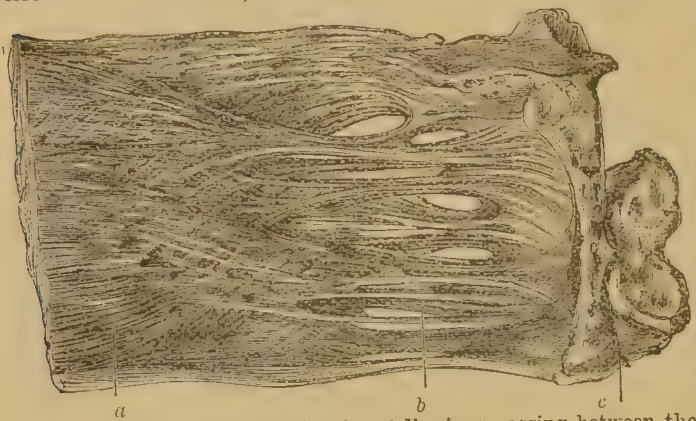

Fig. 10.—A Piece of Lung showing Adhesions passing between the two Pleural Surfaces.
a, Parietal pleura; *b*, adhesions *c*, lung.

In the second place, if the inflammation be extremely violent, stasis may occur over a very large area, and when the inflammation comes to a standstill a portion of that area does not recover, and we have the condition of *gangrene*.

In the third place, and most commonly, when the inflammatory changes have gone on to the stage described, they continue, and lead to the second or later series of inflammatory changes, namely, the destruction of the original tissue of the part which is the seat of the disease, and the formation of a new tissue, which is termed *granulation tissue* (Fig. 11). Where this occurs, it will be found, provided that the inflammation has lasted sufficiently long, that the original structure of the part has completely disappeared. If the inflammation be situated in muscular tissue, the muscular elements will be destroyed, and after a time nothing will be seen but a mass of round cells with embryonic blood-vessels. Similarly, if the skin be the seat of the disease, as, for example, in small-pox pustule, the papillæ of the skin are destroyed, and their place is taken by this same granulation tissue. In bone, also, the solid tissues of the bone disappear, and instead of a dense osseous structure, we find nothing but a mass of soft granulation tissue. This granulation tissue is, as I have just said, composed of a mass of round cells containing embryonic blood-vessels (Fig. 12), but when an inflammation has lasted for any time, these cells, more especially at the parts most distant from the focus of inflammation, have a great tendency to become elongated and spindle-shaped, and to develop into fibrous tissue; and hence, in examining granulation tissue, it is seldom that one finds—unless it be in the very early stage, or at the focus of the inflammation—that there are only round cells and embryonic blood-vessels. It is most common to find at other parts of the inflammatory growth that more or less complete fibrous tissue formation has taken place, and that the walls of the vessels have become thickened, and considerable numbers of them obliterated. This tissue is called granulation tissue wherever it occurs, because its structure is exactly the same as that found in the granulations or buds on the surface of a healing sore (Fig. 8).

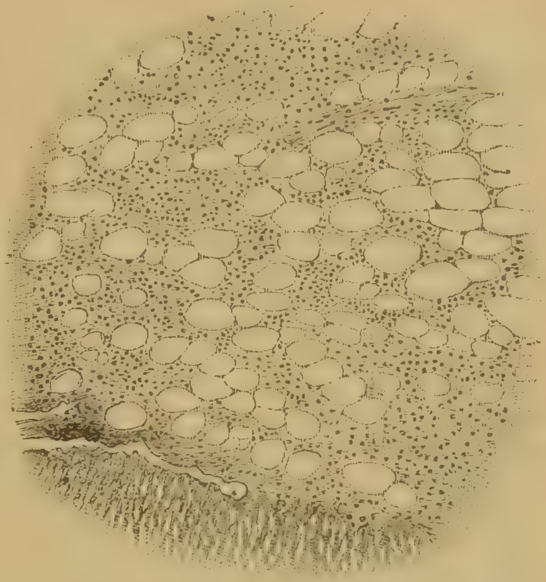

Fig. 11.—Inflammation of Fat. Cells are accumulating between the fat cells, which are being compressed and disappearing; at some parts only a young cellular (granulation) tissue is to be seen.

As regards the origin of these granulation cells, much debate has taken place. Formerly it was held that the cells which formed the granulation tissue originated entirely from proliferation of the connective tissue cells in the part; but when Cohnheim demonstrated the migration of the white corpuscles, many at once took up the view that these cells were leucocytes or derived from leucocytes which had passed out of the blood-vessels during the early stage. It is now, however, pretty generally held that leucocytes do not possess the function of forming permanent tissue, and that the cells

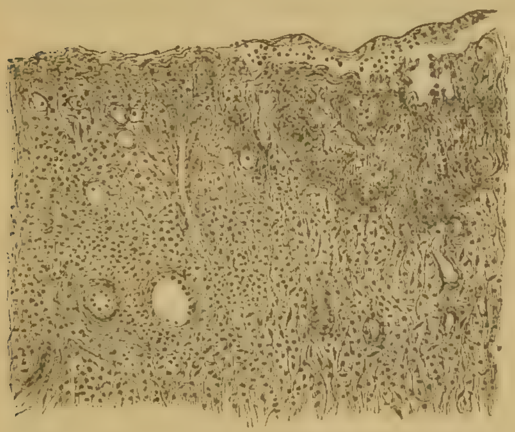

Fig. 12.—Section of Granulation Tissue, showing its cellular structure and the presence of numerous young vessels.

of the granulation tissue must be derived either by proliferation of the fixed connective tissue corpuscles in the part, or by proliferation of the wandering plasma cells, which, again, are apparently derivatives of the connective tissue corpuscles.

As regards the origin of the blood-vessels in granulation tissue, they apparently arise in the various methods in which they are formed in the embryo, more especially by budding from the endothelial cells of existing capillaries, which buds join others from neighbouring capillaries, become hollowed out, and form new channels.

When inflammation has passed on to the stage of complete destruction of the tissue and the formation of granulation tissue, various things may happen. In the first place, the cause of the inflammation may cease to act, and retrogressive changes take place in this granulation tissue, consisting essentially in the organisation of the granulation cells into more completely formed fibrous tissue. Where complex tissues are destroyed, there is no reproduction of the original tissue of the part (Fig. 9). There is, in fact, no real resolution of the disease. The result of the healing process is the formation of a *fibrous cicatrix*. This new fibrous tissue assumes the characters of the connective tissue in which it is formed; for example, where fat was previously present, it afterwards develops into loose, fatty, areolar tissue. Where denser fibrous tissue is present, as in tendons, the result is the formation of dense fibrous tissue; or where the inflammation occurs in bone, the new cicatrix becomes converted into bone.

A second thing that may happen where the inflammation has gone on to the stage of granulation is that the cause of the inflammation ceases to act with the same violence, but still remains in the part and keeps up the changes to a slighter degree, and we have the condition of *chronic inflammation* established.

Thirdly, and most commonly, where the inflammation has gone on to granulation as the result of a series of acute changes, its further continuance ends in *suppuration*.

Lastly, in cases where inflammation affects a free surface—especially of the skin—*ulceration* may be the result.

The causes of inflammation.—As regards the causes of acute inflammation, I need not here enter into particulars. As has already been stated in the definition, any injury to the tissues which is sufficiently severe will set up a certain degree of inflammation in the first instance, the exact stage to which the inflammation proceeds depending, however, on the length of time that the irritant continues to act and on various accessory circumstances. Inflammation, in fact, only continues so long as the cause or causes remain in action. Once these causes are got rid of, healing processes commence. Apart from the exciting causes of acute inflammation, a certain predisposition on the part of the tissues, or of the body generally, may be necessary in order that these causes shall act. We shall, however, have opportunity in treating of suppuration to refer more particularly to these causes.

The exciting causes of acute inflammation come, for the most part, from without, and they may be divided into two groups, from the point of view of treatment—namely, firstly those which are momentary in their action or are removable, and secondly those which cannot be readily got rid of. In the first group of cases we have such things as injury, which may be either of a mechanical or chemical nature. As regards mechanical injuries to the tissues, these are usually only momentary in their action, and it is seldom—unless they have caused actual death of a portion of the tissue, which may then act as a foreign body—that the inflammation produced by mechanical injuries goes so far as the stage of granulation. In most cases the inflammation subsides before this period has arrived, and resolution is the result. Chemical substances applied to the tissues, and constantly renewed, may carry on the inflammation to the stage of granulation; but it is comparatively seldom that these substances come into play in ordinary circumstances. Again, the presence of foreign bodies—such as bullets, pieces of dead bone or tissue, etc. —are also causes of inflammation under certain circumstances, and may lead to the destruction of the tissue and the formation of granulation tissue. With regard to the second group of cases, by far the most common cause of acute inflammation is the growth of micro-organisms in the tissues of the affected part; and as these micro-organisms generally carry the inflammation to a further stage than granulation—for example, to suppuration—we need not consider them here in detail. (*See* Art. III.) It is these micro-organisms which I have grouped under the head of irremovable causes, because, at the present time, once they have become established in an inflamed part, we have no certain means of getting rid of them.

The symptoms of acute inflammation.—The symptoms of acute inflammation are both *local* and *general.*

(*a*) **Local symptoms.**—The local symptoms are always described in text-books as four in number; namely, redness, swelling, heat, and pain. These four, when they occur together, always imply the presence of an inflammatory process; but, occurring separately, they do not necessarily possess this significance. For instance, *redness* alone may be due to dilatation of the blood-vessels, quite apart from any inflammatory change—that is to say, from any suspension of function in the blood-vessels—as occurs after division of the sympathetic nerve in the neck, where the blood-vessels of the ear become greatly dilated, and the ear itself becomes intensely red, but no other phenomena of inflammation result, because the vessel walls are healthy. *Swelling*, also, does not of itself indicate inflammation, because many swellings are due to new growths of various kinds; and the increased *heat* of the part and the *pain* are also not necessarily indicative of inflammation. When, however, the whole four symptoms are combined, it is almost certain that we have to do with an acute inflammatory process, the only exception being some very rare cases of sarcomata growing excessively rapidly.

In the first place, where the inflammation is superficial, the skin over the part is of a bright red colour, due, of course, to the presence of an excessive amount of arterial blood in that region, this *redness* being, however, much more extensive than the actual inflamed area, owing to reflex paralysis of the blood-vessels around. The redness of the ordinary acute inflammation also shades off gradually at the circumference, there being a gradual passage from the normal condition of the vessels beyond to the dilated condition of the vessels at the centre. It is only in certain specific inflammations—such as erysipelas—that there is a definite limitation of the reddened area. As the inflammation continues, this redness becomes darker towards the centre, owing to the greater difficulty which the blood there finds in passing through the capillaries, and its consequent increased venosity. There also the redness is, after a time, not only due to dilatation of the blood-vessels and increased quantity of blood, but also to actual pigmentation of the tissues, as the result of the passing out of the red corpuscles from the blood-vessels, their disintegration in the tissues, and the deposit of the blood pigment there. Consequently, when the inflammation has lasted for a considerable time, a certain amount of staining of the tissues will often remain for some weeks or months. When the inflammation subsides, the redness of the region also disappears; but this disappearance is for a time not complete, because the recovery of the tone of the vessels is somewhat slow. Hence for some weeks or months the part which has been the seat of an acute inflammation can be recognised from its increased vascularity after exertion, and also from the pigmentation of the skin, when the inflammation has been intense or long-continued.

An acutely inflamed part always gives the sensation of greatly *increased heat* to the hand; and it was at one time a question that was thought to be of importance, and was consequently much debated, whether this increased heat was actually generated in the inflamed part, or whether it was merely noticeable because there was such an increased amount of hot blood in that area. It seems to me that—taking into consideration the various experiments which have been made—it may be asserted that there is a slight, though very slight, generation of heat in the inflamed area, and that the blood which returns from that part is very slightly higher in temperature than that which comes to it. The matter, however, is one which, according to our present views, is of practically no importance, and an increased production of heat in the inflamed part is only what one would expect as the result of the active chemical changes which must be going on there.

The *swelling* which occurs as the result of acute inflammation in a part varies in character according to the portion of the inflamed area which is examined. In the centre of the inflamed area the swelling is hard and brawny, and it is not easy to press the finger into it. On the other hand, at the margin the swelling is soft, and the finger readily causes pitting. The explanation is that in the centre of the inflamed area the swelling is due to the presence of solid material which is deposited in the tissues, this material being in the main coagulated exudation from the blood-vessels, and in part, also, large numbers of inflammatory cells. At the margin, on the other hand, the cause of the swelling is the presence of a serous fluid in the tissues, which is readily driven along the interstices of the tissue under pressure by the finger.

Lastly, as regards the *pain*. This varies in character and degree, according to the seat of the inflammation. It is least where the inflammation occurs in loose cellular tissue; it is greatest where fasciæ and other dense tissues which do not readily yield to pressure are affected. This pain is of a throbbing character, and is much worse when the part is allowed to hang down, that is to say, when the distension of the blood-vessels is increased. It is mainly due to the pressure of the distended blood-vessels on the nerves of the part, these nerves being probably also themselves the seat of inflammatory changes.

(*b*) **General symptoms.**—A patient who is suffering from acute inflammation also presents, in most cases, certain constitutional symptoms, varying in degree and in character according to the extent of the inflammation, and according to the causes which have set it up. In the most ordinary or *sthenic form of inflammatory fever*, the patient complains of headache, his eyes are suffused, there is more or less complete loss of appetite, the tongue is furred, white and moist, the bowels are constipated, the urine is scanty and high-coloured, and the skin is dry. The temperature rises rapidly at the commencement of the inflammation, and on an average attains a height of about 103° F., and for three or four days this temperature of between 102° and 103° is maintained in cases where one has to do with a somewhat extensive, but not particularly dangerous, form of inflammation. Where the inflammation has gone on to suppuration, the temperature not uncommonly begins to fall or becomes oscillating, and, in any case, this fall takes place when the inflammation subsides. The pulse-rate is also increased, varying usually from 100 to 108, and the pulse is full, not easily compressible, and regular. In some cases where the inflammatory fever is high, or where the inflammation attacks the head, there may be delirium, which is generally of a more or less violent character, the patient shouting and tossing about.

This is the ordinary type of fever in cases that are not of a very serious nature, but in certain forms of inflammation the general symptoms assume a much graver character, indicating very grave depression of the patient's vitality. This form of inflammatory fever, which is spoken of as *asthenic fever*, or a typhoid state, may be present from the commencement, but most usually is preceded for a day or two by the sthenic type, which I have just described. In this asthenic form the temperature is higher and somewhat oscillating, the pulse, instead of being full and bounding, is soft, thready, easily compressible, and numbers from 120 to 150 the minute. The tongue, instead of being moist and white, is brown and dry. Delirium is almost always present, and is of a low muttering character, the patient lying in bed in a semi-conscious condition. This type of fever usually occurs in cases where the inflammation is very extensive, more especially where we have the form spoken of as diffuse cellulitis; where it rapidly ends in gangrene; where certain serous membranes, such as the peritoneum, are the seat of the disease; or in certain cases of acute inflammation of the periosteum and bone.

The cause of this inflammatory fever is no doubt due to the absorption of poisonous materials from the area of inflammation, and as in almost all cases where these general symptoms are present, the inflammation ends in suppuration or gangrene, these poisonous materials are most usually the products of the bacteria which are the exciting causes of the inflammation (page 18).

Treatment.—The treatment of acute inflammation resolves itself into local and general treatment.

(*a*) **Local treatment.**—In treating a case of acute inflammation, the first thing to be done is to ascertain, and if possible remove, the causes that are at work. If this can be done, the inflammatory process will at once come to an end, and very little further treatment will be required. I have already referred to the removable causes of inflammation, such as the presence of foreign bodies, the action of chemical substances, etc., and if these are known to be present and are accessible, they ought at once to be taken away. Practically, however, in almost all cases where we have the symptoms of acute inflammation which I have described, the active cause at work is the growth of micro-organisms in the tissues, and is, therefore, according to our present knowledge, irremovable, and all that we can do in the first instance in the way of treatment, is to attempt to render the growth of these organisms difficult, to minimise the effects they produce, and to modify any symptoms which are sources of danger or discomfort.

The most obvious symptom to treat is the congestion of the part, and if we succeed in diminishing the congestion, we succeed also in diminishing the pain and the amount of exudation. The congestion of the part may be diminished in a variety of ways. In the first place, an essential in the treatment of an inflamed part is to elevate it so as to favour the return of blood to the heart and diminish the venous engorgement. The advantage of elevation of the part is at once evident from the patient's sensations. If an inflamed part be allowed to hang down, the throbbing becomes very severe and the tissues feel as if they would burst; while if it be placed on a higher level than the heart, a sensation of relief is at once experienced. Another way of diminishing the congestion of the part is to reduce the rapidity of the heart's action by giving aconite, one or two minims in two or three doses with about an hour's interval between each, the patient being carefully watched; the result is that with the diminished frequency of the heart's beat a diminished amount of blood is sent to the inflamed part in a given time.

Again, by blood-letting one can diminish the local congestion.

and this is better done by local than by general blood-letting. The effect of general blood-letting is to lessen the vigour of the heart's action, and consequently to reduce the force and rapidity with which the blood, is sent into the inflamed part. It is possible, also, that where a considerable amount of blood is withdrawn, the composition of the blood-serum is altered in a direction antagonistic to the life of the micro-organisms, and thus the growth of the causal agents may be interfered with. The mode in which local blood-letting acts is not very clear, but it is probable that to a considerable extent the action is a reflex one, namely, that in emptying superficial vessels, reflex contraction of those which are going to the inflamed part occurs.

The forms of local blood-letting in acute inflammation consist of *leeching*, *cupping*, *scarification*, and *free incision*. The action of the leech is to saw through the surface of the skin in a triradiate manner and then to suck a quantity of blood from the part. The amount which a leech will usually take is about a drachm and a half, and once it has sucked its full, it generally falls off. It is important to remember that in the leech's pharynx a substance is secreted which has the power of preventing the coagulation of the blood, and in this way the flow of blood can go on. The importance of this point is with reference to the after-bleeding from leech bites, which is sometimes very dangerous and which may possibly in some cases be due to the presence of this secretion in the wound. In applying a leech, care must be taken to confine its movements within the area in which it is required to act (that area having been previously thoroughly cleansed), and if it does not quickly take hold, this will be promoted by pricking the part and drawing a little blood, or by rubbing a little cream on the surface of the skin. After the leech has filled itself, it usually drops off, but if it does not, the application of a little salt and water will produce that effect, and then the bleeding can be promoted for some time by the application of hot fomentations. When it is desired to stop the bleeding, the application of pressure is usually sufficient; but sometimes there is a great deal of trouble in arresting the hæmorrhage. If that be the case, it may be necessary to apply styptics, such as perchloride of iron, etc.; but generally it is sufficient to pinch up the skin around the leech bite, dry it thoroughly, and paint it over with collodion. If the bleeding persist, excision of the leech bite will usually stop it. On account of this risk of bleeding, leeches should only be applied on parts where pressure can be subsequently and efficiently employed—for instance, not to the eyelid or the scrotum—and also, the leeches should be used in the morning and not at night, so that if bleeding goes on it can be easily detected.

Cupping, either dry or wet, has practically the same effect. In employing wet cupping, the air in the cup is exhausted by a match or a piece of burning blotting paper, and the cup quickly inverted and closely applied to the skin. After it has acted for a few minutes, it is removed, scarifications are made over the part, and the cup is reapplied, care being taken that the scarifications should not go through the whole thickness of the skin, otherwise retraction of the vessels will take place, and satisfactory bleeding will not occur. Hæmorrhage can be subsequently promoted, if desired, by the use of warm fomentations.

The most useful method of local blood-letting in cases of superficial inflammation is the employment of *incisions*, which are of value not only in removing the congestion of the part by the bleeding, but also in permitting the exuded materials to escape, and thus diminishing the pressure on the blood-vessels and the consequent interference with the circulation of the blood. This method is of especial importance where the inflammation attacks dense tissues, and where the pressure of the exudation may completely arrest the circulation and lead to the occurrence of gangrene. For example, in cases of inflammation of the periosteum, it is of extreme importance to make early and very free incisions through the inflamed tissues, otherwise necrosis of the superficial layers of the bone is very apt to occur.

The congestion of the part may also be relieved by the employment of *cold*. Cold applied to a part causes contraction of the blood-vessels, and over an inflamed area it will lead reflexly to the contraction of the vessels going to that area. Cold, however, has a deleterious action in two ways. In the first place, it depresses the vitality of the part if too long applied and too intense, thus favouring the occurrence of gangrene; while, secondly, it leads to slowing of the circulation through the part, as shown by the blueness of the fingers where the hands have been long exposed to cold. While it is desirable to diminish the quantity of blood coming to an inflamed part, it is not desirable to slow the circulation; on the contrary, it is of advantage that the circulation should be active in the part in order that the deleterious materials may be quickly carried away and diluted in the blood, either by means of the blood-vessels themselves, or by means of the lymphatic system. Hence, cold must be very judiciously applied in acute inflammations, and as a matter of fact, its use should be mainly limited to the early stage of the affection; in the later stages it is very apt to precipitate gangrene.

There are various methods in which cold may be employed. Where the degree of cold required is not great, some of the evaporating lotions are quite sufficient. For example, an evaporating lotion composed of chloride of ammonium (half an ounce), rectified spirits of wine (an ounce), to eight ounces of water, acts extremely well, a piece of rag being placed over the inflamed area and kept constantly wet with this lotion. Another very good lotion is the lead and opium lotion, consisting of the dilute liquor plumbi subacetatis, with ten to twenty minims of tincture of opium to the ounce. Where more efficient cold is required, dry cold must be employed, because it is less apt to lead to gangrene. This dry cold may be applied by means of ice contained in an indiarubber ice-bag, a piece of lint being placed between the bag and the skin in order to absorb any moisture. If still more efficient cold be requisite, any desired degree is obtained by means of Leiter's tubes. These are thin leaden tubes arranged in coils, which can be moulded to the irregularities of the surface. A piece of lint is placed between the skin and the tubing, and then water of the desired temperature is passed through from one end in a continuous stream. These tubes are extremely efficient in keeping up the cold, and must therefore be carefully watched. It is well not to leave them on for more than two hours at a time, on account of the great depression of the vitality which results. They are also of advantage in cases of hæmorrhage.

Another plan of treating acute inflammations, which is of more universal applicability, is the employment of *heat*, which, like cold, is found to diminish the congestion of the part in certain cases, and thus to lead in some instances to resolution. The action of heat is not so clear as that of cold, but it also is probably in most cases a reflex one. Where the inflammation is quite superficial, and where the skin and the inflamed part are fed by the same vessel, it is conceivable that the dilatation of the superficial vessels of the skin over the inflamed area, as the result of the application of the heat, will lessen the supply of blood which can go to the inflamed part; but in most cases the vessels of the skin and of the inflamed part are independent of each other, and this explanation will not suffice. It is possible also that the effect of heat is to lessen the excitability of the deeper-seated nerves, and therefore to diminish the dilatation of the vessels leading to the part. Certainly, where heat is applied to the skin, the irritability of the nerves is reduced, as shown by the easing of the pain, etc.; and it is possible that this action is communicated to the deeper nerves, leading to diminution in the calibre of the afferent arteries. Heat also acts beneficially in that it increases the rapidity of the circulation through the part, and thus washes out the morbid products which tend to accumulate there.

Heat may be applied either by means of poultices or fomentations. Of these the poultice is no doubt the most effectual for the time being; but it is a decomposing vegetable substance, and should subsequent operation in that part become necessary, the thorough disinfection of the skin is a matter of great difficulty. Hence, in cases where poultices are used over a part which may be subsequently incised, it is well to combine them with some antiseptic substance, or to have them entirely composed of it, the best materials that may be used to impregnate the poultice being eucalyptus oil or boracic acid. Linseed meal is what is usually employed for poultices, and its advantage consists chiefly in the presence of the oil, but other materials will act almost as well if only oil is added. The poultice must not be too heavy—about four tablespoonfuls of linseed meal to half a pint of water—and everything must be as hot as possible, the water boiling, and the material on which the poultice is spread previously heated. Where a poultice is used in the case of superficial inflammation, it is applied directly to the skin; but in deeper-seated inflammations, where the poultice is applied at a higher temperature, a piece of muslin or lint must be interposed. Outside the poultice a mass of cotton-wool is laid, and the whole fixed on by a bandage. It does not usually require changing for from two to three hours. In cases where an operation will probably be subsequently required it is best to be content with hot fomentations. These are made by pouring boiling water on a piece of thick flannel, which is then wrung dry, and quickly applied to the skin; and outside it, overlapping it in all directions, a piece of mackintosh cloth, and outside this again some cotton-wool and a bandage. The disadvantage of the fomentation is that it requires to be changed more frequently; but in cases where pressure can be borne the fomentation or the poultice can be kept hot for a much longer time if an indiarubber hot-water bottle, containing a small quantity of boiling water, be laid over the part, outside the cotton-wool.

(*b*) **General treatment.**—As regards the treatment of the general condition, the indications are to keep up the strength of the patient, to dilute the poison in the blood, to lead to its excretion from the blood, and to treat any special symptoms which may arise. Of these, the most important is to attend to the restoration of the excretions, and this should be done by the administration of purgatives, more especially sulphate of magnesia; by the use of diuretics, the best of which is probably bitartrate of potash, usually given in gruel—one tablespoonful of oatmeal, half a pint of water, a drachm of cream of tartar, and a little sugar and brandy, or spirit of nitrous ether; and by sudorifics, such as liquor ammoniæ acetatis, or Dover's powder. The purgative acts not only in expelling the excretions, but in leading to transudation of a quantity of serum from the blood, and it probably acts also as a counter-irritant leading reflexly to diminution of the inflammatory condition; it should be administered in every case, even although the bowels are acting. The poison should also be diluted in the blood by allowing the patient to drink large quantities of fluid, which he is always very ready to do. This fluid should, of course, be as much as possible of a nutritious character, such as milk, barley-water, weak beef tea, etc. A good fluid is also the "Imperial" drink, which is composed of a drachm to a drachm and a half of cream of tartar, with a little sugar and a little lemon-peel to the pint of boiling water. As regards the food during the acute stage of inflammation, it must consist essentially of milk and fluid preparations of meat (the patient can digest nothing else), and if he gets plenty of milk (four to six pints a day), he will obtain sufficient nutriment for the time being. Stimulants are not usually necessary or desirable. As soon as recovery commences, the patient will require as nourishing a diet as he is able to digest, and he will then in all probability be the better for a little stimulant.

Prognosis.—As regards the prognosis in cases of acute inflammation, questions which are constantly asked by the patient are:

How long is the trouble going on? And does it show any signs of improvement? These are difficult questions to answer; but we may take it that if an acute inflammation lasts for more than three or four days, it will almost certainly go on to suppuration: if, on the other hand, it is noticed after three or four days that the skin, instead of being tense and shiny as the result of the great distension from the exudation, becomes wrinkled, the inflammation is subsiding, and will probably do so without the occurrence of suppuration.

Chronic inflammation.—So far I have been speaking of acute inflammation, but we have also to consider another inflammatory process which is much more difficult to describe and understand—namely, chronic inflammation. By this, we mean an inflammation which runs its course slowly, and which is not accompanied by any severe local or constitutional symptoms.

Causes.—The causes of chronic inflammation differ from those of acute, more especially in the fact that it is not due to the ordinary pyogenic organisms, but is most commonly the result of some other morbid process which is going on in the tissues. At the same time, the presence of a foreign body in the part, such as a bullet, or a piece of dead bone, etc., may keep up this condition of chronic inflammation for an indefinite time. The retention of pus after the opening of an abscess—where the opening has been too small—will also keep up a condition of chronic inflammation in the surrounding tissues. Similarly, the obstruction to a duct of a secreting gland, such as a salivary gland, will set up chronic inflammation in the substance of the gland from the pressure of the retained secretion. Again, chronic inflammation is kept up by certain conditions of the blood, as in rheumatism, or by deposits from the blood, as in gout. By far the most common cause of chronic inflammation is, however, the presence of an infective disease, more especially tubercle or syphilis. The greater part of the swelling which occurs in tuberculous diseases is due to chronic inflammation of the tissues surrounding the tubercular deposit, and not to the tubercular deposit itself; and herein lies the difficulty in understanding or describing chronic inflammation by itself as apart from the disease with which it is associated.

Processes.—The processes which go on in chronic inflammation are in their essence the same as those which occur in acute inflammation, except that they progress very much more slowly, and extend over a longer period. These processes consist in the gradual destruction of the tissue in which the inflammation is occurring, and its replacement by young connective tissue. In chronic inflammation one does not meet with typical granulation tissue, the multiplication of the cells goes on very slowly and they very quickly undergo organisation (Fig. 13), and the ultimate result of chronic inflammation is an increase in the connective tissue of the part which is the seat of the inflammation, leading in the first instance to great thickening of the part (for example, in chronic inflammation of bone), and subsequently, in some instances, as in cirrhosis of the liver, to marked contraction and diminution in the size of the organ (Fig. 14). Chronic inflammatory conditions of themselves do not go on to suppuration.

Symptoms.—As regards the symptoms of chronic inflammation, there is no constitutional effect from the inflammation *per se*, unless it attack some vital organ, any general trouble which results being due to the other morbid process, which sets up the inflammatory condition, such as tuberculosis. Of the local symptoms, the most marked is the swelling of the part, which may in some cases attain great size, more eepecially in cases of chronic inflammation of the periosteum and bone. This increase in size is, as I have already mentioned, sometimes followed, after the disease has disappeared, by marked diminution. As regards the second cardinal symptom of inflammation, pain, that will vary according to the seat of the inflammation. In some cases the pain is only very slight, in others there is a good deal, and it depends on whether nerves are involved in or pressed upon by the inflammatory new growth. In the case of bone, for example, there is generally a good deal of pain of an aching character, worse at night. In chronic inflammation of the soft tissues, such as the breast, the pain is of a more neuralgic character, due to the compression of the nerves. In most cases there is, however, a certain amount of tenderness on pressure. There is always some increased heat over a chronically inflamed area, as compared with the opposite side, and naturally, also, the part which is the seat of the inflammation is more vascular than the healthy tissues; at the same time, unless the inflammation affects the skin itself, there is no redness. The skin at the seat of a chronic inflammation, however, may be of a somewhat duskier colour than elsewhere, and the

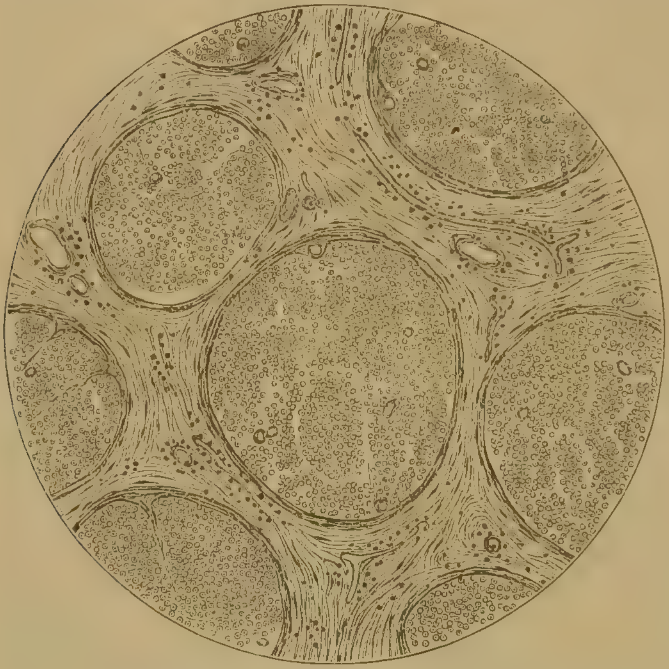

Fig. 13.—Section of Nerve from a Stump, showing the great Increase of the Fibrous Tissue as the result of Chronic Inflammation. This new tissue is very vascular, and as it contracts presses on the tubules, leading to their atrophy.

increased vascularity of the part may be shown by the presence beneath the skin of enlarged veins.

Treatment.—The continuance of chronic inflammation being entirely dependent on the presence of the irritating cause, the first essential in the treatment is to ascertain what the cause is, and to get rid of it if possible. Where the inflammation is due to a foreign body, such as a bullet or a piece of dead bone, naturally the treatment is to open up the part and to remove the offending agent. Where the inflammation depends on some constitutional condition, such as rheumatism or gout, the removal of the cause must be attempted by means of suitable constitutional remedies. Where, however, the essence of the trouble is some local infective disease, such as tubercle, the question of the removal of the cause must depend upon various circumstances, which will be discussed in speaking of these affections in different situations. In cases where the cause cannot be ascertained, or cannot be removed, various methods of treatment may nevertheless be employed, with the view of diminishing or even arresting the inflammatory process, and the first and most essential of all the methods is to place the part completely at *rest.* This rest should be in bed, especially if the chronic inflammation affects the lower part of the trunk or the lower extremities, and the affected part should, as far as possible, be placed at a higher level than the heart. In cases where the chronic inflammation affects the upper extremities, it is not so essential to place the patient in bed, provided sufficient rest is obtained by suitable forms of retentive apparatus. The patient may in that case be allowed to go about, and thus obtain the benefit of fresh air and exercise, which may be of great value in getting rid of the cause of the disease.

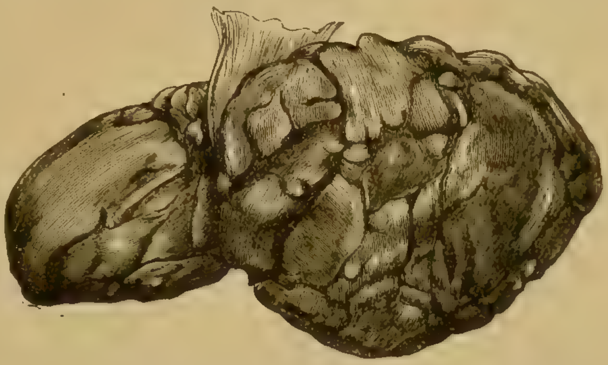

Fig. 14.—Cirrhosis of the Liver, showing Diminution in Size; and Depressions resulting from the Contraction of the Fibrous Tissue formed in the course of Chronic Inflammation.

The second great principle in the treatment of chronic inflammation is the employment of *counter-irritants,* of which we have a considerable variety. The action of these counter-irritants is by no means clear. The most generally received theory is that, on the one hand, by causing congestion and inflammation of the skin, they lead reflexly to diminution in the vascularity of the inflamed part; and on the other hand, by the irritation of the terminal ends of the nerves in the skin, they in some way or other alter the nervous balance, and lead to some salutary change in the innervation of the diseased part. Experiments have undoubtedly shown that where the skin is severely irritated, the vascularity of certain parts beneath, which are in some sort of special nervous relation to the irritated part of the skin, is diminished. Thus it has been found that, where the skin over one side of the chest has been severely blistered, on making a transverse section of the thoracic cavity the lung on that side is distinctly anæmic as compared with the lung on the opposite side.

Whatever the explanation of the action of the counter-irritant may be, it nevertheless remains as a clinical fact that this kind of treatment is of great value in many cases of chronic inflammation. The counter-irritants usually employed produce various degrees of action on the skin. The mildest counter-irritant is a poultice, but in the treatment of chronic inflammation, the counter-irritation produced by a poultice, whether of linseed or mustard, is seldom efficacious. Nevertheless, in certain cases of chronic mastitis the continued application of poultices over the inflamed breast will sometimes apparently hasten the disappearance of the inflammation.

The forms of counter-irritants now most usually employed are in order of efficiency—the actual cautery, blisters, and iodine. In former times other methods were used, such as tartar-emetic ointment or croton oil (applied to the skin), the employment of the seton or of the moxa; but nowadays these forms of counter-irritation are for the most part discarded, and reliance is placed on the three to which I have referred. Of these, the least efficacious is iodine, which, indeed, unless in superficial inflammations, does not as a rule seem to exercise any action. It is a substance which is very commonly employed in chronically enlarged glands in the neck; but, beyond diverting the attention of the patient and his friends, it does not seem to possess any real salutary effect.

Much more effectual than iodine is the employment of blisters or blistering fluid; and undoubtedly a considerable number of cases of chronic inflammation—especially those dependent on constitutional conditions—are improved by the use of this remedy. In blistering the skin for chronic inflammation, the blister should not be applied directly over the inflamed area, unless, indeed, the inflammation be deep-seated. It should be placed a little away from it, but over the area of nerves which are in direct relation to those which run to the inflamed part. That there is a distinct connection between the distribution of nerves in certain parts of the skin and their distribution to deeper organs has been very well shown by Dr. Head in his papers on this subject, more especially by the observations which he has made as to the relation of attacks of herpes zoster to disease of deeper-seated organs. Where the chronically inflamed part is situated near the skin, the application of a blister over it is very apt to increase the congestion at the seat of the inflammation, and thus, if used while the disease be advancing, the treatment may lead to its more rapid progress. Where, however, the inflammation has passed off, or has nearly done so, and where there is still a considerable amount of thickening, the application of the blister directly over the part may be of greater value than if used at some little distance away, from the very fact that it increases the activity of the vascular and lymphatic circulation through the thickened tissues, and thus aids the rapidity of absorption of the deposited materials. For example: in the treatment of callous ulcers a blister applied over the thickened tissues around the ulcer is one of the most rapid methods by which the disappearance of the exuded materials can be brought about.

Where a blister is applied to a part, it should as a rule be left on for about ten hours; and if at the end of that time it does not seem to have produced sufficient effect, its action may be increased by placing a poultice over the part. This may also be effected by rubbing a drop or two of croton oil over the surface of the blister before its application; or, again, where the effect is very slight, blistering fluid may be painted over the region where the blister had been previously applied. As a rule, however, where the blister is applied over the skin of the limbs or of the trunk—that is to say, over parts where the skin is thin—ten hours are sufficient to produce a satisfactory result. It is mainly in cases where blisters are applied over thickened skin—as over the knee, where there has been much kneeling, or about the hands or feet—that there is trouble in obtaining the necessary irritation. After the blister has risen, it is not, as a rule, necessary to puncture it. A piece of thin muslin on which boracic ointment has been spread may be applied over the blister, and outside that a mass of salicylic wool. If, however, the tension of the blister be causing much pain to the patient, there is no real harm done in puncturing it before applying the ointment and wool as just mentioned, unless in very weakly persons. In most cases where blisters are employed in chronic inflammation, a single blister does not suffice. The blisters must be repeated at least three or four times before one can feel satisfied that no good is to be done by this method of treatment; and therefore the directions usually given are that as soon as the first blister is healed—say, within a week—a second blister should be applied, which, it may be remembered, will probably rise more quickly and more effectually; and after the second blister has healed, a third may be used. In children, of course, where the skin is very thin, the blister should not be kept on so long; otherwise it is apt to produce actual sloughing of the skin.

Still more effectual than blisters in cases where the chronic inflammation is deeply-seated, more especially in deeply-seated inflammation of bone, is the use of the actual cautery. The value of the actual cautery has been variously estimated by different surgeons, some, indeed, stating that it is of no value whatever; but certainly, in my own experience, it has proved in many cases to be of the greatest benefit, and probably the inefficient results obtained by others may be in part due to imperfect application of the method. In employing the actual cautery for chronic inflammations, two methods are used—in the one the cautery iron is small and round (this is termed a button cautery) and a number of small burns are made. In the other, which is much more effectual, the flat cautery, which is large and flat, is used, and with it one, or at most two, large sores are produced.

In applying the cautery, the skin should in the first instance be shaved for a considerable distance around, otherwise the hairs are very apt to prove a source of annoyance after the eschar has separated. The cautery itself should be at a white heat, and in the case of the flat cautery the iron is passed twice or three times over the region to be treated. If the cautery be not at a white heat, one has no guide as to the degree of burning that will be produced. We know that with a white-hot cautery a sufficient effect will be produced by passing it over the part two or three times; but where it is only red-hot, one is very apt to do either too much or too little. The same rule applies to the button cautery, which, however, is not rubbed over the part, but is simply pressed on it. After the application of the cautery, poultices—which had better be aseptic poultices—are applied for a few days till the slough separates, and the best effect is produced where only about half the thickness of the skin is destroyed. Less good is done where the whole thickness of the skin sloughs. On the other hand, where only half the thickness of the skin is destroyed, the sore is, no doubt, a more painful one, because many delicate nerve terminations are exposed; but, as I have said, the effect is also greater.

After the eschar has separated, the sore should be prevented from healing for from four to six weeks, and this is usually done by the employment of savin ointment. The ointment is spread thickly on muslin, and changed once or twice a day. Many patients, however, complain greatly of the pain of this application, and this is more especially the case where only a superficial portion of the skin has been destroyed; and in these cases the ordinary savin ointment should be diluted with two or three parts of vaseline. In some cases, indeed, the pain is so severe that this ointment cannot be employed at all; and in these instances all that one can do in the way of preventing healing is every day or two to touch the surface of the sore with nitrate of silver wherever epithelial formation is evident; and where this is not sufficient, and healing occurs in spite of it, the sore may be opened up by the use of potassa fusa. At the end of about six weeks the sore is allowed to heal.

Another method which is of great value in the treatment of chronic inflammations is the employment of *free incisions* into the inflamed part. Where these inflammations are not dependent on some specific disease, such as tubercle or syphilis, simple incision into the inflamed part, or, if it be the wall of a sac secreting fluid, drainage of the sac will usually suffice to arrest the inflammation. This is more especially the case where the seat of the inflammation is the periosteum or the bone. In cases of chronic periostitis and osteitis, by far the most efficient treatment is to cut down very freely on the seat of the disease, divide the thickened periosteum, and, in fact, dissect away most of it, and then gouge a trough in the thickened bone. Of course, this operation must be done antiseptically, otherwise very serious results may follow; but in most of these cases of chronic periostitis and osteitis, it is the most rapid and often the only method of cure.

Among other methods of treating chronic inflammation, I may mention *pressure* and *massage;* but these plans are more especially applicable to cases where the actual inflammatory process has passed off, and where we have chiefly to do with residual thickening of the tissues. In employing pressure, care must be taken to regulate it carefully, because if applied too strongly where the inflammation has not perhaps completely subsided, it is very apt to set up the process again. Pressure over an inflamed part may be produced in various ways, the most effectual of which is to wrap the part first in large masses of cotton-wool, or, better, silk-waste, which retains its elasticity longer, and then apply the pressure either by means of an elastic bandage or by means of a silicate dressing, which hardens and keeps up constant pressure. By using a large mass of cotton-wool, a considerable amount of pressure can be employed without any material danger of irritation, because the cotton-wool distributes the pressure equally over the whole of the affected area. Perhaps the most favourite method of employing pressure in chronically inflamed joints, is by the use of Scott's dressing, in which we have not only the effect of pressure, but also the effect of counter-irritation. In applying Scott's dressing, chamois leather on which is spread the compound ointment of mercury is cut into strips, and these are put on like ordinary strapping, the strips overlapping each other, and the pressure being as equably applied as possible. I have not, however, myself seen any advantage in the use of Scott's dressing over elastic pressure through a mass of cotton-wool.

Massage, with the view of getting rid of the products of inflammation, is a very valuable method, but I do not think that it is judicious to employ it in parts where the inflammation is actually going on. The object of massage is to break up the exudation which has been poured out and to force it into the lymphatic vessels; and with this view, four chief actions are employed, which, to use the French names, are effleurage, friction, pétrissage, and tapotement. By effleurage is meant light rubbing of the part. Its object is simply to increase the lymphatic circulation. This is the form of massage which is employed in the first instance in cases of severe sprains, the part being gently pressed or rubbed, the hand always passing in a direction towards the trunk: that is to say, always forcing the effused material into the lymphatics. This form comes into play in all the others as the final action with the view of forcing the products which are broken up by the other methods along the lymphatic vessels. In friction massage, the material is broken up by rapidly rubbing the part, more especially in a circular manner, with one hand, while with the other hand the broken-up inflammatory materials are steadily pressed upwards along the lymphatic vessels. By pétrissage is meant kneading of the thickened parts, and this is of especial value in cases where there are adhesions, especially among tendons and muscles. The muscle is grasped with the spread-out fingers, squeezed, and kneaded, the action always commencing at the distal part of the muscle and gradually passing towards the upper end, and being carried out with both hands at the same time; subsequently to the kneading, effleurage is employed in order to drive the broken-up material into the lymphatics. The fourth method is used where the particles are still more difficult to break up, and in it the thickened tissues are tapped with the fingers, in the first instance lightly, and afterwards more forcibly, this attempt to break them up being followed by kneading and effleurage.

As regards the *constitutional treatment* in cases of chronic inflammation, there is very little to be said, because the constitutional treatment depends essentially on the disease which is the cause of the chronic inflammation. In most cases the patient ought to be supplied with suitable nourishing food, plenty of fresh air, and exercise as far as is consistent with the requisite rest of the part.

III. SUPPURATION.

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In the previous article the subject of acute inflammation up to the point of granulation was considered. The present chapter is connected with the study of what happens when the inflammation continues after this stage: namely, with the consideration of suppuration.

By acute suppuration we mean an inflammatory process which goes on to the production of a fluid, which is termed pus. This process of suppuration may occur either on a free surface or in the depths of the tissues; and it is with the latter that we shall deal in the first instance. Where acute suppuration occurs in the substance of the tissues, it may assume two aspects: in the one the pus is found in a closed, fairly well-defined cavity, the circumscribed acute abscess; in the other the pus infiltrates the cellular tissues without any proper circumscription—the condition known as diffuse cellulitis.

I. CIRCUMSCRIBED ACUTE ABSCESS.

Pus.—A circumscribed abscess is a cavity containing a fluid which is called pus, the surrounding wall being composed of embryonic granulation tissue. Pus is an albuminous fluid, which is full of innumerable small corpuscles resembling in size and character the white corpuscles of the blood, these corpuscles being termed the pus corpuscles. Under the microscope these *pus corpuscles* generally contain lobate or tripartite nuclei, and in addition to these corpuscles some granular matter may be noticed in the fluid. To the naked eye, normal pus presents the appearance of a creamy or yellowish-white fluid with an animal smell, a specific gravity of about 1030, and, some say, with a musty or sweet taste. Pus presenting these characteristics was formerly spoken of as *laudable* pus; but we meet with several varieties of pus in varying circumstances. For instance, where it is mixed with blood, and especially where it is thin, due to the smaller number of corpuscles which it contains, it is spoken of as *sanious* pus. Again, it may contain various other materials, giving it special characteristics. For example, the pus that comes from the liver in cases of liver abscess is of a reddish-yellow instead of a yellowish-white colour, and not unfrequently contains bacilli or amœbæ, and the knowledge of this character of *liver pus* may be of considerable importance in cases of difficult diagnosis. Then, again, pus which comes from *bone* generally contains large quantities of fat globules, and in other cases of bone abscess the fluid evacuated at first may be comparatively clear, and then towards the end a quantity of turbid fluid is pressed out. In the latter case the pus is strongly acid, from the presence of lactic acid, and it is the action of this acid on the pus corpuscles which produces the effect described. Again, the pus on the surface of a wound may be of a bright blue colour, the so-called *blue pus*, due to the growth in it of a particular micro-organism called the bacillus pyocyaneus (page 3). Or in other cases the pus from an abscess, even when freshly evacuated, may be found to have a very *foul smell:* as, for example, in abscesses in the neighbourhood of the intestinal tract, abscesses in the brain in connection with ear disease, etc., or abscesses connected with the gums or in the neighbourhood of the mouth.

It is now generally accepted that these pus cells are in the main leucocytes that have emigrated from the blood-vessels, and which have lost their vitality more or less completely. It is, however, possible that a small number of the pus corpuscles may be derived from the tissue cells falling into the abscess cavity, as the result of the liquefaction of the tissues by the peptonising action of the bacteria; and it is also possible that the leucocytes may multiply after their exit from the blood-vessels. The *liquor puris* is essentially the fluid which has passed out of the blood-vessels during the inflammatory stage, and which has been prevented from coagulating by the peptones resulting from the growth of the bacteria.

Formation of acute abscess.—The following is the method in which an acute abscess is formed. The stage of granulation, which has been previously described (page 60), having been reached, the inflammation continues, and the next thing which happens is that towards the centre of this mass of granulation tissue, liquefaction takes place, and a cavity is formed containing a fluid with corpuscles: that is to say, pus. Around this cavity active inflammatory processes go on, a wall of granulation tissue is present, and active emigration of corpuscles takes place from the vessels. The abscess so formed enlarges by the extension of the inflammatory process in the tissues around, and by the liquefaction and disappearance of the layer of granulation tissue which lies next the interior of the abscess. When an acute abscess has formed, there is little probability of a cessation of the process. In practically all cases inflammation extends at the periphery, while the liquefaction goes on in the interior, and thus the abscess gradually increases in size. The inflammation at the periphery naturally extends most readily, and leads to the stage of complete granulation most quickly in those parts where the tissues are the most vascular and of the greatest vitality. Thus, if the abscess have formed beneath the skin, granulation of the skin will occur more rapidly than granulation of the fat beneath, and hence it is that abscesses in these situations have a constant tendency to destroy the skin and to point and burst through it. On the other hand, where an abscess is deeply seated beneath a dense fascia, granulation occurs much more rapidly in the cellular tissue beneath the fascia than in the fascia itself, and consequently the abscess may extend for a very considerable distance —may *burrow*, as it is termed—before an opening has formed in the fascia over it, either as the result of a sloughing process or as the result of granulation. Hence, where an abscess is somewhat deeply seated, and more especially where it is situated in an organ such as the breast, where there are numerous septa of fibrous tissue, we have an irregular cavity formed with recesses and pockets extending in various directions. This is a most important point to bear in mind with regard to the treatment.

Ætiology of acute abscess.—We may divide the causes of acute suppuration into those which are essential and those which are accessory. There is only one cause which is essential, and without which acute suppuration will not occur, namely, the presence of pyogenic organisms; but there are many causes which are accessory, and without the co-existence of some of which the pyogenic organisms may in certain circumstances be powerless to act. Therefore, on the one hand, suppuration will not occur unless these pyogenic organisms are present, while, on the other hand, even although they are there, it does not necessarily follow that suppuration must ensue.

We have a considerable number of micro-organisms which are found in acute abscesses, and which are apparently the cause of the suppuration; but by far the most common of these are the Staphylococcus pyogenes aureus, Staphylococcus pyogenes albus, and Streptococcus pyogenes (page 25). Other forms are described which are much rarer and which appear also to be less virulent, such as the Staphylococcus pyogenes citreus, Staphylococcus albus and flavus, one or two forms of bacilli, etc. (page 26). As regards frequency with which these different forms occur, the most common is the Staphylococcus pyogenes aureus, next the Staphylococcus pyogenes albus, much more rarely the Streptococcus pyogenes, and then, quite as exceptions, the other organisms to which I have referred. The staphylococci occur in about 77 per cent. of acute abscesses, the streptococci in about 16 per cent., and the other organisms in the remainder. Thus the staphylococci are the chief causal agents of circumscribed acute abscesses; on the other hand, it may be mentioned that the Streptococcus pyogenes is the essential cause of diffuse suppuration of the cellular tissue. The two chief forms of pyogenic staphylococci grow readily outside the body on all sorts of culture media. When inoculated into the ordinary nutrient jelly, both forms grow readily at the ordinary temperature of the air, and both of them tend after a time to render the material fluid, the growth in the case of Staphylococcus aureus becoming of an orange-yellow colour, while that in the case of Staphylococcus albus remains milk-white. The Streptococcus pyogenes grows much more slowly on nutrient jelly, and forms small, round, white balls which do not liquefy the gelatine (page 27). A very important point with regard to all these organisms is that they peptonise albumen, the staphylococci more especially doing so with great readiness. The importance of this fact is great, as explaining the liquefaction of the tissues during the process of suppuration, and the peptones produced during this process prevent the coagulation of the fluids which are effused from the blood-vessels. As to the other products of growth of these organisms, but little is definitely known. Among numerous other things they are said to produce ammonia, trimethylamine, and toxic albumoses (page 19). It is, no doubt, by virtue of these products that they give rise to inflammatory disturbances; and we can readily understand that if there be a constant production of some irritating chemical substance, such as ammonia, in the tissues, acute inflammation will be kept up; while the toxic albumoses, like those produced by many other organisms, when absorbed into the circulation will set up fever and the other constitutional signs of acute suppuration. When injected into animals under suitable circumstances these organisms cause inflammation and suppuration, and in large quantities may lead to death.

The sequence of events after the entrance of these organisms into the tissues is well seen—though in an exaggerated form—in cases where the smaller blood-vessels are plugged with emboli containing pyogenic cocci, as is the case in pyæmia. (*See* page 177.) On staining sections of tissues containing such emboli in an early stage, we find that while the central mass of organisms is deeply stained, and while the nuclei in the greater part of the section have become well coloured, there is a ring of tissue around the central mass of organisms which does not take on the stain, and which presents a homogeneous, translucent appearance. This translucent ring is evidently the result of the action of the concentrated products of the micrococci, the tissue being brought into the condition of what is termed *coagulation necrosis.* At a somewhat later period a second ring, which is composed of a dense layer of leucocytes, appears outside this clear area, and for a time these three circles can be clearly made out—a central one, composed of a mass of organisms; a middle clear circle, where the nuclei are unstained, and into which leucocytes have not yet penetrated; and an outer dense wall of leucocytes (Fig. 15). As time goes on, however, the intermediate translucent area becomes infiltrated on the one hand with cocci from the breaking up of the central plug, and on the other hand with cells from the outer ring, and the original tissue in that part rapidly disappears, probably, to a great extent, due to the peptonising action of the cocci; and thus the final result is a central collection of fluid containing leucocytes and micrococci, surrounded by a wall of leucocytes and cocci—in other words, an abscess.

In the ordinary circumstances of the formation of an acute abscess, the cocci are not, of course, present in such numbers, nor are they massed together, as in pyæmic emboli, and thus the appearances at the early stage just described would not be so typical, although without doubt the changes which are going on are essentially those which I have detailed. While these micro-organisms are necessary for the production of acute suppuration, and while without their presence acute suppuration could not occur, it does not follow that whenever they are present acute suppuration must take place. On the contrary, in ordinary circumstances, it is probably the rule that other conditions must come into play to assist the action of these micro-organisms; but how far these *accessory conditions* are essential depends on various points in connection more especially with the kind, the numbers, and the virulence of the organisms themselves. As I have pointed out, the effects of the organisms on the tissues vary with the *kind* which is introduced; for example, the staphylococci tend to form circumscribed abscesses, while the streptococci are especially concerned in the production of diffuse spreading suppuration. And among the staphylococci the different species differ in the severity with which they act, and further, while in the case of the staphylococci—more especially of the rarer forms—the presence of various accessory conditions is almost essential to their action, the streptococci, on the other hand, are apparently more or less independent of such secondary circumstances. That the *virulence* of the individual varieties differs at different times is evident, both from clinical observation and from experiment. And the less virulent the organism is, the more does it become necessary for other factors to come into play in order that suppuration may arise; and *vice versâ*, the more virulent the organism, the less necessary are these secondary causes. Then, again, much depends on the *number* of the micro-organisms which have entered

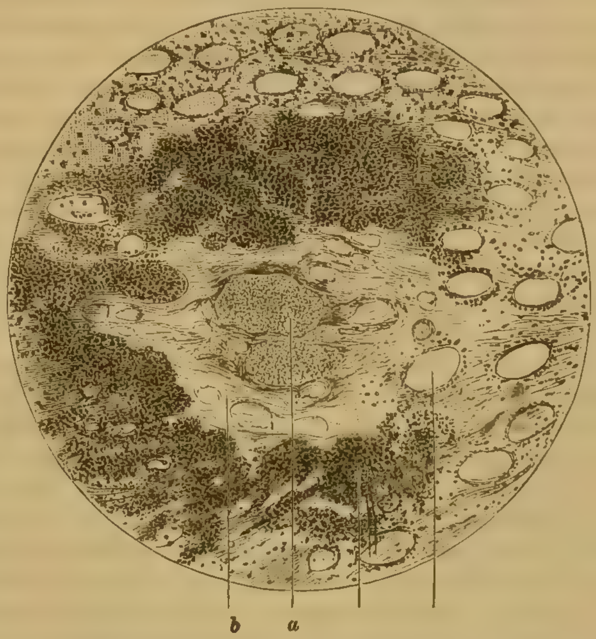

Fig. 15.—Section of Kidney, showing Changes following an Embolus composed of Pyogenic Cocci.

a, Central plug of cocci; *b*, clear space around; *c*, commencing infiltration with leucocytes; *d*, kidney tubules.

the part in the first instance. If they enter in very large numbers, they will overcome the resistance of the body and produce very grave disease; while if few in number, they may be destroyed before they have had time to act. In rabbits, which are not readily susceptible to the action of the Staphylococcus pyogenes aureus—probably not nearly so susceptible as man—I found that something like a hundred million cocci must be injected at one part in order to produce an abscess, while a thousand million are required to kill the animal. In man, no doubt, the numbers are very much smaller, but nevertheless, the principle remains the same.

Among the *factors on the part of the body* which are more or less necessary in order to enable the organisms to act, a very essential condition is that it may be possible for them to remain at rest for some time in the part. If they be only present in the circulating blood, and are constantly carried on in it, they may die out without causing any disease; and as regards the staphylococci, at any rate, it is probable that when they are present in the blood, they are only able to exert their pathogenic action when deposited in some part which has been previously weakened and thus rendered fit for their reception. Hence the great importance of *embolism* in the formation of abscesses, and hence also one of the chief ways in which *local injury* may lead to the formation of an acute abscess. In the latter case, where an acute abscess follows a local injury, these pyogenic cocci have been present in the blood at the time of the injury, or have entered it very shortly afterwards and have passed out of the ruptured vessels, and thus found a place where they could develop.

Another very important condition on the part of the tissues is that they shall be in a weak state, and this *weakened condition* of the part may be brought about in various ways. For example: inflammation which has been set up from some other cause, such as an injury, the entrance of a foreign body, etc., will so weaken the part that if cocci reach it from the blood or lymphatic vessels, or otherwise, they may be able to grow and set up the typical changes. A similar weakening of the part may be brought about by cold or injury, or the action of irritating chemical substances which may be adventitious in origin, or the products of bacteria themselves. A good deal also depends on the seat of the disease and the anatomical arrangement of the part. Thus, in the neighbourhood of the epiphyses, the circulation is apparently slower than elsewhere, and particles floating in the blood—for example, particles of pigment or bacteria—have a better opportunity of becoming deposited in that part. Hence one explanation of the frequency with which inflammation in the substance of bones begins in the neighbourhood of the epiphyses. Again, in the case of the peritoneum we have a very striking example of the influence which the anatomical arrangement of the part exerts on the question of suppuration. Contrary to what was formerly believed, the peritoneal cavity is a place where suppuration in many cases is less likely to occur after the entrance of micro-organisms than the cellular or muscular tissues, and many points with regard to the anatomical and physiological characters of the peritoneum come into play in explaining this fact; to mention one, the peritoneum is a membrane which absorbs fluids with the very greatest rapidity, and in this way deprives bacteria of the necessary pabulum, and brings them into immediate contact with active cells, either epithelial or plasma cells.

As to the mode in which these micro-organisms reach the part and set up the abscesses, they must come either from the blood—in which case they have entered from some mucous or cutaneous surface which is in an unhealthy condition—or they may come from the lymphatic vessels, having reached them through some lesion of the surface epithelium; or they may have penetrated directly from the skin through ducts, as not uncommonly happens in cases of mammary abscess, where the organisms spread up the milk ducts, reach the acini, and then pass into the tissues and set up acute inflammation. These pyogenic organisms are constantly present on the surfaces of the body, both on the skin, especially in regions where moisture is present and the sebaceous glands are large, and on mucous membranes; and thus, when a lesion occurs in these parts, or when they are weakened from some cause, the organisms can get into the blood or the lymphatic vessels (page 11).

Symptoms of acute abscess.—As regards the *local symptoms* of acute suppuration, we meet with the four cardinal signs of inflammation which have been previously considered (page 63), namely, redness, heat, swelling, and pain; and, indeed, the description there given refers more especially to inflammation which is going on to suppuration, and need not, therefore, be repeated here. It will be noted that the centre of the inflammatory swelling is described as hard and brawny, while the surrounding parts are soft and œdematous (page 64). As time goes on and suppuration occurs, the centre of the brawny swelling begins to soften, till by-and-by the presence of fluid can be distinctly made out.

The presence of fluid in an inflammatory or other swelling may be determined in various ways. In the first instance, it may be ascertained by the sense of *fluctuation:* that is to say, by the sensation felt by the fingers when a wave of fluid is set in motion by the hand at some other part of the swelling. Unless the abscess is very near the surface of the skin, it is not always easy at the early stage of an abscess to make out this sensation of fluctuation, on account of the great pain which the firm pressure required in these circumstances will cause to the patient. Hence it is not uncommon for fluctuation to be missed although pus is actually present; on the other hand, it is not unusual for the opposite mistake to be made, and the presence of fluctuation asserted when an abscess is not present. In order to ascertain the presence of fluctuation, a finger or fingers of one hand are placed on one side of the suspected part, and are kept at rest and exercising a certain amount of pressure, while with the other hand pressure is made on the swelling as far distant as possible from that which is placed to receive the sensation.

If fluid be present and no other fallacy have crept in, the fingers of the hand which is kept at rest will feel the impulse of the wave of fluid that has been set in motion by the other hand. Now, in cases where the area to be examined is quite small, the two fingers may be of necessity so close together that the soft tissues are pushed by the one and felt by the other, and thus fluctuation is erroneously diagnosed. Perhaps the most common mistake is caused by the false fluctuation induced when palpation is made transversely to muscular fibres. In such circumstances, a very distinct feeling of fluctuation may be produced across the belly of a muscle even although the hands are kept at a considerable distance from each other; and, therefore, where a mass of muscles is present in the region to be examined, great care must be taken to test for the presence of fluid by the fingers placed in the direction of, and not transversely to, the muscular fibres. Where the collection of fluid is small, fluctuation may also be ascertained by placing the finger of one hand over the centre of the swelling, in order to feel the sensation, and, grasping the swelling between the fingers of the other hand, which are then sharply approximated to each other; if fluid be present, the finger of the first hand will be lifted by the wave so set in motion. Or, again, if a finger be rapidly pressed into the part and then held at rest, it sets in motion waves of fluid which are readily felt; this method of investigation, however, is not suitable to acute abscesses where the fluid is in a state of tension, it can only be used in cases where the fluid is contained in a lax cavity, and also where there is but little pain on sharp pressure. As a matter of fact, where the centre of a brawny swelling is becoming softened—that is to say, where pus has formed and is approaching the skin—the presence of an abscess is readily ascertained by passing the fingers gently over the part; where pus is present and superficial, a *softened circle* will be felt in the middle of the brawny swelling, and an abrupt, sharp, *hard edge* around, and where this is found it is quite unnecessary to cause pain to the patient by testing for fluctuation, because it is in itself an absolute sign of the presence of pus.

Where the inflammation is deeply-seated, and the skin is not affected, the presence of *œdema* of the skin over the region of the inflammation is almost always indicative of the occurrence of suppuration. For example: in an abscess connected with the appendix vermiformis the skin of the abdomen over the seat of the inflammation not uncommonly becomes œdematous; and when this is the case, one may feel certain, without further examination, that pus is present beneath. Besides, as has been already said, where an acute inflammation has lasted for four or five days, it is almost certain that pus is present, whether fluctuation can be felt or not.

In cases where it is held to be absolutely essential to know whether pus is present before making an incision, punctures may be made by means of an aspirating needle.

The *skin* over the softening portion of the swelling is of a dusky purple colour, and becomes gradually thinner till the yellow pus can be seen through it; and by-and-by it gives way, and the pus escapes externally. Accompanying these local signs, there are the various *constitutional symptoms* which have been already referred to under acute inflammation.

Diagnosis of acute abscess.—The diagnosis of acute abscess is seldom difficult once pus has definitely formed. The first difficulty is to be sure that pus has actually formed in the case of deep-seated inflammations; but the length of time which the inflammation has lasted, the continuance of the pain and swelling, the presence of œdema of the skin, and the other signs already mentioned, will enable one, at any rate, to form a strong suspicion on the matter. In some cases it is very difficult to distinguish an acute abscess from a very rapidly growing sarcomatous tumour. I have myself seen such a sarcoma of the upper jaw incised under the impression that it was an acute abscess, and in that case there were present all the local signs of an acute inflammation in the form of rapid formation of the swelling, redness of the skin, indefinite sensation of fluctuation, and great pain. It is said also that aneurysm has been mistaken for an acute abscess. This, however, is hardly likely to occur, unless suppuration has actually taken place in or around the aneurysmal sac, in which case, of course, the surgeon has really to do with an acute abscess. Where there is any doubt of this kind it may be well to introduce an aspirator needle in the first instance, to ascertain the nature of the fluid present. As a matter of fact, apart from the suppuration of an aneurysmal sac, the only other condition in which a mistake might be committed is where the connection between the sac and the vessel has been more or less completely cut off, so that pulsation has become abolished, and where the contents of the sac still remain fluid. Such a case has been opened under the impression that it was a chronic abscess, and the fluid which came out in the first instance was clear serum, very quickly followed, however, by red blood.

Treatment of acute abscess.—In the early stage, before suppuration has definitely formed, the treatment is that which has been already described under acute inflammation (page 66); but when once it is certain or probable that pus is present, means must be taken to open the abscess without further delay, and to provide a free exit for the pus. There is no object whatever in permitting an acute abscess to go on till it reaches the skin. To do so is simply to allow the formation of a much larger cavity, and the consequent destruction of or interference with the tissues in the neighbourhood: and if it should happen that pus is not found on making the incision after all, the best thing has been done with the view of cutting the inflammation short.

When it is decided to open an abscess, the external opening should be free, and should in all cases—unless about the face or neck, where it is undesirable to have a larger scar than necessary—be large enough to enable the surgeon to introduce his finger into the cavity. As regards the position of the opening in cases of acute abscess, the general rule is that it should be at the most dependent part of the cavity, so as to allow the discharge to escape freely. This rule is, no doubt, wise in cases where the pus has been burrowing underneath the skin; but it is not essential in deeper-seated abscesses, and if followed may at times lead to inconvenience. An essential rule as regards the position of the opening in acute abscesses is that it should be as far as possible from the orifice of any mucous canal, and therefore from sources of contamination. If only the abscess be freely and thoroughly opened, and efficient drainage established, the pus does not go on burrowing, even although the opening is not at the most dependent part of the cavity.

It is a matter of great importance in treating acute abscesses to see that all the recesses of the abscess are freely opened. As I have already said (page 81), in the case of deep-seated abscesses, or abscesses among muscles or bands of fibrous tissue—as in the breast, etc.—the pus is apt to spread in irregular directions, forming small recesses, which are not properly evacuated if only an external incision be made. Hence it is necessary, when such abscesses are opened, to break down the septa which separate the various projections of the abscess, and this is much better done by means of the finger than by any instrument. Where the abscess is quite superficial this is not, as a rule, necessary, and an opening sufficiently large to admit a proper-sized drainage-tube will answer the purpose; but in the case of all abscesses which have passed through fascia it is very essential to open up the deeper recesses. In the neck, where it is desirable to avoid a scar, a pair of dressing forceps may be introduced through the wound which has been made, and by separating the blades the hole in the fascia through which the pus has reached the surface may be enlarged; and then, by pushing on the forceps into the various recesses of the abscess, and opening them from time to time, the cavity may in most cases be satisfactorily drained.

In some instances, where the suppuration has occurred in the neighbourhood of important structures, where the pus is deep-seated, and where it might be dangerous to cut freely till one reaches the collection, the plan known as *Hilton's method* is usually employed. This consists in making a small incision through the skin, and then, by means of dressing or sinus forceps, gradually boring one's way into the abscess cavity. That the abscess cavity has been reached is evident from the sudden cessation of resistance to the passage of the forceps, and in most cases by the escape of pus along the side. Having in this way reached the abscess cavity, the blades of the forceps are thoroughly expanded so as to tear up the intervening tissues, and are then pushed on into the various recesses of the abscess and expanded from time to time with the view of opening the cavity completely.

Whichever method is employed, a *drainage-tube* of sufficient size should then be introduced. In selecting a drainage-tube it must be remembered that pus is a thickish fluid and does not readily flow away, and therefore a tube of sufficient size should be employed. In fact, one cannot make a mistake in selecting too large a tube. The tube must be of proper length, and not project beyond the surface of the skin, and the portion of the tube which enters the cavity should be perforated laterally with holes, so as to allow the pus to enter it freely. The tube is best introduced by means of a pair of Lister's sinus forceps. The forceps are, in the first instance, passed into the cavity in order to ascertain its depth, a tube is then cut somewhat shorter than the depth to which the forceps go. It is then introduced into the cavity, and the projecting portion cut so as to be flush with the skin. It is either finally secured in its place by a stitch through the skin and the wall of the tube, or else two strings are passed through the wall of the tube and knotted, and it is kept from slipping in by the pressure of the dressing over it. Where Hilton's method has been employed, it is well before withdrawing the forceps which have dilated the canal to introduce a probe beside them into the cavity of the abscess, otherwise one may miss the track which has been made by the forceps, and when one comes to introduce the tube there may be considerable difficulty in doing so. If, on the contrary, a probe be left *in situ*, the tube may be readily slipped over the probe until it reaches the abscess cavity.

When the tube has been inserted, the swelling may be squeezed so as to expel the greater part of the pus; but I do not think it is good practice to wash out these abscesses, either with an antiseptic or an aseptic fluid. The whole operation must, of course, be done antiseptically, after the manner which will be described in another article (page 213); and as regards the progress of the case, the dressing should usually be changed on the following day, and then subsequently according to the amount of discharge. After the abscess is opened, the swelling very rapidly subsides, so that even on the next day, if the tube have been introduced quite to the bottom of the cavity, it will be found to project from the wound, and the projecting portion must be cut off. It is well not to remove the tube at all for at least three or four days, and this is more especially the case if the abscess have been opened by Hilton's method. If the tube be taken out too early, considerable difficulty may be found in re-introducing it. As a rule, in an abscess of moderate size the tube may be left out altogether at the end of about a week, and the sinus thus left will generally heal in a further eight or ten days. After the first pus has been evacuated (provided the operation has been done thoroughly aseptically), no further pus is formed, the discharge from the wound consists of thin, fairly clear serum, and as regards the local and general symptoms, immediate relief is experienced, the redness passes off, the skin loses its tense shiny appearance and becomes wrinkled, the fever subsides, and the patient rapidly recovers.

So far I have described the treatment of an acute abscess as it occurs, say, in the cellular tissue. The treatment must of course vary in many cases with the region which is the seat of the suppuration.

In a case, for example, of acute abscess connected with inflammation of the periosteum and bone, the treatment may consist of opening the abscess, and, in addition, of the removal of portions of the inflamed bone. Similarly, in abscesses in serous cavities, such as the pleura, means may be taken to favour the expansion of the lung ; but these different points in connection with the treatment of acute abscesses will be referred to in articles dealing with the diseases of the various tissues and organs, and need not therefore be touched on here.

Acute suppuration from open wounds.—In acute suppuration from open wounds the course of events is very much the same as regards the pathological changes, but, the pus being able to escape freely, the local and constitutional symptoms which are present in an unopened abscess are naturally less marked. In fact, after the wound has become completely covered with granulations, and free suppuration is established, the local and general symptoms of inflammation subside and almost entirely disappear, unless, indeed, some septic complication has come into play. The question of suppurating wounds will be referred to again in speaking of the healing and treatment of wounds (Art. X. page 209).

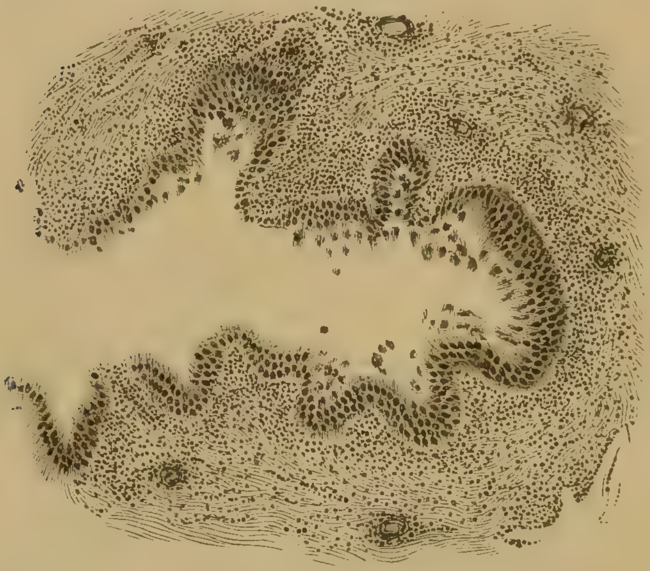

Fig. 16.—Section of the Urethra, from a case of Gonorrhœa of ten days' standing, showing the submucous cellular infiltration and the shedding of the epithelial cells, some of the projections being completely denuded.

Acute suppuration from a mucous surface.—Where suppuration occurs from a mucous surface, such as the urethra, the inflammatory changes take place in the first instance in the submucous tissue, which becomes loaded with leucocytes and young cells ; these pus cells pass out between the epithelial cells, and escape on the free surface, along with fluid derived partly from the blood-vessels, and partly from hypersecretion of the mucous glands, whilst the epithelium itself is partly shed and partly undergoes multiplication, so that epithelial cells are found, which contain in their interior several smaller protoplasmic masses (Fig. 16). It is true that some authors state that these contained cells are not formed by multiplication of the original epithelial cells, but that they are in reality leucocytes which have penetrated into the cells, and they deny the occurrence of proliferation in the epithelium. This view, however, I do not think is correct, more especially when we bear in mind the facts with regard to proliferation of tissue cells in inflammatory conditions, such as the proliferation of the endothelium of the peritoneal cavity. After a time, if the inflammation go on, the mucous membrane becomes, in parts, more or less completely deprived of its epithelial covering, and ulceration takes place, so that when recovery occurs a scar is left on the surface of the mucous membrane. Where this inflammatory condition surrounds the canal and lasts for a long time, the new-formed connective tissue is very apt to constrict the calibre of the canal and produce a stricture.

II. DIFFUSE CELLULITIS.

At the beginning of this article I mentioned that there were two forms of suppuration in the tissues: namely, the circumscribed acute abscess and diffuse cellulitis, and we must now shortly consider the latter condition. (*See also* page 167.) This condition is usually, and probably always, due to the Streptococcus pyogenes (page 27). The inflammatory process is not limited to one part, but spreads rapidly along the cellular tissue, the organisms apparently growing along the lymph spaces and lymph channels. The result is that the tissues become swollen from the presence of the exuded fluid and of numerous cells, but there is not in the early stage any regular formation of a cavity containing pus. If an incision be made into a part affected with diffuse cellulitis, turbid semi-purulent fluid, or even well-formed pus, can be pressed out from the interstices of the tissue, while it is also found that portions of the fat, and fascia, and other structures in the affected area have died and form sloughs. Thus, the characteristic change in diffuse cellulitis is the infiltration of the tissues with pus and the sloughing of shreds of the tissues affected. This condition is a very grave one, because the patient is apt to die of septic poisoning from absorption of chemical products from the seat of disease, or the organisms themselves are very apt to get into the circulation either directly, or after having led to phlebitis and thrombosis, and thus set up the condition of pyæmia. (*See also* Art. VIII.)

Symptoms of diffuse cellulitis.—As regards *local appearances*, the disease generally starts from a scratch or a puncture, and begins with uneasiness in the affected part. Around the seat of the puncture redness of the skin appears, and spreads pretty rapidly. and as a rule red lines are very soon found running along the course of the lymphatic vessels to the nearest lymphatic glands. Locally, the skin becomes much swollen and brawny, and tender, but as time goes on and as suppuration appears the part becomes boggy, pitting on pressure and giving an imperfect sensation of fluctuation. In the course of the inflammation definite localised collections of pus may be found at a later period, but at the early stage this is seldom the case, the tissues being, as I have just said, infiltrated with the pus over an extensive area. Fresh patches of cellulitis also not uncommonly appear along the course of the lymphatic vessels, probably because the organisms have blocked the lymphatic vessel in some part of its course, and spread into the tissues around, and set up the disease afresh. If the case be left to itself and the patient lives, openings by-and-by form in various parts over the inflamed area, from which pus and shreds of tissue, portions of muscles, tendons, etc., ultimately escape.

At the same time, the patient suffers from very severe *constitutional symptoms.* At the early stage the disease generally begins with a rigor and high temperature, and the temperature in this stage often runs up very rapidly to 104° or 105° Fahr. Very soon, as the disease advances, the patient passes into the asthenic or typhoid condition previously described; his pulse becomes very rapid and weak, and often irregular, his temperature remains high and oscillating, his tongue becomes brown and dry, his face flushed, and he has delirium of a low muttering character (page 65). In most cases, if the disease cannot be checked, the condition ends in death in a very short time, or is very apt to lead to pyæmia (Art. VIII.). The symptoms are gravest where the organisms have, in the first instance, gained admission to serous spaces, such as tendon sheaths, bursæ, etc., and in most cases this does actually occur, because as a rule this affection commences with scratches or cuts about the arm or fingers.

Apart from the immediate gravity of the condition, even if the patient recover, this affection generally leaves very serious results behind, more especially in the case of the arm: results such as the matting together of muscles, the loss of tendons, adhesions between tendons and their sheaths, etc., which lead to loss of movement of the fingers and hand.

Treatment of diffuse cellulitis.—The treatment of diffuse cellulitis must be from the very first of the most vigorous nature; and as regards the constitutional treatment, it consists in supporting the strength of the patient, free administration of stimulants, the use of quinine in large doses, and also of antipyretics with a view of keeping the temperature down, etc. Locally, the proper treatment at the very earliest period is to make free incisions into the inflamed area, with the view of promoting the escape of the fluids and sloughs, so that there shall be less absorption of the poison. These incisions must be extremely free, and, in fact, the whole brawny tissue should be thoroughly laid open from end to end, and any recesses from which pus can be squeezed should be opened up. The incisions in these cases cannot be too free, for if any recesses escape inspection and free opening, the disease is certain to spread from them. After having thoroughly laid open the whole area of the disease, it is well, I think, in the first instance, to remove the pus and blood as completely as possible, and then to sponge the whole surface with undiluted carbolic acid. I have in several instances brought the process to a standstill—even in very grave conditions of the patient—by free treatment of this kind. Where the condition is a very serious one, and where there is considerable destruction of the tissue, I think it is well, after having sponged the exposed parts with undiluted carbolic acid, to pack them with lint which has been squeezed out of the pure acid. Where one does not wish to increase the destruction of the tissue which naturally follows this method, or where the result of the packing is the cessation of the process, the best subsequent treatment is, I think, to stuff the wound and all its recesses with cyanide gauze sprinkled with iodoform. After a few days the whole surface will be covered with granulations, and then the ordinary applications suitable for healing granulating wounds, and which will be referred to in discussing the treatment of wounds, should be substituted (Art. X.). In some cases great benefit is derived from continuous irrigation after free incision or from the use of a continuous water bath, the fluid in both instances containing some antiseptic (such as permanganate of potash, or sanitas), which does not coagulate albumen.

When the inflammatory symptoms have come to an end, one must bear in mind the great risk in the hand of adhesions between the muscles or tendons, and the consequent chance of permanent disablement of the hand; and therefore, as soon as possible, passive and active motion of the fingers and hand should be freely carried out. It generally suffices if the passive motions be carried out once or twice a day in the first instance, the patient being encouraged to move the fingers at any time. As soon as the wound has healed, the part should be thoroughly massaged, and the muscles kneaded, with the view of breaking up and getting rid of the inflammatory effusion.

SUBACUTE ABSCESS.

Apart from acute abscesses, we also speak of subacute and chronic abscesses. By subacute abscesses we mean those which are slower in their course than those which I have already described, and in which the local and constitutional symptoms are correspondingly milder. As a rule, the course of a subacute abscess will be from two to three weeks, instead of less than half that time, as is the case in acute abscess. These subacute abscesses are for the most part due also to the pyogenic organisms, but usually to the rarer forms, which are less virulent. The treatment of these abscesses is essentially the same as that of the more typical acute abscesses. The so-called chronic abscess, however, differs absolutely from the acute abscess and subacute abscess both in its pathology and in its treatment, and with very few exceptions, if any, it is a tubercular affection.

CHRONIC ABSCESS.

By a chronic abscess we mean an abscess which increases in size very slowly, and which produces little or no local or constitutional disturbance till it is opened. These abscesses may originate in the soft parts; but perhaps most of them originate as the result of tuberculous disease in glands, joints, or bones (Fig. 17). The chronic abscess is, in fact, a tuberculous tumour with a softened centre, and when it begins—for example, in the subcutaneous tissues—it appears in the first instance as a hard nodule, which consists of tubercles with some inflammatory thickening around. As this nodule enlarges, the older tubercles in the centre undergo caseation and break down, and lead to the formation of a fluid (the so-called pus), which is surrounded by a wall containing numerous tubercles (Fig. 18). The abscess so formed will, in most cases, gradually extend, sometimes, however, taking several months before reaching any large size. The tuberculous new growth steadily invading the surrounding tissues and breaking down at the centre, the abscess by-and-by reaches the skin, which becomes thin, and ultimately gives way, and the contents are evacuated.

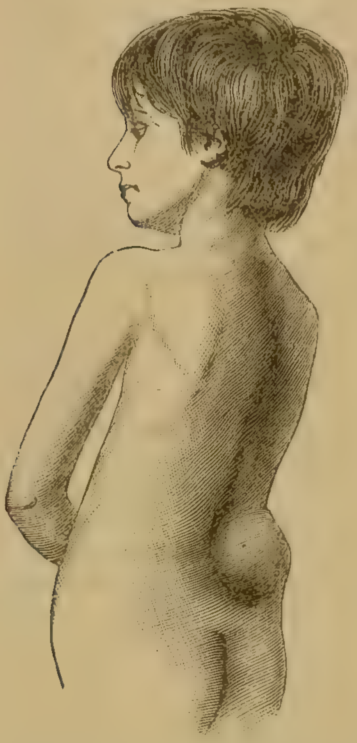

Fig. 17.—Lumbar Abscess in connection with Spinal Disease.

Symptoms of chronic abscess.—So long as the abscess is unopened it seldom gives rise to any noticeable constitutional symptoms beyond, possibly, slight elevation of temperature, but after an opening forms, septic organisms enter; and where the cavity is large, as in the case of psoas abscess, it is not uncommon for more or less violent suppuration, with all its local and constitutional symptoms, to ensue. In some cases, indeed, the immediate effect of opening a psoas abscess without any precautions against the admission of micro-organisms is violent febrile disturbance, quickly followed by a condition of collapse, and the death of the patient within two or three days. In this case the cause of the trouble is septic intoxication, from rapid decomposition of the contents of the abscess, as the result of the growth in them of saprophytic organisms (page 40). Most usually, however, the effect of sepsis in a large chronic abscess is, as I have just said, the occurrence of suppuration and acute local and general inflammatory disturbance. This may be followed by acute septicæmia or pyæmia (Art VIII.), but most commonly the acuteness of the symptoms subsides, and a profusely suppurating sinus is left. This suppuration from the sinus goes on indefinitely, and very often, if the exit of pus be imperfect, fresh accumulations form, with recurrence of the acute attacks, or abscesses may arise on the opposite side or in the loin.

As the result of this continued suppuration, the patient rapidly loses ground, becomes emaciated, and suffers from hectic fever. *Hectic fever*, in fact, is nothing more or less than chronic septicæmia, and it only occurs in cases where sepsis is present. Its symptoms consist in the first place in certain peculiarities of the temperature, the evening temperature being two or three degrees higher than the morning, which is very often normal; at the same time the patient wastes, his face is flushed—especially over the malar bones—there are profuse night sweats, and the appetite becomes impaired.

After this condition of hectic fever or chronic septicæmia has lasted for some time, a condition which is termed *amyloid degeneration* occurs in various parts, more especially in the liver, spleen, kidneys, and intestines. This amyloid degeneration begins in the walls of the smaller arteries, which lose their definite cellular structure and become converted into a peculiar structureless substance which presents certain definite reactions to staining reagents. Thus, with iodine, the parts which are in a state of degeneration become dark brown as compared with the yellow staining of the rest of the tissues. Or again, on staining with ordinary commercial gentian violet, the parts which are the seat of amyloid degeneration come out a pink colour as compared with the bluish violet stain of the rest of the tissues.

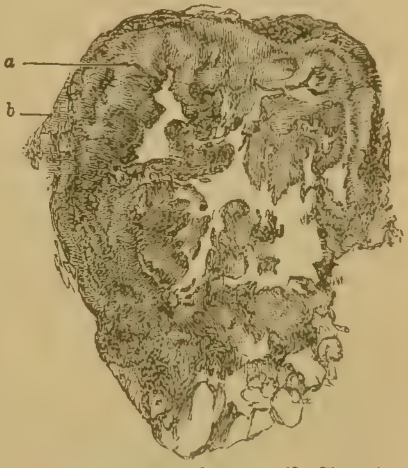

Fig. 18.—Section of a small Chronic Abscess, showing the central Cavity surrounded by a very ragged and caseating wall (*a*). Outside that is the inflamed part and cellular tissue (*b*).

Amyloid degeneration, though beginning in the walls of the vessels, seems after a time to extend into and affect the tissues around. The result of this condition is marked enlargement of the organs which are the seat of the degeneration, the liver more especially becoming very much increased in size and also in weight; the spleen becoming also enlarged, and on section presenting the appearance of what is termed the "sago" spleen, small, hardish, sago-like granules being scattered throughout the substance of the organ. In the kidneys, the vessels first affected are chiefly those in the glomeruli; and in the intestine, the vessels in the villi.

The cause of this degeneration has been variously explained, the majority of pathologists up till recently attributing it to changes in the constitution of the blood from the continued loss of pus, more especially loss of the alkaline and potassium constituents of the blood. My own opinion, however, is that it is a direct effect on the vessels of the circulation through them of the products of the bacteria which are keeping up the septic condition. This form of degeneration practically only occurs where there are suppurating wounds, and it is most usually found in cases of tubercle where the suppurating wounds remain a long time open; and further, the fact that the disease in the first instance affects the wall of the small arteries seems to bring the pathology into relation with changes in the condition of the blood circulating through the arteries, just as we have the condition of endarteritis set up in various other circumstances where irritating materials, such as alcohol, acetone, etc., are circulating in the blood. The result of this condition of the arteries is, more especially with regard to the kidneys and the intestines, that they become leaky, resulting in increased secretion (which is albuminous) by the kidneys and in diarrhœa, which ultimately assumes the form known as colliquative diarrhœa, and cannot then be arrested by any known means, and which leads in the course of a few days to the death of the patient.

Treatment of chronic abscess.—As the above mentioned results only appear after a chronic abscess is opened, it is evident from our present knowledge that they are directly due to septic decomposition taking place in the wound; hence, in tuberculous abscesses we must be particularly careful to employ all the best measures for preventing the entrance of bacteria—in other words, the abscess must be treated rigidly aseptically. If this be done, none of these troubles, namely, profuse suppuration, septic diseases, hectic fever, amyloid degeneration, occur. Ample evidence of this has been afforded by the results of rigid asepsis in the treatment of spinal abscess, and I have elsewhere published statistics showing that a large proportion—something over 70 per cent.—of cases of spinal abscess treated by aseptic drainage recover completely without having passed through any stage of illness after the abscess was opened. (For an account of Spinal Abscess, *see* Art. XXXIX., DISEASES OF THE SPINE, Vol. II.) With regard to the treatment of these abscesses by drainage, it must be borne in mind that the wall is the essential part of the disease, and while treatment adopted simply with the view of evacuating the contents may, after a long time and with proper care, lead to recovery, yet by this plan the destruction of the disease is left to nature, and the result remains doubtful. Besides which, an open wound must continue for some months, into which, as the result of some error on the part of the surgeon, septic bacteria may one day enter and set up all the troubles which have been already alluded to. Hence, with few exceptions, simple drainage (of course under antiseptic precautions) of these abscesses is now abandoned in favour of more vigorous measures, in which attempts are made to get rid of the wall more or less completely; and the treatment of chronic abscesses may now be considered under the following three heads:—

1. The ideal treatment of chronic abscess is to dissect out the

abscess as one would dissect out a cyst without opening it. If this can be done, a clean cut wound is left, which is not infected with tubercle bacilli, or other bacteria, and we have healing by first intention. This method of treating chronic abscesses may be carried out in a very considerable number of cases; for example, in the subcutaneous abscesses of children (*gomme scrofuleuse* of the French) cure is at once effected by dissecting them out, while months may elapse before healing is obtained by the other plans of treatment. Hence, whenever the abscess is not too large (and after all, a long skin incision is not a matter of the slightest consequence, except in exposed parts of the body) and wherever the connections of the abscess are not such as to render it anatomically impossible to remove it, this is the treatment which should be adopted. This method need not be confined to abscesses originating in the soft tissues, or in glands—where the abscess and the gland together are excised—but can also be carried out in a considerable number of cases of abscesses connected with bones, more especially abscesses associated with superficial bones, such as the ribs or clavicle. In the case of abscess connected with carious ribs, unless it be very large indeed, a free incision should be made over the centre of the abscess, extending well beyond it on each side, and in the direction of the muscular fibres which overlie it. The wall of the abscess should be exposed and freed, care being taken that the cuts are made in the healthy fat around, and that portions of the wall of the abscess are not left behind. The abscess wall having been thoroughly exposed—superficial to the rib—but not yet opened, the point at which it joins the periosteum should be defined, and the rib cut through on each side behind that point. The enclosed portion of rib belonging to the abscess is then removed, along with the abscess wall. It will be found in most cases, however, that the pus has accumulated, not only superficially to the rib, but also on its deeper surface between it and the pleura, and this deeper portion of the abscess, which is generally quite small, cannot usually be removed without running the risk of opening the pleural cavity. Hence, with regard to this part of the abscess, thorough scraping with a sharp spoon must be employed. As a rule, the whole abscess can in this way be removed, and the wound is then closed and heals by first intention. In cases where the abscess is connected with disease in the long bones, more especially with the enlarged epiphysial ends of the bones, the abscess should be freed in a similar manner, and then, by means of a chisel and mallet, the piece of bone should be chipped away underneath the point of attachment of the abscess; if it be then found that there is a tuberculous deposit in the interior of the bone, this deposit must be thoroughly cleared out by gouging or scraping. (*See* Art. XXX.)

2. Where it is not possible on account of the size and anatomical connections of the abscess to dissect it out completely, the surgeon must be content with some less efficient measure. In such a case a free incision should be made over the abscess, laying it open, if possible,

completely, and then, wherever it can be managed, the wall should be cut or clipped away. In the parts where this is not advisable or possible the sharp spoon must be employed thoroughly, with the view of excising any remaining portions of the wall. Bone deposits, or diseased portions of bone, must of course be removed as completely as possible. Where the wall has in this way been thoroughly exposed and pretty completely removed, one may stitch up the wound again, and obtain healing by first intention, and in most cases no recurrence takes place; in fact, in most cases the tuberculous material has been completely taken away. One may, before closing the wound in such cases, inject a small quantity of a 10 per cent. emulsion of iodoform and glycerine, and leave it in. Where considerable portions of the abscess wall are left behind in the depth of the wound, it is best not to close it in the first instance, but to pack it with cyanide gauze, which has been thoroughly sprinkled over with iodoform. This packing will not require renewal for several days—possibly five or six, and then subsequently it should be changed every day or two, according to circumstances. After a time it will be found that the wound (even in the parts where tuberculous material was left behind) has become covered with healthy granulations, and when that is the case, the stuffing may be left out, the edges of the wound stitched, and healing by first intention permitted. It has been supposed by Krause that while iodoform has no real antibacteric action in ordinary circumstances, it may interfere with the vitality of the tubercle bacilli when oxygen is absent, and in plugging a deep cavity firmly with gauze and iodoform, the oxygen is more or less completely excluded, and in addition the surface of the wound is irritated and made to granulate more rapidly by the mechanical action of the gauze itself.

3. In cases where it is impossible to carry out the first or second plan of treatment, and where one can only make a limited opening into the abscess cavity, and cannot reach the whole of its wall efficiently, as in the case of psoas abscess, the next best plan is to make an incision sufficiently large to admit the entrance of the finger. The contents of the abscess are then allowed to escape, and a sharp spoon is introduced, preferably one of Barker's flushing spoons, where the handle is hollow, and a stream of fluid can be made to flow constantly through the wound at the same time that it is scraped. By means of the spoon, the wall of the abscess cavity is everywhere scraped as thoroughly as possible, care of course being taken, on the side towards the veins or the peritoneum, not to open them. Having thoroughly scraped the whole of the soft and as much of the hard tissue of the wall as possible, the material so detached is thoroughly washed out (where Barker's spoon is used this washing-out goes on simultaneously with the scraping) by a stream of weak warm antiseptic solution, such as 1 to 6,000 bichloride of mercury, and afterwards it is well to dry the cavity, and in addition to remove any adherent portions of tuberculous material by introducing a rough sponge into it and thoroughly scrubbing

the wall in all directions. Subsequent to this treatment, one or two ounces of a 10 per cent. emulsion of iodoform and glycerine are injected and left in the cavity, the skin wound and also that through the muscles being stitched up and an antiseptic dressing applied.

As a result, the incision through the skin usually heals by first intention, and in a certain proportion of cases there is no reaccumulation of fluid in the cavity, the trouble being at once cured by a single operation. In other cases, however, while the skin wound heals, a certain amount of reaccumulation takes place in the cavity of the abscess, although it does not attain the same size as it was previously. This accumulation may with rest become again absorbed, but in most cases it is best to re-open the wound, evacuate the fluid (which is usually a glairy, clear, dark brown material containing a quantity of iodine), scrape out the cavity again, and, either after injection of a fresh quantity of iodoform and glycerine, or without such injection, stitch up the wound. In the great majority of cases permanent cure is obtained, either after the single operation or after repetition on two or three occasions.

In a small number, however, not only does the fluid reaccumulate, but the line of incision which had healed by first intention breaks down and a sinus forms. These are generally cases in which either the recesses of the cavity have not been reached in the scraping and washing process, or where a bony sequestrum is present, or where the patients are unhealthy and have a great tendency to tuberculous disease. In such cases I have several times succeeded in getting a permanent cure by dissecting away the orifice of the sinus and its wall as it passed through the muscles, thoroughly scraping out the cavity in the manner previously described, and stitching up the wound once more. In some cases, however, in spite of repetition of this process, I have ultimately had to resort to the old plan of aseptic drainage.

As regards *the treatment of the septic complications* which occur where these abscesses are not opened with aseptic precautions, I need say very little at present, because this matter is referred to in the article on Wounds (page 222). Where, immediately following an incision into a psoas abscess, symptoms of septic intoxication occur, the clear indication is to make the external opening as free as possible, and wash out the decomposing fluid which is present in the cavity. This fluid should be washed out with some non-irritating antiseptic solution such as boracic acid, permanganate of potash, or very weak sublimate solution, because the stronger antiseptic solutions will not in any way disinfect the cavity, but will injure the cells at the surface, and thus possibly provide an entrance for bacteria into the system. After opening up such a cavity, a large drainage-tube should be introduced, and in some cases where the patient is very ill, it might be of advantage to arrange for some hours of constant irrigation through these tubes with one of the fluids previously mentioned kept at the body temperature.

In cases where the septic change occurs somewhat later, and is due to the pyogenic organisms—that is to say, in cases where there is free suppuration from the cavity—all that can be done locally is to provide for very free drainage, while, at the same time, the strength of the patient is supported, and quinine and febrifuges are administered if necessary. Attempts may be made to diminish the sepsis and the amount of poisonous products by injecting iodoform and glycerine into the cavity from time to time, but the effect is really extremely slight.

As regards the *treatment of hectic fever*, the first essential is as far as possible to diminish or remove the septic condition of the wound, and in cases where the suppuration has become more chronic and these hectic symptoms have set in, I think it is worth while opening up the wound, scraping the wall of the cavity as thoroughly as possible, and, after the bleeding has been arrested, sponging the wall with undiluted carbolic acid, and powdering the surface with iodoform. Where we have to do with such a cavity as a psoas abscess, one must then introduce large drainage-tubes and see that the discharge escapes freely; but where the incision into the cavity is freer and one can reach its recesses, it is best to stuff it with cyanide gauze sprinkled with iodoform, the stuffing being renewed whenever it is necessary till the wound has completely granulated. In a certain proportion of cases the parts will be rendered aseptic by this treatment, and the hectic fever will subside. If the result be not perfect, or if the hectic fever and suppuration recur, and if the patient be strong enough, the process may be repeated, and I have succeeded in more than one instance in the case of psoas abscess in checking the septic condition and obtaining healing of the wound by persevering in this line of treatment.

I have described this treatment in connection with the more prolonged and less severe suppuration and the condition of hectic fever. Cases where the suppuration is profuse, that is to say, where active septic processes are going on in the wall of the abscess, are not adapted for such treatment, unless, indeed, one can obtain free access to every portion of the cavity. If one cannot do this, but have to work in the dark, as in cases of psoas abscess, the attempt to scrape and disinfect the cavity will certainly be unsuccessful, and fresh paths are apt to be opened for the spread of the septic organisms left behind into the body generally.

As regards the general treatment of hectic fever, that must be carried out on the lines of the treatment of the various symptoms that arise. Antipyrine and other antipyretics should be employed if the evening temperature goes high, being administered an hour or so before the temperature reaches its highest point. Quinine in all cases is of value, as also is iron. The night sweats are particularly exhausting, and may be checked either by the administration of balladonna internally, or by sponging the skin with a weak solution of acetic acid or sulphuric acid. Stimulants should be freely administered, and as nourishing a diet as can be assimilated should be given.

SINUS AND FISTULA.

In certain cases where abscesses have been opened, the wound is a long time in healing, or refuses to close altogether, a small opening being left which leads into the deeper tissues. Where this opening ends in the depths of the tissues, it is spoken of as a *sinus;* or where, being in the skin, it leads to an opening in a mucous membrane, it is spoken of as a *fistula.* Sinus and fistula may also occur in operation wounds, but as I have said, they are much more commonly the result of a previous abscess.

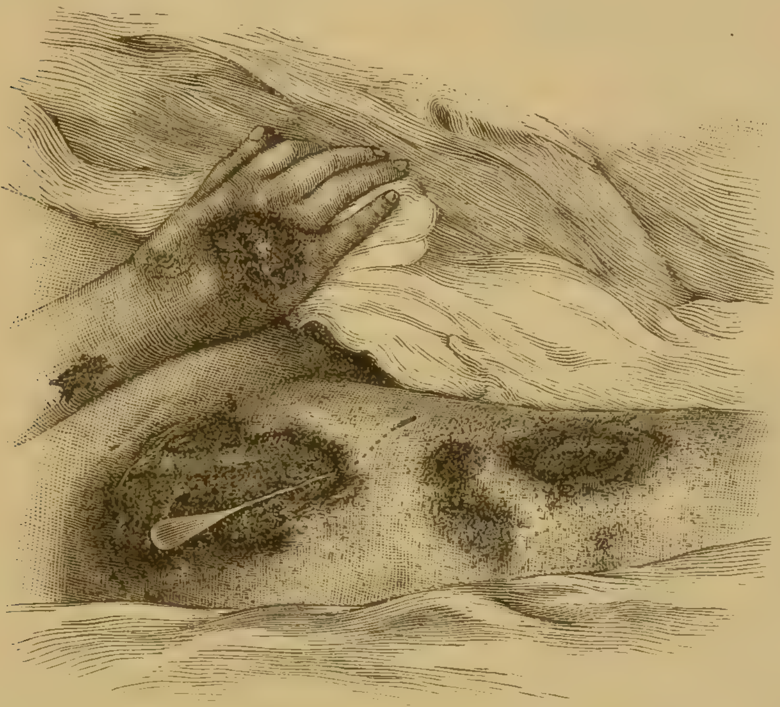

Fig. 19.—Tubercular Sinuses and Ulcers in connection with Disease of the Hip and Wrist Joints.
The dotted line, extending from the probe, indicates the extent to which the skin is undermined around the opening of the sinus.

Causes of sinus.—There are various reasons why these sinuses may not heal. One of the common causes of non-healing is the presence at the bottom of the sinus of a foreign body, more especially of a piece of dead bone. In such a case, suppuration is kept up around the foreign body, and the pus must find a channel for escape, and therefore keeps the sinus open. Indeed, if the orifice of the sinus be not sufficiently large, pus accumulates in the deeper parts, and presently finds its way to the surface elsewhere, leading to the formation of a fresh abscess, and subsequently of another sinus. The most common cause of sinus is, however, the presence of tuberculous disease in the wall, more especially where pyogenic organisms have entered the abscess cavity at the time of opening or subsequently. (*See* Fig. 19.) These tuberculous sinuses are extremely common after chronic abscesses have been opened, and if the wall of the sinus be examined, it is found to be lined by tubercles, just as the wall of a chronic abscess itself is. In such cases, although there may be no foreign body, the sinus may persist indefinitely, especially if pyogenic organisms be present, the tubercle having but little tendency to disappear. Where the entrance of pyogenic organisms is prevented, in most cases the tissues ultimately overcome the tubercular process, and the sinus heals. Other causes of sinuses are movement in the part, as where the abscess is formed between planes of muscles which are kept in constant movement, and imperfect exit for discharge, quite apart from the presence of a foreign body or of tuberculous disease. Where the opening is incomplete the septic pus does not escape freely, and thus a condition of inflammation is kept up, and the deeper portions of the wound are kept apart by the presence of the pus.

Treatment of sinus.—The treatment of a sinus consists essentially in the removal of the cause of the persistence of the opening; hence, in all cases the sinus must be opened up freely so that there shall be no difficulty in the escape of pus from the deeper parts, and if a foreign body, such as a piece of dead bone, be present, this must be removed. Where the cause of the sinus is muscular action, the part should be placed on a splint, or by other means be kept at rest; where it is imperfect escape of discharge, the opening must be enlarged and large drainage-tubes inserted, counter-openings being made if necessary. Where we have to do with a tuberculous sinus, the best treatment is to enlarge the wound and dissect away the walls of the sinus and any disease which may be present at the bottom. Where, from the situation of the sinus, this treatment is impracticable, the orifice should be opened up and the lining membrane thoroughly scraped away. In cases where the sinus leads to dead bone, which is comparatively easily accessible, it is well to make a very free incision along the track, and after having scraped away and removed as much of the disease as possible, stuff the wound with gauze sprinkled with iodoform, and make it heal from the bottom.

Varieties and causes of fistula.—Fistula is a condition where there is an opening in the skin and an opening in the mucous membrane, with a canal between them. It occurs in various parts of the body, especially around the orifices of the mucous canals. Thus there are salivary fistulæ, where there is an external opening leading into a salivary duct, the salivary duct again leading into the mouth. (*See* Art. XLIV., on AFFECTIONS OF THE MOUTH, Vol. II.) There are various congenital fistulæ, more especially in the neck, which are remains of branchial clefts (Art. XLII., on INJURIES AND DISEASES OF THE NECK, Vol. II.). We have intestinal fistulæ, where an opening communicates between the intestinal canal and the surface of the skin, this resulting most usually as the consequence of disease or injury, such as after strangulated hernia with gangrene of the bowel, after operations in the abdomen where the bowel has been opened, after the operation of colotomy, etc.; or it may be congenital where the intestine communicates with the surface, this being generally situated at the umbilicus. (*See* Art. XLVII., on INJURIES AND DISEASES OF THE ABDOMEN, Vol. II.) The most common fistulæ are the anal fistulæ, following abscess under the mucous membrane of the bowel, the pus afterwards making its way into the ischio-rectal fossa, and opening both internally into the rectum and externally through the skin. (*See* Art. XLIX., on DISEASES OF THE RECTUM, Vol. II.)

The chief reason why these fistulæ do not heal is the constant passage through them of substances from the mucous canal. For example, in the case of salivary fistula there is constant discharge of saliva, which prevents the healing of the wound; in the case of intestinal fistula there is constant passage of the bowel contents, and similarly in the case of the anal fistulæ. In the case of the congenital fistulæ, especially in the neck, the cause of the non-closure is due to the fact that the whole track of the canal is lined with epithelium, and consequently no healing is possible.

Treatment of fistula.—The treatment of the various fistulæ is considered in the accounts of the surgery of the various regions, and I need not therefore go into it in detail. It essentially consists either in laying the canal between the skin and the mucous membrane completely open, and causing healing to occur from the bottom, or in diverting the fluid which is passing over the canal and preventing the healing of the fistula, or in removing the epithelial lining of the canal. Thus, in the case of the salivary fistula, operations are carried out by which the saliva shall find a direct opening into the mouth; and when that is done, the opening in the skin may be refreshed and stitched up. Similarly, in the case of intestinal fistulæ, means are taken to make it more easy for the fæcal matter to pass along the intestinal tract, than to make its way out through the fistulous opening. Thus, where the fæcal fistula has resulted from gangrene after strangulated hernia, the contents of the bowel cannot find their way into the lower part of the bowel, on account of a spur of mucous membrane which acts as a valve over the orifice and the lower part of the canal; and steps must be taken to remove this spur of mucous membrane, either as was formerly done by the use of Dupuytren's enterotome or, if necessary, by freeing the two ends of the bowel and subsequently uniting them. In the case of anal fistula the only satisfactory treatment is to lay it freely open, scrape away, or even cut away the wall of the fistula, especially if it be in a tuberculous condition; and then stuff the wound for some days, and make it heal from the bottom. In the case of perineal fistulæ, which are almost always due to the presence of stricture, the first act in the treatment must consist in the removal of that stricture, either by dilatation or by urethrotomy, or in some other manner. (*See* Art. LI., on Injuries and Diseases of the Urinary Organs, Vol. II.) In cases where we have to do with congenital fistulæ, nothing short of complete removal of the whole track by dissection will effectually cure the trouble. Attempts at destroying the mucous lining of the track by scraping, or by means of caustics, almost invariably fail, probably from the fact that islets of epithelium escape the action of the remedies.

IV. ULCERATION.

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It has already been pointed out (Art. II. page 60) that inflammation, when it has reached the stage of the formation of granulation tissue, and is affecting a free surface, may terminate in ulceration. Instead of the healing process taking place there ensues a gradual extension of the sore, either with or without suppuration, owing to breaking-down of the tissue which forms its surface, the result either of degenerative changes from imperfect nutrition, or of actual gangrene of microscopic portions of the tissue.

True ulceration is an inflammatory process, and no breach of surface can be called an ulcer in which the inflammatory changes have not already gone on to the stage of granulation. This definition distinguishes true ulceration from a recently made wound on the one hand, or from the breaking-down of a tumour on the other.

Classification of ulcers.—There are *three* kinds of sores which are included under the head of ulcers.

1. In the first place we have those which are not due to any specific virus, but which are caused by various local disturbances which interfere with the vitality of the part, more especially disturbances in the circulation or in the innervation of the tissue. These are generally spoken of as *chronic non-infective ulcers*, the chronic character varying considerably in certain circumstances, some, indeed, almost deserving the title of acute ulcers.

2. The second group is composed of ulcers which result from the development in the part of a definite specific virus, more especially of the virus of tuberculosis or of syphilis; they are termed *chronic infective ulcers*. In these specific ulcerations there is a tendency to degeneration of the surface of the sore as the result of the action of the specific virus, quite apart from disturbances in the circulation or in the nervous supply of the part.

3. The third group consists of tumours of various kinds, more especially malignant tumours which have invaded the skin or led to sloughing of the skin over them, and the surface of which is breaking down; these are either cancerous or sarcomatous ulcers. From what has been already said it is evident that these sores do

not conform to the definition of true ulcer; they must, however, be referred to in describing ulcerative processes. They may be classed under the heading of *ulcerating tumours.*

I. THE CHRONIC NON-INFECTIVE ULCER.

Causes.—The causes which lead to the development of these ulcers are partly and, indeed, mainly *local*, partly also *general.*

Local causes.—The chief local causes that lead to ulceration are those which interfere with the circulation of blood in the part.

1. Anything which prevents the venous return, or which diminishes the arterial supply, interferes with the nutrition of the part, and if the part so affected be already the seat of a sore, that sore is very apt to break down and ulcerate instead of healing. The most common obstacle to venous return leading to the formation of an ulcer is the presence of *varicose veins.* Where the veins are much dilated, the valves become incompetent, and when the patient walks about, the weight of the column of blood interferes with the onward flow of blood from the part. Varicose veins are more especially likely to lead to the formation of an ulcer if the small veins in the skin be affected. Persons may walk about for a long time with enormously enlarged varicose veins, so long as the cutaneous veins are healthy, without developing a varicose ulcer.

2. Then, again, the presence of an *atheromatous condition of the arteries* will act similarly, in that a smaller quantity of blood is brought to the part and the nutrition of the part is thus impaired; and if we have the combination of atheromatous arteries with varicose veins, the probability is very great that ulceration will take place, more especially if a sore be actually present. Indeed, where the circulation is feeble or imperfect, the mere dependent position of the limb is sufficient to keep up or lead to the ulcerative process, quite apart from the presence of varicosity of the veins.

3. Apart from the presence of atheromatous vessels, anything which tends to *press on the vessels from without* will also tend to lead to ulceration. If, for example, a sore have formed, say as the result of an accident, in skin over loose connective tissue, and the part be allowed to hang down, or a more or less acute attack of inflammation supervene, the exudation which is poured out into the loose connective tissue presses on the vessels and interferes to such an extent with the circulation in the part as to lead to the formation of an ulcer, and, as we shall see presently, this is the chief cause of extension of the ulceration and the chief obstacle to healing in callous ulcers.

4. Further, a naturally *weak condition of the tissues* also favours the occurrence of ulceration, and hence it is that ulcers are much more frequent in old people than in young.

5. A similar condition is brought about by exposure to *cold*, especially in persons whose nutrition is imperfect, more particularly as the result of bad food and bad hygienic conditions. One of the

first effects of cold is to produce the condition known as chilblain; and partly as the result of local irritation of the chilblain, such as rubbing, and partly as the result of further exposure to cold, ulceration is apt to occur pretty rapidly.

6. Another local cause which leads to ulceration is *movement* of the part. For example, where a sore is situated over a muscle, or over a fascia which is in frequent movement, and more especially if the sore be of an oval shape and transverse to the long axis of the fascia, healing may not occur, and indeed ulceration may take place.

7. A very important cause of want of healing in a sore and of the occurrence of ulcerative changes is *interference with its contraction.* As will be seen later, in speaking of the healing process (page 205), one of the necessary occurrences in the healing of an open wound is contraction of the newly-formed granulation tissue as soon as the irritation has so far subsided as to permit it to develop into new fibrous tissue, and if this contraction be impossible, healing may come to a standstill; and, indeed, in the case of large sores, after coming to a standstill ulceration may actually take place and the sore again enlarge. Thus where a sore is situated over and adherent to a bone, and where therefore contraction cannot occur, ulceration is very apt to take place, and in any case the scar formed in such a situation is a weak one, and is very liable to break down on the slightest provocation; and similarly, where there was in the first instance a very large sore or an extensive burn on the trunk, the time may come when contraction during the healing process will be no longer possible, and when that is the case, the sore which up to that time had been healing readily, ceases to heal, or actually extends again. No doubt, in this case, we have in addition to the simple impossibility of contraction of the sore, another condition or factor to bear in mind, namely, that as the result of the contraction which has already taken place the blood supply to the centre of the sore is very much interfered with, and thus we have a combination of want of contraction with deficient blood supply to explain the occurrence of the ulcerative process.

8. Further, *irritation of a sore* from whatever cause, if long continued and of sufficient severity, may interfere with the healing process, and ultimately lead to the occurrence of ulceration. Thus application of injudicious lotions, such as carbolic lotion, to a granulating wound is very apt to interfere with the healing of the wound, and by setting up a considerable amount of irritation to lead to exudation into the deeper tissues, which presses on the vessels running to the part, and thus, partly as the result of the *irritation* of the lotion, and partly as the result of this interference with the circulation, the sore which in other circumstances would have healed readily enough ceases to heal and becomes an ulcer. The same holds good with regard to mechanical injury, such as the rubbing of dressings over the part, too great pressure, and so forth.

9. Perhaps one of the most frequent locally irritating causes which leads to the formation of an ulcer is the *septic condition of*

the discharge which comes from the wound. These decomposing discharges are often highly irritating, and if anything interfere with the rapid escape of this discharge from the surface of the wound, the secretions accumulating on the part act in the same way as other irritating chemical substances, such as carbolic acid, and lead to ulceration. Thus, one of the most important points to attend to in the treatment of a sore in which there is marked sepsis is that crusts shall not be allowed to form on the surface of the wound, for if they do, the decomposing secretions accumulate beneath these crusts, and almost to a certainty when they are picked off it will be found that the healing process has come to a stop underneath them, and that, in fact, ulceration is taking place.

10. Then again, the ulcerative process may be set up by some *accidental infection of the wound* either leading to a very acute attack of inflammation, in which case the sore rapidly breaks down —what is known as the inflamed ulcer—or, where some specific organism, such as that of diphtheria or phagedæna, has entered and grows on the surface of the granulations, leads to a semi-gangrenous process. In the latter cases the surface of the wound becomes converted into a pulpy mass, which consists of numerous cells and bacteria, and the parts which have already healed, and subsequently the skin around, become rapidly destroyed, as the result of the spread of these organisms into them.

11. Lastly, we have ulcers occurring as the result of *deficient innervation* of the part. For example, in cases of paralysis, or in cases where there is as yet no actual paralysis, the occurrence of rapid ulceration in the paralysed part is no doubt to some extent explained by the fact that, owing to the loss of sensation, the patient is apt to press on the part too long and too heavily. This, however, does not altogether account for paralytic ulcers, for it is found in cases of hemiplegia, where the patient is lying absolutely motionless, that ulceration in the form of bed sores will occur much more rapidly and extensively on the paralysed side than on the other, although the amount of pressure on the two sides has been the same, and also in infantile paralysis, where sensation is not affected, these paralytic ulcers are very apt to occur. Of the various ulcers which are attributed to deficient innervation, the most peculiar and the one most deserving of notice is that which is known as the *perforating ulcer of the foot.* (*See* Art. XXVII.)

Constitutional causes.—Of constitutional conditions which lead to the formation of ulcers, perhaps the most important is the existence of *diabetes.* In such circumstances, ulceration and also gangrene are very apt to occur, more especially in the lower extremities, after very slight injury, partly, no doubt, because the tissues of a diabetic individual are particularly prone to become infected with micro-organisms, partly also because the arteries are very apt to become affected by endarteritis, and possibly in part also owing to interference with the proper innervation of the body. However that may be, there is no doubt that in cases of diabetes, ulceration occurs with marked rapidity, and is very apt to pass into gangrene. In *scurvy*, again, ulceration is not unlikely to take place over parts where hæmorrhage has occurred subcutaneously, more particularly towards the upper part of the leg, these ulcers being especially liable to bleed. Neurotic women are also apt to become affected with ulceration, one form of which is known under the name of the irritable ulcer, and this is chiefly associated with *disorder of the uterine functions.*

Results following the non-infective ulcer.—It is very important to recognise the cause of the ulcerative process, and to carry out proper treatment for its arrest, because, if it be allowed to go on, it may lead to very serious local disturbance. For example, where an ulcer occurs over muscles, inflammation of the muscle beneath takes place, resulting in exudation among the muscular fibres and permanent interference with its proper action. This is equally the case where the ulcer is situated over tendons, where the inflammation spreads to the tendon sheaths, leading to adhesion of the tendons to their sheaths and imperfect movement of the parts. Another disadvantage of the continuance of ulceration is that the circulation in the part below is interfered with, and in this way the foot, for example, may be rendered useless. This is most marked where the ulcer surrounds the leg more or less completely, in which case the contraction of the sore tends to constrict the leg and to press on the blood-vessels, and thus leads to imperfect venous and lymphatic return and imperfect supply of blood to the part, and hence to permanent loss of function of the foot; and this contraction is also objectionable, even when the ulcer can heal fairly readily, if the sore be situated over a joint, it which case marked contraction of the joint may take place; or if it be situated, say, in the neck, where the features may become distorted, from the skin being pulled on during the healing process; or about the axilla, where the arm may become bound to the side. (*See* Fig. 41.)

Apart from these objectionable local results, the presence of an ulcer implies a certain amount of danger to the patient. Thus, the veins in the vicinity of the ulcer may become inflamed, and extensive phlebitis and thrombosis result, and, of course, if this inflammation be of a septic character, very serious or even fatal consequences may ensue. In addition, the presence of an open sore presents a point of entrance for various infective diseases, more especially for the erysipelas virus; and lastly, I may mention that in certain cases where sores have existed for a long time, epithelioma may develop at their margins, and may lead to the death of the patient as the result of this cancerous growth.

Varieties of the non-infective ulcer.—Chronic non-infective ulcers are divided into several classes, according to their appearance and tendencies. The following are the chief groups.

1. **Simple ulcer.**—The simple ulcer is a sore which does not heal on account of various local troubles, such as pressure, friction of

dressings, irritating applications, movement of muscles underneath, inability to contract, etc. In these simple ulcers the surface of the sore is nearly on a level with the surrounding skin, and it is fairly well covered with granulations, which are of a yellowish or brownish red appearance. The margins of the sore are sharp, and the surrounding parts are œdematous and firm. These sores are generally situated about the middle of the lower third of the leg, and where no proper care is taken they may extend fairly rapidly. These ulcers may in certain cases become the seat of an acute inflammation, as the result of septic infection, and we have :—

2. The form which is known as the **inflamed ulcer.** The inflamed ulcer is a sore which has become the seat of acute inflammation as the result of some mechanical or chemical irritation, or bad methods of treatment, or, more especially, of some septic infection of the surface. In these cases the surface of the ulcer becomes intensely red, bleeds readily, secretes large quantities of pus, extends with great rapidity, and is not infrequently covered with small pieces of actual gangrenous tissue; the skin around is also bright red and œdematous, the margins are irregular and eaten away, and we not uncommonly find fresh ulcers forming around the margins of the original sore, and merely separated from it by bridges of inflamed skin. These ulcers are acutely painful.

3. **Irritable ulcer.**—This form of ulcer is especially met with in neurotic women. It is usually situated above the external malleolus. The base of the ulcer is somewhat elevated, not unlike a boil, the apex of which has been cut off, and it is intensely tender to the slightest touch, and in some cases bleeds readily. It is generally associated with menstrual disorders.

4. **Weak ulcer.**—Simple ulcers, or even healing sores, are very apt to become weak as the result of defective blood-supply, either from too small a quantity being sent to the part, as in cases where the vessels are diseased, or where there is some obstruction to the return of the blood, or from deficient quality of the blood, for example, where some constitutional disease is going on. In this form of ulcer the granulations become smooth and yellowish, the secretions thin and small in amount, and very apt to dry up and form scabs. The edges are pale and flat. In other cases the edges and granulations become œdematous, and this more especially takes place where there is some general cause of œdema, such as Bright's disease, or valvular disease of the heart, or some local interference with the circulation, such as the presence of varicose veins, compression of the veins from the contraction of the sore, etc. A third form of weak ulcer is one in which the granulations show excessive growth; it chiefly occurs where there is interference with the contraction of the sore. In such cases the granulations become prominent, vascular soft, and bleed readily, and there is then the condition which is popularly spoken of as "proud flesh."

5. **Varicose ulcer.**—The varicose ulcer is a form of ulceration which depends on varicosity of the veins, more especially of the

smaller veins in the skin, and it chiefly occurs in oldish people and those who stand much. As the result of the varicose condition of the veins, the nutrition of the skin becomes imperfect, and eczematous inflammation is very apt to occur in these circumstances. The skin is very itchy, the patient scratches it and produces a wound, which then gradually enlarges and takes the form of an ulcer. In certain cases, however, the varicose ulcer commences by the formation of a small abscess around an inflamed vein, which bursts through the skin, and then leads to extending ulceration. These are the cases in which bleeding is very apt to occur from the formation of an opening into the vein itself. However produced, these varicose ulcers are usually in the first instance small and superficial ulcers, resembling in their general appearance simple ulcers with œdema of the tissues around, and they often have prominent, soft, or œdematous granulations, not uncommonly surrounded by a patch of eczema. If the patient continue to walk about, the congestion of the part is kept up, and the tissues around become thicker and thicker, ultimately leading to the condition of a callous ulcer.

6. **Callous ulcer.**—Any ulcer of the leg may become a callous ulcer under certain conditions, more especially those which interfere with the venous return. Hence, a callous ulcer is most commonly associated with varicose veins, and is the further development of the varicose ulcer as the result of the continued interference with the venous return. In such cases, from the dependent position of the limb, œdema of the part takes place, lymph is exuded into the meshes of the cellular tissue and coagulates, and may even become organised, with the result that the parts surrounding the sore become hard and the arterioles are pressed upon, and the nutrition of the part is much interfered with. Hence the skin and tissues around a callous ulcer are much thickened and hard, and do not pit on pressure. The base of the ulcer is depressed considerably below the surface of the skin owing to this great thickening of the parts around. The base is also fixed and contraction is impossible. The surface of the sore is pale and has only few and very imperfect granulations, and secretes a small quantity of thin fluid. The margins of the sore are elevated, thickened, and white (Fig. 20).

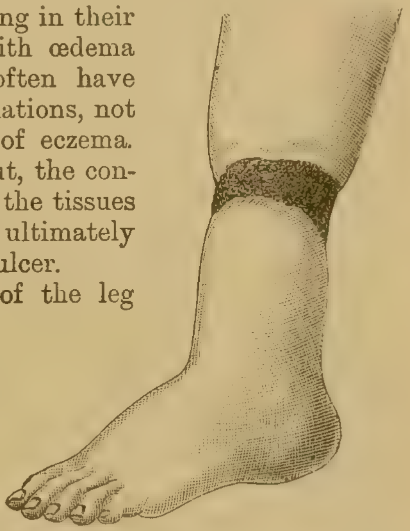
Fig. 20.—Callous Ulcer, completely surrounding the Leg. The figure shows the thickening of the surrounding tissues, the apparent depth of the sore, and the absence of any attempt at healing. This case was cured by skin grafting.

7. In certain cases there is developed what is known as the **hæmorrhagic ulcer**, which occurs more especially in patients who are suffering from scurvy. In these cases the surface of the sore is

swollen, red, and bleeds very readily, blood sometimes coagulating on, and adhering to, the surface, forming projecting clots.

8. **Diabetic ulcer.**—In patients who are affected with diabetes it is not uncommon for wounds resulting from scratches or cuts to extend rapidly, and lead to the formation of an ulcer, the result partly of the condition of endarteritis present in this disease, and partly of the great proneness of the tissues to septic infection. These ulcerations spread rapidly, the skin around is inflamed and red, and most commonly, in addition to the ulceration, there are also visible sloughs of the skin or subcutaneous tissues.

9. **Ulcers the result of pressure.**—Pressure ulcers especially occur on the sole of the foot, and result from long-continued but not necessarily severe pressure. The first effect of the pressure is to lead to thickening of the epithelium, and the formation of a callosity. If the pressure be continued, inflammation and suppuration occur beneath this callosity; and when the thickened epidermis is removed, an ulcer is seen, which heals with the greatest difficulty, and which extends if the pressure be kept up. The great characteristic of these sores is the marked thickening of the epidermis surrounding them, the sore itself being considerably below the level of the skin, very vascular, and with a tendency to exuberant granulations. The "bed sore" is considered in the article on Gangrene (page 134).

10. **Ulcers due to deficient innervation.**—In limbs which are paralysed it is not uncommon to find atonic ulcers, which develop with considerable rapidity, are painless, sometimes multiple, and often covered with imperfect granulations. These ulcers chiefly occur about the phalanges of the fingers and toes, and also on the sole of the foot, and are no doubt in many cases the result of long-continued pressure, and are usually described as pressure ulcers. In connection with these ulcers we may describe especially the variety which follows.

11. **Perforating ulcer of the foot.**—This occurs in parts of the foot which are subject to pressure, more particularly under the heads of the metatarsal bones, chiefly that of the great toe. In the first instance these ulcers present the appearance of ordinary pressure ulcers, beginning as a callosity, followed by suppuration underneath, but differing from the ordinary pressure ulcers in the rapidity with which they extend into the substance of the tissues. These ulcers generally attack men over forty years of age, and are not necessarily associated with any paralytic condition of the limb, being usually associated with tabes, or possibly in some cases with commencing peripheral neuritis. The ulcer may extend in depth till it reaches the bone, and then the bone becomes the seat of a rarefying osteitis, and ultimately may completely disappear, the metatarso-phalangeal joints also being opened. The ulcer thus presents a funnel-shaped appearance, its base being generally covered with reddish, warty granulations, and the secretion being of a very foul character. The epithelium spreads down the sides of the funnel, and there is great proliferation of the epidermis, so that the cavity of

the ulcer becomes filled with masses of decomposing epidermis. These ulcers are supposed by some to be essentially pressure ulcers, starting in the first instance like the ordinary pressure ulcer, with the formation of a callosity. The pressure is kept up by the growth of the epidermis down the sides of the ulcer, thus leading to the formation of a hard circle, which presses on the deeper parts. These ulcers are very chronic, and will not heal under rest or the ordinary methods of treatment, the reason being the presence of this epidermic growth down the sides of the ulcer, preventing the adhesion of the two sides to each other. (*See also* Art. XXVII.)

12 **Phagedænic ulceration.**—Any of these ulcers, more especially the ones earlier described, may become attacked by some specific infection, such as the diphtheritic or phagedænic poison, with the result that there follows the development of what is known as a diphtheritic or phagedænic ulceration. I shall speak of phagedæna more especially in connection with gangrene; and it is not necessary, therefore, to do more here than simply to mention the existence of this class of ulcers. (*See* page 149.)

Treatment of the chronic non-infective ulcer.—The conditions which are necessary for the healing of an ulcer are that the surface of the sore must become level with the surrounding parts, that the margins of the sore must be movable and contraction must be possible, and that the granulations on the surface must be healthy. The principles of treatment are therefore in the first place to get rid of the various causes which are keeping up the ulceration, and in the second place to improve the condition of the surface and the margins of the ulcer. These principles may be arranged under four heads—

(1) The first essential in the treatment of all ulcers is *rest*. As I have already said, any movement of the part will tend to keep up the ulceration; and therefore all movement of the affected area must be completely abolished. Hence in most cases the limb should be fixed on a splint, so arranged as to prevent the movement of the joints above and below. Further, it is of essential importance in the treatment of ulcers to place the circulation of the blood through them under as favourable conditions as possible. Hence the dependent position of the part, hindering as it does the venous return, must be abolished, and consequently, for the most rapid healing of ulcers, rest in bed is advisable; and not only rest in bed, but *elevation of the limb*, so that the sore is at a higher level than the heart. The improvement which follows absolute rest and elevation of the limb is extremely marked in many instances, more especially in cases of callous ulcer, where, without any other special treatment, the thickened condition of the tissues around will rapidly subside, and the surface of the sore become healthy and on a level with the surrounding skin. The return of the blood from the part may also be favoured by *massage*, which should be combined with rest in the elevated position. In employing massage in the case of callous ulcer, the area operated on should in the first instance be the upper part

above the ulcer, this area being extended downwards as the tissues around become softer, and the lymphatic circulation becomes more fully re-established. With the view of getting rid of this œdematous condition, and of improving the circulation, *pressure* is also a good deal employed, especially by means of indiarubber bandages; but this does not materially hasten matters, as compared with the effects of rest and the elevated position.

(2) Another very essential point in the treatment of all ulcers is to get *rid of all the causes of irritation* of the surface of the sore, that is to say, to get rid of irritation from movement of dressings, and to avoid the presence of any irritating fluid in contact with the surface of the sore. If antiseptic lotions be employed with the view of cleansing the sore, they should be of a weak and unirritating character, such as boracic acid solution (saturated), or weak sublimate solutions (1 to 4,000 and 1 to 6,000). The employment of irritating solutions such as carbolic acid, especially if strong, will interfere very greatly with the healing process.

The most important irritating substances which have to do with the extension of the ulcerative process are the products of sepsis in the discharges from the sore, and it is, therefore, one of the most essential points at the commencement of the treatment of an ulcer *to remedy*, as far as possible, *the septic condition.* In order to do this, the following is the best method of procedure, and it should be carried out in all cases at the commencement of the treatment of an ulcer, and more especially at the commencement of the treatment of a case of callous ulcer. In the first place, the skin around the ulcer for a considerable area should be thoroughly disinfected, because to disinfect the surface of the ulcer alone and leave the infected skin in the neighbourhood would mean reproduction of the sepsis in a very short time. In the first instance, the skin around is thoroughly washed with soap and water, and all hairs are shaved off. It should then be saturated with turpentine in order to dissolve off the fat, and then the skin is very thoroughly scrubbed with soap and 5 per cent. carbolic acid solution, or, better still, with that lotion containing a 500th part of sublimate in solution, a nail brush being employed with the view of removing all the loose epithelium on the surface. The disinfection of the skin around a septic ulcer is a matter of considerable difficulty, and it is well to repeat the process on one or two days in succession. The disinfection of the surface of the ulcer itself is also by no means an easy matter. The first method employed by Sir Joseph Lister was to apply a solution of chloride of zinc, 40 grains to the ounce, to the surface of the ulcer, but I think that in ordinary circumstances the most satisfactory results are obtained by the use of undiluted carbolic acid. The pain caused by this application is, even at the moment, not greater than that caused by the chloride of zinc, while it very quickly passes off, as the result of the anæsthetic action of the carbolic acid. In cases where there are prominent granulations or actual small sloughs on the surface, it is well before applying the carbolic acid to scrape the surface with a sharp spoon (in such a case, of course, the patient being placed under an anæsthetic), the sore and the skin around being at the same time disinfected. Cyanide gauze soaked in 1 to 2,000 sublimate solution should be applied daily for two or three days. This no doubt injures the surface of the sore, but a delay of two or three days before the commencement of the healing process is immaterial, while it greatly adds to the certainty of disinfection. Disinfection of a foul sore may also be obtained by the use of strong carbolic oil 1 to 5, lint dipped in this oil being carefully packed into all the recesses of the ulcer and the dressing changed every day ; it will usually be found in the course of a few days that the sepsis has been completely got rid of. Iodoform is much used by some surgeons as a means of disinfecting ulcers, but as I shall mention, when I come to speak of the treatment of wounds, its antiseptic action is very slight, and it is really of very little value for this purpose. (*See* page 225.)

(3) Having got rid of the septic condition of the sore, one must then cease the employment of irritating applications, otherwise one would interfere with the healing process, and among the best methods of *treating an ulcer subsequently to its purification* is the use of boracic acid in the form of a lotion (saturated solution) and boracic lint which has been wrung out of this same solution, the mechanical irritation of the lint on the sore being prevented by the interposition of a piece of Lister's protective oiled silk, which is first dipped in 1 to 20 carbolic lotion to disinfect the surface, and afterwards in the boracic lotion to wash away the irritating carbolic acid. This dressing is changed daily for some time. In cases where there is much discharge, it is well to cut a few holes in the protective, so as to allow the discharge to escape into the lint, and in any case the protective should not be larger than the sore itself, and should be well overlapped in all directions by the antiseptic lint outside.

Where there is a tendency for the granulations to become œdematous, this dressing is unsuitable, because the fluids are confined beneath the protective and the œdematous condition is increased. In such cases the œdematous granulations should be destroyed by the application of nitrate of silver, and then either some dry dressing or some of the antiseptic ointments should be used. The objection to the dry dressing is, in the first place, its mechanical irritation, and in the second place, that when it is removed, one is very apt to peel off the delicate layer of epithelium which has been forming around the edge. Of the antiseptic ointments, perhaps the best is boracic ointment, but it must not be used of the full pharmacopœial strength, because in most cases this does not permit satisfactory healing. As a rule, it is well to dilute it with vaseline or with the ointment basis to about a quarter of its original strength. This ointment should be spread on cambric or thin butter-cloth ; it should not be spread on lint, as is the usual custom, because when the lint is stretched cracks form in the ointment, and thus a complete antiseptic layer is not applied to the surface of the sore. Outside the butter-cloth, boracic lint or other antiseptic dressing is applied, and the whole fixed on with a bandage.

Where the ulcers are very painful, or where there are sloughs on the surface, or where there is any acute inflammatory action, it is well to employ the boracic lint in the form of a fomentation, that is to say, the boracic lint is applied wet, the lotion being only very imperfectly squeezed out of it, and outside the lint a piece of mackintosh cloth is placed, overlapping the lint in all directions. This should be changed twice daily, but should not be continued after the irritable condition of the sore has ceased, or after the sloughs have separated, otherwise the granulations are very apt to become œdematous, and one form of weak ulcer may be produced.

(4) A fourth object in the treatment of ulcers common to all of them is that attempts should be made to get *rapid healing* with as little contraction as possible, and further, to obtain a scar which will subsequently be sound. In the case of ulcers affecting the lower extremities, especially in oldish people, the scar obtained where an ulcer is allowed to heal of itself is usually weak, and commonly breaks down again if the patient stands or walks much, and this is more especially the case where imperfect contraction of the sore has taken place, as, for example, where the base of the sore is adherent to the bone. Where a really sound scar is desired—and it is desired in all cases of ulcer of the leg—the best method is to employ skin grafting, preferably by the plan introduced by Thiersch.

Skin grafting.—The earliest method of skin grafting is that known as *Reverdin's method*, which is, however, really epidermis grafting rather than true skin grafting. In his plan, minute portions of the superficial layer of the epidermis were shaved off and applied at numerous places over the surface of the sore, at a distance from each other of about a quarter to half an inch. These grafts were left undisturbed, care being taken not to wash them off, and in the first instance not to change the dressing for some days, and for the best success in this plan it is necessary that the sore should be aseptic. These minute grafts of epidermis adhere to the surface of the granulations, and very soon we find spreading around each graft a narrow line of young epidermis, so that instead of the sore having to heal altogether from the edge, the epidermis sprouts over it from numerous points on the surface. The result is that much more rapid healing is obtained than by the ordinary treatment of the sore, and consequently less granulation tissue is formed and the resulting contraction is correspondingly less. It is necessary to place these grafts pretty close together, because it is found that the epidermic formation around each graft only extends to an area of about the size of a sixpence, and then seems to come to a standstill. While the sores so treated heal rapidly, and the resulting contraction is less, there is, nevertheless, a large amount of contraction, while the resulting scar is not materially stronger than that which follows healing without this method of epidermic grafting.

With the view of obtaining a sounder scar and of avoiding the contraction, it is necessary to employ much larger and thicker portions of the skin, and to leave no interval at all between the grafts. In *Thiersch's plan* about half the thickness of the skin is used, and the result of his method is extremely satisfactory. In describing it we have to consider: first, the preparation of the sore, and, secondly, the preparation and application of the grafts. As regards the preparation of the sore, it is in the first instance necessary to get rid of all existing sepsis, and this can be done by the method of disinfection which I have described. The wound must then be treated till the surface of the sore has become quite healthy, as indicated by the presence of healthy granulations and of commencing healing around the edges. Some surgeons allow this healing process to go on for several weeks, and repeatedly rub the granulations with nitrate of silver with the view of obtaining a firmer and better surface. For my own part, I do not think that it is necessary to wait or to prepare the surface in this way. All that is requisite for the success of the application is that the sore shall be healthy (as indicated by the appearance just described) and aseptic. Having obtained a healthy sore, the soft layer of granulations on the surface should be scraped away, the deeper newly-formed fibrous tissue, which is highly vascular, being, however, left behind. By scraping away the surface of the granulations, a mass of tissue is removed which would organise into fibrous tissue and subsequently contract, while, at the same time, a smooth, firm base is obtained on which to lay the grafts. In my opinion, not only should the granulations be scraped away, but it is well also to remove the line of cicatrisation which has already formed. I have found that where this is not done, although the area covered with skin grafts remains quite sound after the patient walks about, a line of ulceration is very apt to occur around, corresponding with the line of cicatrisation which had formed as the result of the natural healing process, and I therefore deliberately cut away the new epithelial layer so as to remove all the parts that have healed naturally. The surface of the sore having been prepared in this way, steps must be taken to arrest the hæmorrhage completely before the grafts are applied, and this is best done by taking a piece of protective which has been disinfected in carbolic lotion and subsequently in some weak antiseptic such as weak sublimate solution, and placing it next the sore, and then, by means of sponges, either held firmly over the part by the assistant or bound down by means of a bandage, the pressure is kept up till the bleeding has stopped. The object of interposing the protective between the sponges and the sore is in order that the bleeding shall not be started again when the pressure is removed, and this is very apt to occur where no protective is used, because the sponges stick to the surface.

While the bleeding is being arrested in this way, the cutting of the grafts may be proceeded with. In the first instance, the skin which is to be removed for the purpose of skin-grafting should be thoroughly disinfected in the manner just described in reference to the treatment of the ulcer, and it should also be carefully shaved, because the presence of hairs is very apt to interfere with the proper healing of the graft. The skin is most conveniently taken from the front or sides of the thigh, but of course it may be taken from any part of the body which is healthy and suitable. The skin of the part is put on the stretch vertically by the assistant, and transversely by the surgeon grasping the limb, and the surface should be made as flat as possible. A broad razor is then taken, which is always kept wet with boracic lotion, otherwise the graft is very apt to tear, and strips composed of half the thickness of the skin, as broad and as long as possible, are cut. While the various strips are being prepared, those already cut should be left on the part bathed in the blood which exudes from the cut surface. As soon as the bleeding from the ulcer has stopped, these strips are transferred to the surface of the sore, and are very readily spread out by means of a couple of probes, care being, of course, taken that the deeper surface of the graft is placed next to the surface of the sore. These grafts should be applied close together; in fact, their edges should overlap, and they should also overlap the edge of the skin, and in this way the whole surface of the ulcer is entirely covered with grafts. When this is completed, narrow strips of protective disinfected in the first instance in carbolic lotion, and subsequently washed in weak sublimate or boracic lotion, should be pressed firmly over the surface of the grafts, so as to expel any air or blood which may have collected beneath during the process of skin-grafting; and the whole surface is then covered with a layer of protective, outside which a mass of cyanide gauze or other antiseptic dressing is applied. In the first instance, this dressing should be left on for at least five days, by which time the grafts will have sufficiently adhered to withstand the washing with the lotion. Subsequent dressings must be carried out at necessary intervals, and after a week or ten days, when the grafts have firmly adhered, I generally give up the moist dressing for some antiseptic ointment, such as weak boracic ointment. As regards the part from which the grafts have been taken, it may be dressed at once with weak boracic ointment and boracic lint outside, or the protective and boracic lint dressing just described may be employed. These parts generally heal without any difficulty whatever and with great rapidity, and it is possible after a time to utilise the same place for subsequent skin grafts.

The third method of skin grafting consists in the employment of the *whole thickness of the skin*, but the results obtained by this method are not nearly so satisfactory as those got by Thiersch's plan. In the first place a very large amount of skin is requisite to cover a comparatively small sore; on account of the contraction of the skin, it is also necessary to cut the original strip into small pieces, and even then one is very apt to find that the central portion of the graft sloughs, no doubt because the skin curls up and this central part is not kept in contact with the raw surface.

Whichever method of grafting is employed, the patient should be kept in the recumbent position for a long time. In the case of

Thiersen's grafts it has been found that several months elapse before absolutely complete union—more especially union by elastic tissue—has taken place between the skin and the part beneath, and if the patient be allowed to walk too soon, hæmorrhage or œdema is very apt to occur beneath the surface of the graft and lead to its ultimate detachment. From three to six months is generally the time that should be specified as necessary for the patient to lie in bed, or at any rate to keep up the leg, after the commencement of the treatment.

Treatment of special forms of the chronic non-infective ulcer.—It may be well to indicate one or two points with regard to the treatment of some of the special forms of ulcer which have been referred to.

In the case of the *inflamed* ulcer, it is necessary to take active measures with the view of combating the inflammation, and, perhaps, the most successful is the employment of free incisions into the margins of the ulcer, which act by locally depleting the part and also by permitting the escape of exudation. Where bridges of skin are present, it is of especial value to cut them across. These bridges would very quickly give way if they were left alone, whereas by cutting them across the portions of skin may be saved. As regards local applications in these cases, it is best to employ the boracic fomentations in the manner already described, and, of course, all the other principles of treatment come into play.

In the case of the *irritable* ulcer, it is well to destroy thoroughly the surface of the sore by rubbing it with nitrate of silver, and to attend more especially to the menstrual functions.

In dealing with the *weak* ulcer, the cause which leads to this condition should be sought for and removed, more especially adhesion of the ulcer to the bone beneath, or any other cause of difficulty in contraction. In some cases, healing of these ulcers has been obtained where all other means have failed by resecting a portion of the bone or a joint, and thus permitting contraction of the sore. Thus, in sores about the elbow joint, excision of that joint has led not only to the healing of the sore, but also to a useful arm; and even in the case of ulcers of the leg, satisfactory results have been obtained by removing one or two inches of the tibia and fibula, shortening the leg, and at the same time permitting a sound healing.

The *varicose* ulcer must be treated on the lines already laid down, and as soon as the sore has healed, either of itself or preferably by skin grafting, the cure of the varicose veins themselves by operation must be carried out. (*See* Art. XXIV.) This operation must be delayed till the sore has healed, otherwise, if there be any sepsis, the wounds might become infected, and very serious results might follow.

In the case of the *callous* ulcer, the chief obstacle to healing is the callous condition of the surrounding parts, and the surgeon's first efforts must be directed to getting rid of this condition. As a matter of fact, if the part be put at rest, the leg elevated and the sore rendered aseptic, this callous condition will quickly subside, so that in the course of two or three weeks the sore will present a healthy appearance, and healing will commence. Where it is desirable to expedite matters, or where the thickening of the tissues does not yield to these measures, perhaps the best method is to apply one or more blisters. These blisters should be applied over the callous area around the sore, but it is not advisable to place them over the actual surface of the sore, otherwise the cantharides is very apt to become absorbed and produce congestion of the kidneys, and as in these cases it is not uncommon to find that Bright's disease is present, this congestion might lead to very serious results. The effect of the blister is to set up inflammation of the skin, and consequently more rapid circulation through the part and more rapid absorption by the lymphatics, and in a few days after the application of a blister it is usually found that the thickening has very markedly diminished or even disappeared.

Another method which is employed to get rid of the callous condition is the use of pressure, either in the form of strapping—the strips of strapping beginning behind the leg, and the ends crossing over the surface of the ulcer, a hole being cut in the strapping to permit of the escape of discharge—or in the form of Martin's elastic bandage, which is of considerable advantage. Massage, also, is a rapid method of getting rid of the effused material, the parts first subjected to the treatment being those above the ulcer, and subsequently the parts below. The callous condition having been got rid of, and the sore having become healthy, skin grafting should be employed, and if, when the wound has healed, varicose veins are found, they should be treated by operation.

Callous ulcers occur more especially in the very poor, who are unable to give the necessary time for the thorough and sound healing of the sore, and who must therefore often be treated as out-patients. In that case one hardly hopes for a cure, but rather for alleviation of the pain and discomfort, and arrest of the ulcerative process. If the patient is to be allowed to walk about with a callous ulcer, the first essential, after having rendered the sore aseptic, is to give the part adequate support, so as to aid the return circulation; and for this purpose the use of Martin's bandage is the plan which has met with the greatest favour. Originally it was applied to the leg, beginning at the foot, and passing upwards without any dressing being placed between it and the sore; and it was taken off at night, washed, and thoroughly dried, no bandage being employed while the patient was in bed. The objection to this is, however, that the bandage becomes very foul, and thus great irritation of the sore is produced; and further, as the perspiration is confined beneath the bandage, an eczematous condition is very apt to form. With the view of avoiding the latter objection, Martin's bandages are now for the most part perforated with holes, so as to allow the evaporation of the sweat; but I believe it is also advisable in all cases to apply a suitable antiseptic dressing to the sore before putting on a Martin's bandage. So long as no greasy application is employed, which would destroy the india-rubber, there is really no objection to proper and efficient dressing of the sore beneath the bandage. In applying the Martin's bandage, it should be put on before the patient gets out of bed, and it should be placed on the limb quite loosely and smoothly, the bandage being simply unrolled around the limb, reverses, of course, being made wherever necessary. If the bandage be put on tightly, the accumulative action of the turns leads to the upper part becoming too tight when the patient has walked about for a short time.

Unna has introduced, in preference to the Martin's bandage, an arrangement by which the ordinary bandages are stiffened, so that they maintain their position and keep up the support. In the first place he thoroughly disinfects the skin, washing it with soap and water, and subsequently with antiseptic lotions, and then powders it and the sore with iodoform. He then takes a double-headed bandage and applies it to the limb, commencing from the middle of the sore. This bandage is porous, and over it he rubs a mixture of gelatine and glycerine—10 parts of gelatine, 40 parts of water, 40 parts of glycerine—with some oxide of zinc. This is melted and rubbed into the bandage, and before it is set another bandage is dipped in hot water and applied. The dressing solidifies, and forms a firm support to the leg; while it does not have the weight of plaster of Paris, and the pressure is more evenly distributed than with Martin's bandage. This dressing is renewed, according to the amount of discharge, usually at first every other day, but as the discharge diminishes, at less frequent intervals. It is readily removed by putting the patient's leg in a pail of warm water, which melts the gelatine, and allows one to unwind the bandage quite easily. When an ulcer has healed, whether by skin grafting or naturally, it is well in all cases to support the part for some time with a light bandage, and, best of all, with Unna's bandage.

In the case of *paralytic* ulcers stimulant applications should be used, in addition to the ordinary methods of treatment, more especially the application of spirits of wine to the parts around. As a dressing, balsam of Peru sometimes acts very well.

In dealing with *perforating ulcer* of the foot very radical measures are necessary in order to obtain healing. One may place such a sore at rest, and in an elevated position, for a long period of time without the slightest attempt at healing taking place, because, as I have already pointed out, the epithelium has spread down the sides of the funnel. Hence, in order to obtain healing, it is necessary to cut away the edges and sides of the ulcer, and this must be done very freely. The bottom of the ulcer should then be scraped, and the whole cavity stuffed with cyanide gauze, sprinkled with iodoform, the patient, of course, being kept in bed.

In the case of the *pressure* ulcer, also, it is necessary to remove the callosity on each side of the ulcer.

The *phagedœnic* ulcer requires energetic destruction of the gangrenous tissue, and this is best carried out by means of the actual cautery, the phagedænic material being scraped away in the first instance, and the part left being then very thoroughly cauterised. Some surgeons have advocated the use of caustic potash or nitric acid in preference to the actual cautery, but nitric acid especially seems to be inefficient, in that it leads to coagulation of the albumen, which stops the penetration of the acid. Subsequent to the application of the destructive agent, a dressing of strong carbolic oil (1 to 5) is perhaps the best.

II. THE CHRONIC INFECTIVE ULCER.

The chief ulcers which belong to this group are those which are due to tuberculosis and syphilis. These will be described in detail in the proper articles, and I need only refer in one or two words to the chief points. (*See* Arts. XVI. and XX.)

As regards the ulcerations of the skin due to tubercle, we have the condition known as ordinary **tuberculous ulcer**, which is characterised more especially by undermining of the skin for a considerable distance around the edges of the ulcer, this undermined skin being extremely tender if a probe be pushed in under it, and being of a reddish colour (Fig. 19). The surface of the ulcer is generally pale, without proper granulations, and with caseating material here and there. These tubercular ulcerations of the skin usually follow the formation and bursting of an abscess, either in the subcutaneous tissue, in a gland, or in some deeper part; and the great difficulty in healing, apart from the presence of the specific tuberculous disease, is the presence of this very thin undermined skin, which has no tendency whatever to adhere to the deeper parts. This difficulty is partly due to the extreme thinness and want of nutrition of the skin; in some cases, also, to the fact that epithelium has already spread over the deeper surface, starting sometimes from the bottom of a hair follicle which has been opened into, and thus we have an epithelial-covered surface opposed to the granulating surface. Where healing does occur, portions of this undermined skin usually slough, while the rest shrinks and forms a lumpy, unsightly scar. (*See* Art. XVI. page 361.)

The other typical ulceration of the skin is the result of the ulcerating form of **lupus**, the character of this being a soft, pale base, with imperfect granulations, and around the edge of the ulcer the presence of numerous nodules, often presenting the typical apple-jelly appearance, which again break down and thus lead to the extension of the sore (Fig. 21). There are various forms of lupoid ulceration of the skin, but the great characteristic of them all is what I have described, namely, the rough, imperfectly granulating base, and the presence of these nodules around, sometimes forming a very marked, thickened edge. (*See* Art. XXVIII.)

Syphilitic ulcers, again, are most usually the result of the ulceration of gummata, which have formed in the skin or the subcutaneous tissues. These gummata, when they reach the surface and burst, lead to loss of the skin over a considerable portion of their extent, so that in the early period they present a circular, punched-out appearance. After a time the ulceration tends to assume a more or less serpiginous form, healing often taking place at one side, while ulceration occurs at the other. (*See* Art. XX.)

In the matter of **diagnosis,** the chief feature in the typical tuberculous ulcer is the thin undermined skin around the edge, and in lupoid ulceration the presence of minute nodules at the margin of the ulcer and around it, often semi-transparent, and presenting the typical apple-jelly appearance. The syphilitic ulceration assumes the serpiginous form, tending to heal at one side while it progresses at the other; or it assumes a tubercular form—the so-called syphilitic lupus —progressing with much greater rapidity than ordinary lupus with much larger nodules, and destroying bones as well as the soft parts.

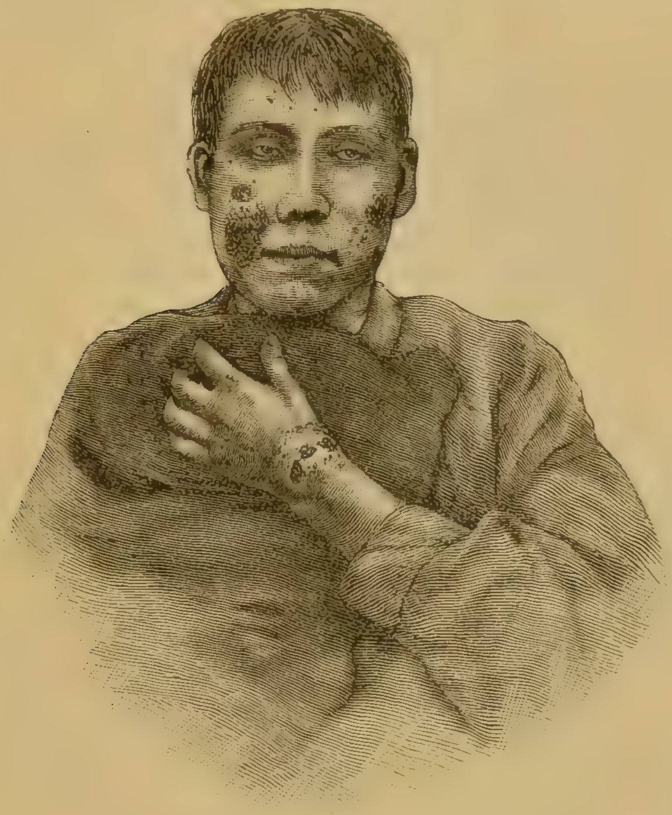

Fig. 21.—Case of Lupus

As regards the *treatment* of the tubercular form of ulcers, local measures are of the greatest importance, the essential principle being to remove the disease as completely as possible. This may be done in various ways, either by scraping, by the application of various caustics, or by excision. I need only here express my preference for excision, followed, if required, by skin grafting wherever it can be carried out.

The treatment of syphilitic ulcers will be fully discussed elsewhere, and need not occupy us now.

III. ULCERATING TUMOURS.

These belong almost entirely to the class of malignant tumours. Ulceration only occurs in connection with simple tumours as the result of pressure, there being no inherent tendency whatever to destruction of the parts around.

In the case of malignant tumours, however, especially in the carcinomata, the disease tends to invade the surrounding parts, converting them into cancerous material, and so, when it reaches the surface of the skin, destroying it, and leading to the formation of an ulcer. The ulcerations, which result from the growth of sarcomata beneath the skin, are due mainly to pressure. Thus, the character of a **sarcomatous ulcer** originating in this way is that of a fungating tumour protruding through a hole in the skin, the sides of which are inflamed but do not present the characteristic clinical appearances of sarcomatous disease. **Cancerous ulcers,** on the other hand, present an ulceration, which is generally below the level of the skin, the base of which is hard, or warty in the case of epitheliomata, and the edges of which are also hard, elevated, and sometimes warty.

Epithelioma is of interest in connection with ulcers, because it is not uncommon for a simple ulcer which has lasted many years to become the seat of the development of an epithelioma, which may either assume the ordinary rapidly-growing tuberous form, or may progress more slowly, and be of the flat variety. (*See* Art. XXII.) It is not at all uncommon for this variety of epithelioma to attack an old-standing lupus; and when it does so, the disease progresses with much greater rapidity. Indeed, it is not improbable that many of the cases described as *lupus vorax* are really cases of epithelioma grafted on a lupus.

Rodent ulcer.—The only malignant ulcer to which we need refer in this place is that known as rodent ulcer, because for a long time rodent ulcer was not recognised as a malignant disease, but was included among the peculiar forms of inflammatory ulcerations. We now know that rodent ulcer is simply a form of superficial epithelioma, and belongs to what is spoken of in Germany as the flat variety of that disease. It commences, in the first instance, quite insidiously and very often on some pre-existing lesion, such as a wart or a mole. By-and-by an ulcer is formed which has a red base, depressed below the surface of the skin without any granulations on it, and somewhat indurated; the edge of the ulcer is also hard, sharply-cut, and may in some cases be prominent, or even warty, resembling the more rapid forms of epithelioma. The margin of induration compared with that met with in the epitheliomatous ulcer is comparatively slight (Fig. 22).

The disease spreads extremely slowly, and may go on for many years, gradually destroying the parts in which it is situated, both in superficial extent and in depth. It is not uncommon, also, to find that the centre, or indeed the greater part of the sore, may become apparently cicatrised, but the cicatrix very soon breaks down again, and the ulceration proceeds.

The disease is, in the great majority of cases, only locally malignant, and it is seldom that there is any glandular enlargement in connection with it, but this does occasionally occur.

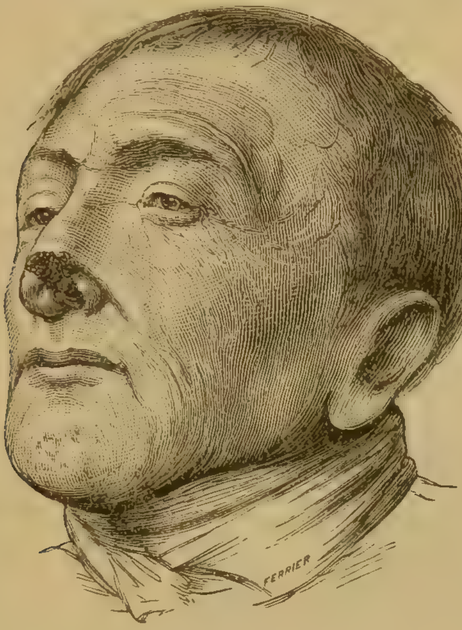

Fig. 22.—Ala of Nose destroyed by a Rodent Ulcer.

It generally attacks oldish people, chiefly men from fifty years of age and upwards.

It most usually begins in the face, more especially about the root of the nose and the region of the eyelids, and it goes on destroying the various structures which it meets with, eating into the nasal bones and the superior maxilla, or the frontal bones; and it may ultimately cause death either from hæmorrhage or, after having destroyed the bones of the skull, from septic meningitis. It may, however, be found in any part of the body, perhaps the next most frequent seat to the face being the back of the hand, just as is the case with lupus; and this may very possibly be explained by direct infection from rubbing the face with the hand.

On making *microscopical sections* of the wall or base of the ulcer, one finds a typical cancerous structure, namely, epithelial cells penetrating into the tissues in the form of tubes, and giving rise to the alveolar arrangement found in epitheliomata (Fig. 23). The epithelial cells present in rodent ulcer are smaller than the ordinary squamous epithelium, and the nuclei are spindle-shaped; and it has been supposed by some that the rodent ulcer originates in the sweat glands, and by others that it originates in the sebaceous glands. It is apparently clear that it does not originate in the surface epithelium. The amount of cancerous material present at the edge or the base of the ulcer is quite small in most cases, and may indeed be in parts entirely wanting, the fact being that the new growth ulcerates almost as fast as it forms, and hence it was that formerly, in spite of microscopical examinations, the cancerous nature of the disease was not recognised.

The *treatment* of rodent ulcer consists in free and complete removal of the disease; and where the ulcer is still limited to the skin and subcutaneous tissue, and has not penetrated into the sinuses in the various bones about the face, if the removal be sufficiently free, the disease will be cured. The cases which recur so obstinately after removal are cases where the disease has passed deeper, and where portions are left behind in some small cavity in the bone. Some surgeons prefer the employment of caustics, with the view of destroying the disease; and in cases where a bone is affected—such as the frontal bone—this is probably the best method of treatment. The caustics which are found to be most efficient are sulphuric acid, Vienna paste, or potassa fusa. In applying any of these caustics the patient should be either anæsthetised in the first instance or, if it is to be a prolonged application, kept fully under the influence of morphia, cocaine having been applied to the sore, and injected beneath it, so as to diminish the pain. In using sulphuric acid, the commercial acid is mixed up with sawdust to form a paste, which is applied, and left on until the action is complete. Probably, in the case of a rodent ulcer, from half an hour to an hour will suffice. Where potassa fusa is used the action is very much more rapid, and must be carefully watched; and as soon as it is considered that sufficient has been done, an application of vinegar will arrest the action of the caustic.

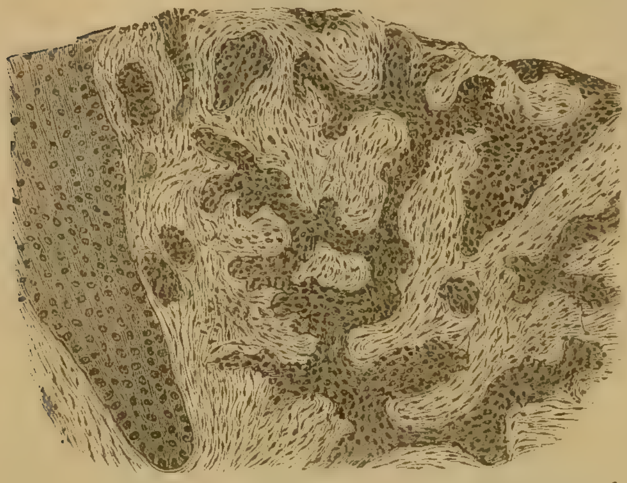

Fig. 23.—Section of a Rodent Ulcer, showing the Tubes of Epithelium spreading in the subcutaneous Tissue. The surface epithelium is also shown on the left side, and the larger size of these cells is evident.

Differential diagnosis.—The distinctions appertaining to the syphilitic, tuberculous, epitheliomatous, and rodent ulcers are placed in comparison with one another in the following table:—

TERTIARY SYPHILITIC.	TUBERCULOUS.	EPITHELIOMATOUS.	RODENT.
1. Present at any age. Previous history of syphilis.	Lupus begins in childhood or young adult life. Tuberculous ulcer secondary to abcess in glands, etc., at any age.	In advanced life. Very seldom before thirty.	Usually old age. Seldom before fifty.
2. Edges sharply cut, not thickened; often healing at one side, and spreading at the other.	In true tuberculous ulcer edges thin, undermined, purplish; no tendency to heal. In lupus soft breaking-down nodules at margin and nodules around.	Edges raised, hard, warty; no tendency to heal.	Edges hard, but not usually markedly raised; sharply cut.
3. Surface smooth; not covered with healthy granulations.	Surface soft, pale; may be prominent. Unhealthy granulations.	Surface hard and warty, sometimes sloughy; and on drying and squeezing the mass numerous small white points may be seen.	Surface hard, depressed, smooth; sometimes temporary patches of epithelium over it.
4. May be single or multiple, and there may be gummata around. No gland infection. Extends fairly rapidly.	Often enlarged glands in vicinity; not necessarily secondary to the ulceration and other tuberculous disease elsewhere. Course sluggish.	Single. Usually; neighbouring glands enlarged after three to six months. Varies in rapidity; the worst forms extending quickly.	Single usually; very slow in its progress. Glands not enlarged, or only very late.

V. GANGRENE.

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GENERAL CONSIDERATIONS.

Inflammation and gangrene.—In dealing with the final results of acute inflammation at the stage of stasis and exudation, it is mentioned that resolution might take place, that it might go on to granulation, or that it might end in gangrene (page 60). In discussing the subject of gangrene, however, we shall find that only a very few cases result in this way, and that there are many other causes which lead to the occurrence of death in a part. Nevertheless, it is convenient to discuss the whole subject here, because inflammation has to do with gangrene in a variety of ways. In the first place, as I have said, it may be the direct factor in its production as the result of extensive stasis in the blood-vessels. In the second place, inflammation may be concerned in the extension of the gangrene, more especially in the form of gangrene occurring in old people and known as senile gangrene, where the inflammation that occurs at the margin of the gangrenous part may of itself lead to further extension of the process; and in the third place inflammation is associated with gangrene, and it is by means of the inflammatory process that separation of the dead part takes place.

Definition.—By gangrene is meant death of visible portions of the tissue, and this may result from many causes. The term is generally only employed where the death of the tissue is extensive, more especially where it affects part or the whole of an extremity. Where the death of the tissue is limited to a small portion, say of the skin, the process is spoken of as *sloughing*, and the dead piece as a *slough.*

In considering the subject of gangrene, we have in the first place, and as the most important point, to discuss its mode of production and the various causes which lead to it. In the second place we have to speak of the mode in which the gangrene extends; and in the third place of the mode in which the dead part is separated.

Classification.—The various forms of gangrene may be classified either according to their clinical characters or according to their ætiology. Clinically, we speak of two forms of gangrene, namely, *moist gangrene* and *dry gangrene.*

Ætiologically, we speak of three forms of gangrene, namely, (1) *direct gangrene,* where the part which dies is the one which has been immediately acted on by the noxious agent; (2) *indirect gangrene,* where the death of the tissue takes place at some distance from the cause which leads to it; and (3) *specific forms of gangrene,* which are due to special micro-organisms.

Of these two classifications, by far the most important from a practical point of view, especially from the point of view of treatment, is the ætiological, and I shall therefore base my description on it. It will, however, save a good deal of repetition and make the matter clearer if, in the first place, I describe the clinical classification into moist and dry gangrene, and mention the chief characteristics of these two forms. The terms "dry" and "moist" express the essential clinical feature of the two types. In dry gangrene the part which dies dries up, without any marked putrefactive changes. In moist gangrene, on the other hand, death of the tissue has occurred before the evaporation of fluid has gone on to any considerable extent, and the result is that the essential features of moist gangrene are the putrefactive changes in the dead part as the result of the growth in it of various micro-organisms.

Dry gangrene.—This form is also spoken of as *senile gangrene* because it occurs more especially in old persons, seldom before forty years of age. In this form the dead part dries up and becomes hard, the fat becomes liberated from the cells and infiltrates the tissue, so that the surface of the dead part becomes greasy, while, as the result of the fat soaking into the skin, it becomes more or less transparent, and tendons and other structures may be seen through it. Dry gangrene spreads very slowly, otherwise, of course, the part would not be dry, and it is generally due to a somewhat gradual diminution in the calibre of the vessels leading to the dying part. As the ordinary putrefactive changes do not take place, the odour from the part is comparatively slight, though there is generally a distinctly musty smell. This form of gangrene usually begins in the lower extremity, especially in the toes, and may appear spontaneously after some slight scratch. It goes on very slowly. Sometimes months may elapse before a

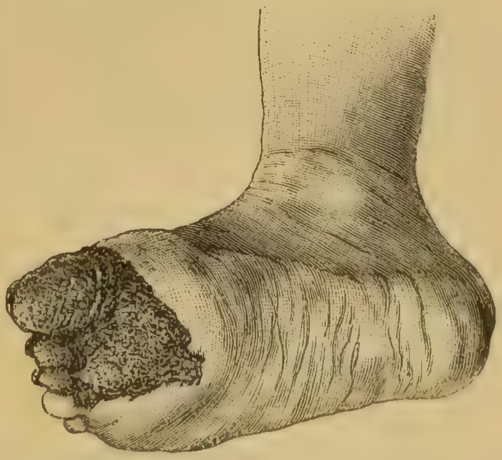

Fig. 24.—Case of Dry Gangrene (senile). The process had gone on for about eight weeks before the photograph was taken.

toe is completely dead (Fig. 24). During that time the patient complains much of pain, not only in the parts which are living, but also in the parts which are apparently dead, the cause of this being in the main that the nerves, especially the axis cylinders, are the last structures to die. This pain leads to loss of sleep and the gradual exhaustion of the patient. In the early stage the general condition of the patient is usually good, but the pain keeps him awake, so that he soon becomes pulled down, his pulse becomes feeble, and he often dies of exhaustion.

Moist gangrene, on the other hand, progresses much more rapidly, and there is not time for the liquids in the part which dies to evaporate. The consequence is that putrefaction takes place in the dead tissue, and the chief symptoms are dependent on this putrefactive process. Bullæ form early on the surface, and contain a dark fluid which has a very foul smell. By-and-by the epidermis peels off, and a moist, foul-smelling surface is left behind. The part is first reddish, and then passes through the various stages of green to black. It also becomes very much swollen, due to the presence in the tissues of gases produced as the result of this putrefactive process. On placing the hand over the gangrenous part and pressing on it, one very often gets the sensation of crepitation from the gases contained in the interstices of the tissue bubbling through the fluid, and the soft parts soon become liquid and separate from the bone as a slimy, foul-smelling, dark mass. Accompanying these local appearances, the patient suffers from absorption of the poisonous products, and has fever, sometimes very acute, and of the asthenic type, and he may die of the septic absorption or of exhaustion from the continuance of the process.

The line of separation.—In both cases when the gangrenous process has come to an end, the part which is dead becomes separated from that which is alive. This takes place as the result of inflammation occurring in the living tissues at the point where the dead and living parts join. The dead part acts as an irritant on the living tissues in the vicinity, and leads to the occurrence of inflammation, which goes on to the stage of granulation and ultimately of suppuration, so that the dead tissues, which in the first instance were directly continuous with the living, become separated from it by a layer of granulation tissue, and subsequently become quite loose from the formation in this granulation tissue of pus. Hence there is at the point of junction of the living and dead tissue an area of redness over the living tissues, which become swollen and present the various signs of acute inflammation—of varying degree according to circumstances—and very soon a furrow forms between the dead and living parts which contains pus.

In the case of dry gangrene it is not at all uncommon for this line of separation to commence at a comparatively early period, and then the tissues which were previously alive on the proximal side of the furrow begin to die, and a fresh line of separation again forms higher up. This process may be repeated several times, so that months may elapse before the final line of separation appears. The cause of this occurrence in the case of dry gangrene varies. In the first place, the inflammation in connection with the separation of the dead piece may be excessive and may lead to the death of the tissues and further spread of the gangrene. In the second place, the inflammation may not be excessive, but the vitality of the tissues may be extremely low, so that they die as the result of even a very moderate attack of inflammation. In the third place, while the line of separation is being formed, the cause which led to the original gangrene has extended, so that parts which were formerly sufficiently supplied with blood are no longer so. This is more especially the case where the gangrene has originated from thrombosis of an artery which was previously diseased, and where as time goes on the thrombosis extends, so that branches arising higher up, which in the first instance kept up the supply of blood to the parts in the immediate vicinity of the dying tissues, become closed, and then the circulation in this part is arrested. And in the fourth place, the extension of the gangrene, both in the dry and the moist forms, may be due to certain specific organisms which have entered and which complicate the process.

Treatment of gangrene.—The great principle in the local treatment in cases of gangrene is, in the first place, to prevent the occurrence of sepsis, if possible, and, in the second place, to permit and, in fact, promote evaporation of the fluids contained in the dead parts. In order to prevent the occurrence of sepsis in the part—which leads, on the one hand, to septic poisoning and general disturbance of the patient, and, on the other, to too violent an inflammation at the line of separation, and to the consequent extension of the process—steps should be taken to disinfect thoroughly the area which is about to die. This is done in the usual way, which is described fully in the article on Wounds (page 214); and in this case special care must be taken thoroughly to disinfect the folds of the nails and the parts underneath their points. When the part has been thoroughly disinfected, it should be wrapped up in antiseptic dressings, more especially in the cyanide gauze, used in the manner described elsewhere (page 219), outside which is placed a layer of salicylic wool. This dressing not only tends to prevent the occurrence of sepsis, but also permits the evaporation of the fluids in the tissues.

In both moist and dry gangrene the question of amputation must be considered from the very commencement, and the answer will depend on the cause of the gangrene, on the symptoms which are present, and on the general condition of the patient. The question of amputation or otherwise will be considered in speaking of the various forms of gangrene according to their ætiology.

As regards the general treatment, it must consist in suitable nourishment and the use of stimulants as the patient is in a weak condition, the employment of various drugs to relieve the pain, more especially the use of opium, and attention to the excretions.

We may now pass on to the consideration of the different classes of gangrene, as founded on the ætiology.

THE VARIETIES OF GANGRENE.

1. Direct gangrene.—Under this head will be described (*a*) gangrene due to crushing; (*b*) gangrene due to pressure; (*c*) gangrene due to acute inflammation; and (*d*) gangrene due to heat or cold.

(*a*) **Gangrene due to crushing.**—The most common cause of direct gangrene is a severe contusion or crush of the part, as, for example, where a limb has been run over. In such a case the parts which are directly subjected to the injury may be deprived of their vitality at once, the vessels which supply them with blood being entirely ruptured, and the whole tissue killed; in such a case death of this tissue is immediate. Injuries of this kind may, however, not only lead to direct gangrene of the part which is subjected to the injury, but may also lead to indirect gangrene of more distant parts. For example, where the wheel of a heavy cart passes over a leg it may not only destroy the tissues over which it passes, but may rupture the blood-vessels leading to the lower part of the limb, with the result that there is, in addition to the direct gangrene, death of the foot and other parts. This form of direct gangrene is moist, because the death of the part is immediate, taking place before evaporation has occurred; but the constitutional symptoms and local appearances after the death of the part will depend on whether one has been able to render it aseptic or not immediately after the injury. If one has failed to render it aseptic, or has not seen the case till some time after the injury, there supervene the typical appearances of moist gangrene already described. The part becomes black, foul-smelling, with bullæ on the surface and peeling of the epidermis, while, at the same time, the patient is suffering from constitutional disturbance, due to the absorption of the poisons which are being formed there. If, on the other hand, the case be seen at once, and efficient measures be taken to render it aseptic, the typical local and constitutional signs of moist gangrene do not occur. Although a considerable portion of the tissues may have died, no putrefaction takes place in them, and consequently we have no bullæ, no foul smell, no liquefaction of the dead part, no crepitation, no inflammation around, and no constitutional symptoms. The dead part remains as a non-irritating body, and does not give rise to the formation of a line of demarcation around. It becomes mixed up with the blood clot which is effused from the torn vessels, and by-and-by infiltrated with cells, which gradually destroy it, take its place, and develop into fresh tissue. In fact, the progress of the wound is the same as that which is described under healing by blood clot (page 206). It may be that after a time a small portion of the dead tissue comes away; but there is, as I have said, no great sloughing, no extension of the gangrene, no inflammation around, and no constitutional symptoms.

Thus, as regards the *treatment* of these cases, it is evident that the first and by far the most important thing to do is to render the injured part and the skin around aseptic, in the manner which is described in the article on Wounds (page 214). The best dressings to employ are the cyanide gauze dressings. It is well here, as in all cases of gangrene, to avoid the use of ointments, so as to allow the evaporation of the fluid and the drying up of the slough.

In cases where a very large portion of skin has been destroyed as the result of the injury—as, for example, in the case of severe lacerated wounds, where the skin may be torn off from the greater part of an extremity—the question of immediate amputation will have to be considered, and the old rule was that in these extensive cases amputation was the best practice. Since the introduction of skin grafting, however, this rule is not so imperative, and many cases which would formerly have been amputated may now be treated conservatively with satisfactory results. The chief reason for recommending amputation in former days was the great extent of the sore which was left, and, apart from the septic risks, the difficulty in getting healing of this sore; further, if healing occurred the great contraction which followed often rendered the parts beneath absolutely useless, both from contraction of the joints and from constriction of the limb, leading to great œdema and deformity of the parts below. With skin grafting, however, employed as soon as possible, these difficulties, which essentially depend on the contraction of the granulations, may be to a very great extent avoided.

In cases where the injury has been such as to destroy the main vessels or nerves leading to the distant parts, or to fracture the bones badly, the question of primary amputation will have to be considered; and formerly most cases of this kind were regarded as demanding immediate removal of the limb. This rule also has been much altered lately, on account of the possibility of avoiding decomposition and septic risks, and the rule which we may now follow is that we do not amputate at once unless we are certain that the parts beneath must die as the result of the absence of the circulating blood, or that they must become absolutely useless from the loss of the nervous supply. In cases where these points are doubtful, or where, although the local injury is very extensive, yet the blood and nervous supply to the parts below is satisfactory, one should delay amputation, and after thoroughly disinfecting the region which has been injured, and placing the distal parts in the best condition for recovery, by keeping them warm and elevating them slightly, one may wait and see whether they are likely to recover and be of use to the patient. If that be not the case—and it will become evident in twenty-four or forty-eight hours after the injury—then amputation may be done, and there is this further advantage in delaying the amputation, that the patient has time to recover from the shock of the accident, for it not uncommonly happens that when primary amputation is performed, the shock of the operation superadded to the shock of the injury, leads to the death of the patient. Of course, if the attempts at rendering the injured parts aseptic be not successful, then we are brought face to face with the various septic dangers in addition to the dangers resulting from the injuries of vessels and nerves passing to the parts beyond, and we have to fall back on the old rules on this matter ; but even then the sepsis is usually not declared till twenty-four or forty-eight hours have elapsed, during which time the patient has recovered from the primary shock. One must also, in considering this question of amputation, take into account the general condition of the patient; his age; the advisability of subjecting him to the long treatment in bed which may be necessary where we have an extensive wound and where we would otherwise decide to attempt to save the limb; and the condition of the kidneys, especially as to whether the patient is the subject of albuminuria, or of diabetes. In the latter instance, the cases in which attempts may be made to save the limb are fewer than where this condition is not present.

(*b*) **Gangrene due to pressure.**—Another cause of direct gangrene is continued pressure on a part, and this is a very important point to remember when one has to place the patient in the same position for a long time. In such cases the parts which are subjected to pressure are very apt to die, more especially the skin over the sacrum, and, indeed, over any prominent projections of bone which may press either on the bed or on the splint; in other words, we have to do with the condition known as *bed-sore*. One must bear in mind the chance of bed-sores in these cases, and be on the outlook for them, and if possible anticipate their occurrence by proper treatment.

As to the various means of avoiding the occurrence of bed-sores, the first thing is to vary the pressure so that it shall not always tell on the same point, and this may be done by altering the position of the patient or of the part. In addition to varying the pressure, it should be distributed. Where bed-sores over the sacrum are feared the patient should be placed on a water-pillow, so as to distribute the pressure equally over a large surface. In using a water-pillow, great care must be taken that a proper quantity of water is introduced into it. If too much water be present the pillow becomes hard and causes as much pressure as if it were not employed, whereas, on the other hand, if too little water be introduced, the patient is not properly floated up, and the part comes in contact with the bed; hence the rule is to put in just sufficient water to keep the patient floating. Another great point in the avoidance of bed-sore is to keep the part which is subjected to pressure thoroughly dry. The patient should be turned over once or twice a day; the region of the sacrum should be carefully washed and thoroughly dried, and then it is well to sponge the part with some stimulating fluid, that generally employed being spirits of wine. This is allowed to dry on the skin, which is then powdered, either with starch or other powder. The best, I believe, is powdered boracic acid, which is quite soft, and being to a certain extent antiseptic, prevents the decomposition of any fluids which may accumulate there, and, as a matter of fact, it is the decomposition of the fluid rather than its mere presence which is of serious import as a factor in the production of bed-sore. Then, of course, the nurse must be careful that the sheet under the patient is absolutely smooth, that there are no wrinkles or turns of bandage passing over the part which is subjected to pressure. Some prefer, instead of the alcohol and powder, a stimulating ointment, more especially an ointment consisting of equal parts of balsam of Peru and resin ointment, which is both stimulating and antiseptic; but I believe what I have already described is the best method, more especially as it enables one to see the condition of the part at any time.

When a bed-sore has once formed, the reduction or avoidance of the pressure over the part, and the other measures just described, should be most carefully seen to, and every effort should be made to keep the surface as aseptic as possible. From the situation of these bed-sores there is but little chance of converting the slough into a dry one, and, therefore, I should in this instance depart from the general rule which I have laid down as to applications to parts which are becoming gangrenous, namely, to avoid oily substances, and employ as a dressing either balsam of Peru or, what is perhaps more convenient in many ways, eucalyptus ointment. Where a bed-sore has once formed, it is perhaps best to use a circular water- or air-pillow in preference to a square water-pillow, because, by means of the circular pillow, pressure over the bed-sore is absolutely avoided.

(*c*) **Gangrene due to acute inflammation.**—Another cause of direct gangrene is acute inflammation. There are certain cases where the result of an acute inflammation is the death of the part which is the seat of the inflammation, apart from the killing of the tissue by the poisons of the micro-organisms which are producing it. This is a distinction that must be carefully made. In certain cases organisms growing in the tissues directly kill the tissue in which they are growing, essentially by virtue of their poisonous products, and this is the way in which certain organisms produce the specific infective forms of gangrene. In other cases, however, while the organisms produce an acute inflammation, they do not directly destroy the vitality of the tissues; but the latter die as the result of the acuteness of the inflammatory changes, more especially as the result of the extensive occurrence of stasis, or of excessive exudation pressing on the vessels from outside and preventing the circulation through them. This form of gangrene, resulting from arrest of the circulation in consequence of a violent inflammation, occurs especially when dense tissues are attacked, and the best examples of it are boils and carbuncles in the skin and death of portions of bone where the bone and periosteum have become acutely inflamed. In the latter case the type of the inflammation is a very acute one, and goes on rapidly to the formation of pus, and is spoken of as acute suppurative periostitis or osteomyelitis. In these circumstances, the exudation from the vessels, both of fluid and of cells, is very marked, and if the vessels be contained in rigid canals in the bone, the exuded material fills up the canals and compresses the vessels that are running through them. Hence, as the result of acute suppurative periostitis or osteomyelitis, the vessels of the bone over a very considerable area are compressed, while at the same time the periosteum is detached from the surface of the bone by the exudation, and the vessels passing to it from the periosteum are torn across or blocked. Thus a large area of bone may become entirely deprived of circulation and die. The condition of acute necrosis is described in its proper place. (Art. XXX.)

In the skin, also, acute inflammation affecting the dermis, especially when commencing in connection with the hair follicles, is very apt to lead to death of portions of the skin for the same reasons as in the case of bone, and we have the condition known as *boil*, where there is an acute inflammation of the dermis, and death of the portion forming the core of the boil ; or as in the case of *carbuncle*, where this inflammation is much more extensive, and where we have either one large slough of the dermis or a number of small ones, in fact, a congeries of boils. That the formation of the slough in boil and carbuncle is essentially due to the density of the tissue in which the inflammation is occurring, and not to destruction of it by poisons secreted by the micro-organisms, is evidenced by the fact that in young children, where the skin is quite thin, inflammation starting in the same parts, which in adults would lead to the formation of a boil, causes in them small abscesses without any sloughing of the tissue. Boils and carbuncles are discussed in Art. XXVIII., in connection with Diseases of the Skin.

(*d*) **Gangrene due to heat or cold.**—A fourth great cause of direct gangrene is the effect of heat and cold. Burns and scalds are dealt with in a separate section (Art. XII.), and I need therefore only refer to the effects of *cold* in the production of gangrene. Where cold produces gangrene, the parts affected are generally those most distant from the heart, more especially the toes (the great toe in particular), the fingers, the nose, and the ears ; and in addition the people generally attacked are those who are in a weakly condition, either aged people, or infants, or drunkards. In the causation of gangrene as the result of cold, wet cold is worse than dry cold, and in fact a great degree of dry cold can be well borne provided there be no wind, but if there be wind playing on the part, even although the temperature is only a little below freezing, we may have all the troubles characteristic of frost-bite.

The first effect of cold is to cause contraction of the blood-vessels and slowing of the circulation, so that we have the well-known blueness of the parts which are exposed to cold. This contraction of the blood-vessels may go on to their complete obliteration, and the part become white; and if the cold have been continued for any considerable length of time, and the patient be then exposed to warmth, the vessels become greatly dilated, and stasis and thrombosis occur both in the parts affected, and sometimes also in the internal organs. Apart from the effects on the vessels, the vitality of the tissues themselves is very much diminished, and they are thus still more predisposed to the occurrence of gangrene.

Various other changes have been described as the result of the action of cold, which may also have to do with some of its after effects. For example, it is stated that rupture of the vasa vasorum occurs, leading to imperfect nutrition of the blood-vessels and interstitial hæmorrhage. Changes have also been described in the nerves in the form of neuritis of an ascending character, and of degeneration of the nerve tubules, and these changes may to some extent explain the atrophy of the muscles, and, which is a striking after effect of exposure to cold, the formation of trophic ulcers, etc.

As to the symptoms of excessive cold, we have both general and local effects. The general symptoms when a patient is exposed to cold air are, in the first place, increase in the rapidity of the circulation, increase in the pulse-beat, and a slight rise of temperature; but as the exposure to cold continues, the temperature falls, an irresistible drowsiness comes on, the gait becomes staggering, respiration difficult, pulse slow, the patient lies down and soon passes into a state of coma and dies.

As regards the local effects of cold, there are three degrees of frost-bite, the first two being the same as the first two degrees of burns; and the third including the other forms or stages of burns. (*See* page 252.)

In the *first degree* the part after exposure to cold becomes of a bright or dark red wine colour. This redness can be pressed away with the finger and is slow in returning, in fact the circulation through the part is much slower, and in addition the vessels of the part are dilated. The skin in this region swells and becomes œdematous, and if exposed to heat there is intolerable itching. This effect will pass off in a few days if there be no further exposure to cold, but if it be repeated, the condition becomes chronic and we have the production of chilblains, which will be described in the article on Diseases of the Skin.

In the *second degree* of frost-bite we have, in addition to this condition of erythema, the formation of blisters upon the surface of the skin, which contain bloody fluid and which are frequently followed by rapid ulceration, and thus as a second result of cold we may have an acute spreading ulcer which is atonic and very slow in healing. In other cases, where the affection is more chronic, we have the condition of ulcerating chilblains, where the swollen œdematous skin cracks and forms shallow fissures covered with crusts. These fissures go on enlarging, forming obstinate ulcers, especially in strumous children and in those who wear tight boots.

The *third degree* of frost-bite is marked by the death of various portions of the tissue, more especially of the skin and subcutaneous tissue. The skin becomes livid and marbled, large bullæ containing rusty-coloured serum form on the surface, and sloughs appear, which are dry, white, or black. This stage of cold is most usually the result of sudden exposure of the cold part to warmth, which leads to thrombosis of the vessels, or at any rate to violent inflammation of the part and death of the whole limb. In this condition there may be no general disturbance at all, but in some cases albuminuria appears, and there is a general septic infection.

Before considering the *treatment* of frost-bite, we must in the first place refer to the measures necessary to avoid its occurrence. Of these the most important is the wearing of warm clothing, particular attention being paid to the exposed parts of the body, so that they shall remain warm, and free from pressure. The diet should contain a large quantity of fat, and alcohol should be avoided as much as possible. Further, the parts should be kept thoroughly dry, for, as I have just said, wet cold is worse than dry cold, and any one who walks about with cold and wet feet is very apt to suffer from chilblains.

Where a patient has been exposed to cold, one must attempt to avoid the effects of frost-bite, and more particularly one should be very careful not to apply heat too quickly. If the extremity has been exposed to cold, the most that ought to be done in the first instance is to rub the parts with snow or cold water, and, above all, not to bring the patient into a warm room, otherwise inflammation is sure to occur, and very possibly also extensive thrombosis of the blood-vessels. The patient should be kept in a cold room, and as he recovers dry friction should be employed, and at the same time he should have cold drinks with very little alcohol. As he gets a little warmer the drinks may be warm, and he may then be brought into a room of moderate temperature.

The chief characteristic of gangrene resulting from cold is the slowness with which it spreads. Its tendency is to become a dry gangrene, and the line of separation being slow in forming, it is, as in senile gangrene, very apt to prove abortive, and the gangrene spreads beyond it, owing to the gradual obliteration of the vessels above. In the case of gangrene after cold the main question to be considered is that of amputation, and the usual rule is not to amputate in these cases, but to wait for a line of demarcation, because we cannot tell how far the gangrene will extend, and some, indeed, not only wait for a line of demarcation, but even leave the part to drop off. While I quite agree that a line of demarcation should be waited for, on account of the difficulty that we have in knowing how much of the tissue is going to die, I should say that once we have a clear indication of the line of demarcation, and once it is evident that we have not to do with a superficial slough of the skin, amputation should be performed as soon as possible at a little distance above that line. The rules, in fact, must be modified under the influence of asepsis, very much in the same manner as the rules with regard to the treatment of senile gangrene.

2. **Indirect gangrene.**—The forms of gangrene which are due to indirect causes are several, and for the sake of clearness we may subdivide them into four groups :—(*a*) Gangrene due to gradual diminution in the calibre of the blood-vessels, (*b*) gangrene due to some sudden obstruction of the blood-vessels, (*c*) gangrene due to imperfect innervation, and (*d*) gangrene brought about by certain general causes, such as diabetes, acute fevers, the use of ergot, etc.

(*a*) **Gangrene due to gradual diminution in the calibre of the blood-vessels.**—The typical form of gangrene due to this cause is that known as *senile gangrene.* This form rarely occurs before the age of forty, and is most usual in persons of sixty and upwards. The patients are men much more often than women; in fact, the proportion is something like twenty men to one woman, and this is probably due to the fact that atheromatous disease of the arteries is much more common in men than in women. Senile gangrene is in the first instance unilateral, and more especially attacks the feet and the toes, and only very rarely the fingers or the ears. The causes of senile gangrene are various diseases which produce gradual diminution in the calibre of the blood-vessels leading to the part, more especially conditions which lead to endarteritis, for example, alcoholism. A patient who is always drinking is very apt to suffer from chronic endarteritis, because the alcohol circulating through the blood irritates the lining membrane of the vessels, and is said in the first instance to cause endarteritis and blocking of the vasa vasorum, and then later to lead to atheroma in the main arteries, the thickening of the wall of the vessel in the case of alcoholism being usually uniform all round.

A second cause leading to the production of senile gangrene is syphilis, which leads especially to endarteritis of the smaller vessels; in fact, this is one of the chief occurrences even in the very earliest stage of syphilis.

A third great cause of senile gangrene is atheroma of the artery, which is, in fact, a chronic inflammation of the deeper parts of the internal coat, and which is again due to syphilis and to a variety of causes that seem to arise in old age. In the typical atheromatous vessels the thickening of the lining coat is irregular, and very often affects only one side or part of the side; and in connection with the atheromatous change in the smaller vessels not only is the calibre of the vessel diminished by the projection into it of the thickened patches of atheromatous material, but the disease is apt to extend to the muscular coat, leading to calcification of this coat, and thus to permanent rigidity of the vessel. Hence the smaller arteries are converted into calcareous tubes, which cannot dilate or contract in accordance with the needs of the tissues below, and the calibre of which is very much diminished from the irregular thickening of the internal coat; and a further effect of this condition of atheroma or of endarteritis is that coagulation of the blood is apt to occur in these vessels, and thus complete blocking of the vessel may result, and once this has taken place the thrombosis may extend up the vessel, and progressively cut off the supply of blood to distal parts.

This condition of the vessels leads to certain symptoms which precede the appearance of gangrene, and which are essentially due to imperfect supply of blood to the part beneath. Thus, one of the chief complaints of the patient, even long before the senile gangrene occurs, is the great coldness of his feet, which is not relieved by any application of warmth. He also complains of perverted sensations in his feet, due to imperfect supply of blood to the nerves. Thus he suffers much from tingling in the feet, and does not feel the ground properly when he walks, and feels in fact as if there were something soft between his feet and the ground.

When a patient comes complaining of these perverted sensations and coldness, examination of the blood-vessels will almost always show that they are rigid tubes instead of being compressible, and that there is little or no pulsation in them. Where these symptoms are present, one must be on one's guard against the occurrence of senile gangrene, and must take measures to prevent, or at any rate to delay, its occurrence as much as possible. In the first place, the patient may try to keep the feet somewhat warmer, but great care must be taken not to apply too much heat. For instance, if the feet were placed in a bath at a temperature of 100° or more, in the normal condition the blood-vessels would dilate, and a large quantity of blood be carried to the part; but in this condition the vessels cannot do so, and the only effect of such excessive warmth will be to weaken the vitality of the tissues. Hence the temperature of the bath should not be higher than 90° to 94°, and in addition to this friction of the limb must be employed. Then the patient must be especially cautioned against any injury to his feet. He must not wear tight boots, and he must avoid any scratches or other injury; for example, he must take great care in paring his nails not to wound the skin, and he must be specially careful if he has corns.

The further history of the case (where these preliminary symptoms have appeared) is that the patient gets a wound on his foot or toe, or a blister from too tight boots, or from walking too much, and this becomes inflamed, and the inflamed part dies. Once the gangrene has started it goes on very slowly, and it may be months before more than the toes die, and in the process of death the tissues dry up and we have the typical appearances previously described as characteristic of dry gangrene. (*See* Fig. 24.) The patient for some time remains in a very good state of health, and his great complaint is the pain which he suffers, and which may be intense. This pain is partly due to imperfect supply of blood to the nerves, partly to neuritis, and partly also to the fact that the axis cylinder of the nerves is one of the last structures to die, and consequently in a part which is already dead the living axis cylinder may still be present and give rise to this intense pain. As the result of this pain the patient becomes sleepless, and after a time his pulse becomes feeble, and he is very restless. If the disease be allowed to run its course, the patient may ultimately die, being worn out by the pain and want of sleep, or some septic disease may develop at the line of demarcation. As I said before, in speaking of dry gangrene, the line of demarcation forms very slowly, and the death of the tissue is very apt to recommence as the result of the various causes which have already been enumerated. Ultimately, however, if the patient live, the process will come to an end either after the death of a few toes or of a portion of the foot, and the dead parts may gradually drop off, leaving the bones and other tissues exposed. In some cases when a line of demarcation forms, the inflammation may be pretty acute, and there may be a good deal of exudation into the part, leading to the occurrence of moist gangrene.

Once it is evident that gangrene is about to occur, the first essential in the *treatment* is to disinfect the parts thoroughly, special care being taken in the disinfection of the nails and the parts surrounding them, and then, having thoroughly disinfected them, they should be wrapped up in antiseptic gauze, with a thick layer of salicylic wool outside, and the limb should be placed in a slightly-elevated position. This dressing permits evaporation of the fluid from the tissues, which is a point of great importance, because one wishes particularly to avoid the occurrence of moist gangrene, more especially if one be not sure of the asepticity of the tissues. Some surgeons apply ointments of various kinds, or carbolic oil or other greasy dressings; but as I have already pointed out, the use of such dressings is not advisable, because they interfere with the drying up of the tissues.

In addition to these local measures, the patient's strength must of course be supported by a generous diet and fresh air, and above all steps should be taken to relieve his pain, more especially by the free administration of opium. Indeed, apart from the relief gained, opium in large doses seems to have a very considerable action in arresting the progress of the disease in certain cases.

At an early period also the question of amputation must be carefully considered. The old rule with regard to this point in cases of senile gangrene was that amputation should never be performed, but that the part should be allowed to drop off, the utmost that should be done by the surgeon being to snip through dead tendons or dead pieces of bone, and on no account to interfere with the living tissues. The reason for this was that before the antiseptic period acute inflammation almost always—to some extent, at any rate—followed amputation; and if inflammation occurs in these weak tissues, it is very likely indeed to lead to sloughing of the flaps and more rapid progress of the gangrene, which then in reality becomes a moist gangrene. At the present time, however, we can avoid this inflammatory disturbance, and therefore the rules as regards amputation in senile gangrene are completely altered. In fact, in my opinion, one should in the great majority of cases of senile gangrene recommend early amputation. By operating at an early period the patient's strength is not worn out by continued pain and loss of sleep, and he is in a very much better condition to survive the operation than if it be delayed to a later period. The only difficulty is that one does not know how far the process will extend; but, as a rule, this can be judged of by ascertaining the point at which the pulsation in the main vessels ceases, and if one cannot ascertain this, then the best place for amputation in most cases is the knee joint. Indeed, even if one found that pulsation was going on lower down, it is a question, which must be decided in the individual cases, whether one should not still amputate at the knee joint.

(*b*) **Gangrene due to some sudden obstruction of the blood-vessels.**—This may be the result of some pressure outside the blood-vessels, of rupture of the blood-vessels, or of some blocking in their interior. Examples of gangrene due to pressure outside the blood-vessels are furnished by the application of *tight bandages*, where the parts below subsequently swell; for instance, where a fracture of the fore-arm is put up with the arm in the extended position, a turn being taken above the elbow to keep the splint from slipping off, and where the arm is then bent at right angles so as to place it in a sling. In such circumstances this turn above the elbow will become too tight, and, as I have seen in one case, will lead to gangrene of the whole of the fore-arm.

Another example is furnished after *fracture*, where, if the bones be not brought into proper position, one of the broken ends may press on the blood-vessels, and prevent the flow of blood to the parts below; or, again, in connection with the fracture there may be rupture of the blood-vessels, diminishing the supply of blood to the extremity, and, in addition, the effused blood collecting in the tissues, presses on the other vessels, and so completes the obstruction. Again, we have the effect of a *ligature* applied around the vessels, in parts of their course where there is but little collateral circulation. This is chiefly seen after ligature of the third part of the axillary artery, of the brachial artery, or of the external iliac. This is, however, a rare cause of gangrene, because even in the examples mentioned there is usually sufficient collateral circulation. It is generally held to be particularly dangerous if, at the same time that the main artery is tied, the circulation through the vein be interfered with either by pressure or by ligature; but I must confess that I do not feel confident as to the danger of this occurrence. I have removed both the axillary artery and vein in operating for cancer in the axilla without gangrene following in the arm, although the axillary artery is one of those in which the collateral circulation is most inefficient.

Of the causes in the interior of the artery, which lead to gangrene, the essential one is the occurrence of *embolism*, which is, of course, followed by thrombosis, and which may lead to blocking of the artery over a very considerable area.

Where the obstruction is arterial alone in the first instance—for example, after ligature of an artery—the first thing that is noticed is that the limb below becomes white from the absence of blood. By-and-by it assumes a dark livid colour, and then the various changes already described as characteristic of moist gangrene follow. If the cause be an embolism of the artery, we have, in addition to the sudden whiteness of the limb, violent pain at the point where the embolus has been arrested, and this is a very valuable sign as showing us where the block has occurred. On the other hand, if there has been venous as well as arterial obstruction at the commencement, the part below becomes œdematous and dark.

The *treatment* of this form of indirect gangrene depends, in the first instance, on whether the block is purely arterial or purely venous, or both. If it be purely arterial, one must wait for twenty-four or forty-eight hours, in order to see whether and how much collateral circulation may be established, because, as a rule, the greater part, and even the whole of the parts below may recover. Once one is sure how much is going to die, and this will be evident in twenty-four to forty-eight hours, one should then amputate, because there is no necessity for waiting for a line of demarcation. As to where to amputate, that must depend on the anatomy of the arteries, because it is not necessary in all cases to amputate as high up as the seat of obstruction. For example, after ligature of the superficial femoral artery we know that the profunda will supply the tissues quite satisfactorily down to well below the knee, and in such a case it is not necessary to amputate higher than through the condyles of the femur. It is generally laid down that in a case where the gangrene is dependent on venous blocking, or on blocking of both artery and vein, one cannot expect any recovery, but should amputate at once.

(*c*) **Gangrene due to imperfect innervation.**—Where a limb is *paralysed*, one constantly sees that the nutrition is deficient —for example, in infantile paralysis—and such limbs are especially liable to the formation of bed-sores and gangrene from pressure. It is often assumed that this tendency is due to the fact that the patient, not feeling properly with the limb, rests longer on the part than he would if it were not paralysed; but this is not altogether the case, for in hemiplegia, where the patient is lying absolutely still, he is extremely liable to suffer from bed-sores, which are worse on the side that is paralysed than on the other. Again, if extension be applied to a paralysed limb and to a sound limb, one is much more liable to get sloughs under the extension plaster on the paralysed side than on the other. The gangrene that occurs in these cases of paralysed limbs generally comes on very quickly, is moist, and is often spoken of as an *acute bed-sore;* hence it is very important to remember that pressure is more liable to cause gangrene in parts which are paralysed, and that in such circumstances pressure should be avoided as far as possible. The limb should be kept warm, and if sloughing does occur, it should be treated like a bed-sore.

There is another form of gangrene in connection with nervous derangement which it is of importance to consider very shortly, namely, what is termed "symmetrical gangrene," or *Raynaud's disease.* The term "symmetrical gangrene" at once brings out one great point, namely, that it occurs on both sides at the same time, thus differing from senile gangrene, which in the first instance is unilateral. A further difference from senile gangrene is that it more often affects the fingers than the toes, and again, that it is much more limited both in extent (generally not reaching beyond the phalanges) and in depth (seldom going deeper than the skin); and further, in Raynaud's disease the blood-vessels are normal, whereas in senile gangrene, as has been already pointed out, they are thickened and hard.

This affection is not a serious one, and chiefly occurs in women between eighteen and thirty years of age who are highly hysterical, and it is most probably associated with uterine or menstrual troubles. It is often developed by cold, and hence is most commonly met with during the winter months. It is supposed to be due to spasm of the arterioles reflexly from the uterus.

The phenomena of Raynaud's disease must be divided in the first place into those which precede the gangrene, and which may or may not be followed by gangrene; and in the second place the gangrene itself. The phenomena which precede the gangrene are of two kinds: the one set is spoken of as *local syncope*, where the arteries become contracted and the fingers become white. As the result very often of a sudden mental shock, the patient's fingers become quite white and lose their sensibility, so that one might push a needle into them without causing pain or leading to bleeding. They are very cold, and generally one or two fingers are contracted, the condition being more or less completely symmetrical. This attack lasts a few minutes to a few hours, and then may pass off, the attacks recurring generally two or three times in the course of the year. When the condition passes off, the fingers become red owing to dilatation of the blood-vessels. In some cases, during this stage, the fingers, instead of being dead white, are slightly livid.

The second form is that which is spoken of as *local asphyxia*, in which the circulation of the blood is not completely arrested, but goes on extremely slowly, so that the blood in the part is practically entirely venous blood; hence the fingers become livid and look as if they had been dipped in ink. This condition is accompanied by some swelling of the fingers, with shooting or burning pain. It may last for a day or two, and may or may not lead to gangrene.

If it persist, we have the final stage of Raynaud's disease, namely, the *gangrene.* The darkness of the fingers continues for some days, the pain and other symptoms increase, and there may probably be the formation of small bullæ on the fingers; indeed, one might think that the patient was going to lose all his fingers.

Ultimately, however, the circulation improves very much, and, as a rule, as a final result, only a very small dry slough separates. The whole process is very slow, and takes from the commencement of the gangrene to the separation of the slough from twenty days to ten months—on an average, three or four months. This disease is very apt to recur, more especially during the winter season.

As to *treatment*, the first essential is to remedy any uterine trouble which may be present, and to improve the general condition of the patient's health. As regards local treatment, chief dependence is placed on the employment of warmth, keeping the fingers in warm baths for some hours, and afterwards wrapping them up in warm cotton wool. Some say that they also obtain benefit from placing the hand in electric baths. If sloughing be likely to take place, then the treatment must be carried out on the ordinary lines, and antiseptic dressings must be employed. In no case is amputation necessary.

(*d*) **Gangrene due to certain general causes.**—Of these, I need only mention three, namely, gangrene in connection with *diabetes*, gangrene after *acute fevers*, and gangrene following the use of *ergot*, and of these, gangrene in connection with diabetes is much the most important.

There are two modes in which *diabetes* may be related to gangrene: (1) we may have the death of the part directly dependent on the presence of diabetes, or (2) we may have gangrene from some other cause taking place in a patient who is diabetic. The term diabetic gangrene is limited to the gangrene which is more or less directly dependent on diabetes. In cases where gangrene occurs in diabetic subjects, the course of the disease is generally very markedly modified in that it spreads with greater rapidity, there is more inflammation around the gangrenous part, and the gangrene, though often dry in the first instance, tends to become moist.

As to the pathology of gangrene dependent on diabetes, it is found that in these circumstances the vessels have become thickened, owing to the occurrence in them of endarteritis; and this is explained by the fact of the sugar or its precedent chemical substance circulating in the blood and causing irritation of the lining membrane of the vessels. The second effect of diabetes in the production of gangrene is that the tissues of the diabetic individual are weak and less able to resist injury than healthy tissues, and are more especially less able to resist the entrance of pyogenic organisms, which appear to grow with special rapidity and virulence in the tissues of such persons. A third point which is stated by some is that in diabetics the innervation of the part is interfered with, as the result of central nervous disturbance.

The characteristic of the diabetic gangrene is that it is more acute in its progress than other non-specific forms of gangrene not due to immediate obstruction of the circulation, and that it is accompanied by an excessive amount of inflammation, and results in extensive sloughing of the skin. It is practically always a moist gangrene, and the characteristics are those already given, with, in addition, the greater inflammation about the line of separation and the more acute spread of the disease, to which I have just referred. The general condition of the patient is bad. He is generally in an asthenic condition; and if left to itself the disease almost always terminates fatally, either from septic complications, from diabetic coma, or from exhaustion.

As regards the *treatment*, one must especially warn diabetics to be very careful to avoid any injury, lest an acute inflammation, and very probably a gangrenous inflammation, should result. More especially they should avoid injuries to the feet, the wearing of tight boots, etc. Next, the general condition should be attended to, and they should be placed on anti-diabetic diet, that is to say, should avoid substances which will tend to the production of sugar, sugar itself, and all starchy foods, potatoes, etc.; they should live generally on animal food, eggs, fish, milk in moderation, and very small quantities of greens. As to bread, they should either have stale bread or toasted bread, or, still better, the special biscuits and loaves which are made for diabetic patients, the most palatable of which are the almond biscuits. Further, if a diabetic patient *does* sustain any injury, one must be particularly careful in the antiseptic precautions, so as to avoid the entrance of micro-organisms. Once the gangrene has occurred, opium should be employed in large quantities, or still better than opium is codeine, which is said to diminish the irritability of the afferent nerves of the liver. Codeine is given in the first instance in quarter-grain doses three times a day, and this may be increased up to five grains.

Once the gangrene has occurred, one must at an early period consider the question of amputation. Formerly the rule was not to amputate in cases of diabetic gangrene on account of the great tendency to inflammation and suppuration in the stump, to the extension of the gangrene, or to death from diabetic coma. Here again, however, the rules have been completely altered by the employment of antiseptic measures, and from recent work it seems quite evident that the best treatment in the great majority of cases is as early amputation as possible. By strict asepsis, the inflammation in the stump is avoided, septic troubles are prevented, and gangrene does not occur if the amputation has been performed sufficiently high up; and as regards the question of diabetic coma, the patient is really not more liable to its occurrence after amputation than he is during the course of the diabetic gangrene.

As to gangrene after *acute fevers*, this especially occurs after typhus and typhoid fever, and here the gangrene attacks the extremities and the parts farthest from the heart, namely, the toes, nose, ears, and sometimes the fingers. This form of gangrene is supposed also to be due to the occurrence of endarteritis and thrombosis. In some cases, however, it is due to embolism. The disease generally commences during the period of convalescence. The type of gangrene is of the dry variety, it is unilateral, and the treatment should be in the first instance to disinfect the parts and wait for a time before amputating, on account of the weak condition of the patient.

Thirdly, as a general cause of gangrene we have the use of *ergot*. This form of gangrene sometimes occurs in epidemics, in certain regions, or in certain families, and this is due to extensive infection of the rye in the district with the *claviceps purpurea*, and to the patients eating large quantities of this infected rye. The early effect of ergot is to produce a tetanic contraction of the smaller blood-vessels, and if this condition be kept up for a long time, it may end in gangrene, more especially of the extremities. Preceding the occurrence of gangrene, the patient suffers from diarrhœa, buzzing in the ears, hyperæsthesia, cramps, coldness of the extremities, diminished arterial pulsation, etc., and this condition may or may not go on to gangrene. If the condition be recognised, the use of the infected rye bread should be stopped, large quantities of coffee should be given, and friction applied to the affected extremities.

Where gangrene occurs, it usually attacks men between thirty and forty years of age much more often than women, the supposed reason being that men drink more than women, and are consequently liable to have a certain amount of endarteritis and atheroma, which, in combination with the spasm of the arterioles, is apt to end in gangrene. This form of gangrene is dry, but in certain cases it may be moist; and as regards its extent, it may vary from the loss of a nail to the loss of a limb. As regards the treatment, one must wait for the line of demarcation, because it is impossible to decide how much of the tissue will die.

3. Specific forms of gangrene.—In former days a variety of gangrenous processes attacked wounds, as the result of sepsis occurring in them. These are seldom or never seen now. It will probably be sufficient if we speak of three forms of specific gangrene, namely, (*a*) acute traumatic gangrene, (*b*) phagedæna, and (*c*) cancrum oris or noma.

(*a*) **Acute traumatic gangrene.**—This is a form which attacks wounds, and is due no doubt to the growth of bacteria in the tissues. The wounds which are more especially attacked are those which have been soiled with earth or dung, and the disease begins very soon after the accident, usually about the second or third day, and before granulation has occurred. The organisms that are at work in producing this form of gangrene, have not yet been isolated, no doubt on account of the great rarity of this trouble at the present time; but judging from the experimental evidence based on the study of organisms, producing somewhat similar processes in the lower animals, there seems little reason to doubt that they must belong to the class of bacilli and also to the class of anaërobes. These organisms spread into the tissues with very great rapidity, and cause their death, no doubt in the main as the result of the poisons which they produce, possibly also to some extent as the result of the violent inflammation and extensive stasis that are set up.

(*See* Fig. 7.) Among their products are large quantities of gases, chiefly hydrogen and carburetted hydrogen, and the great characteristic of this form of gangrene is the very rapid development of gas in the tissues. Of the organisms that produce somewhat similar effects in the lower animals, we have that known as the cause of symptomatic anthrax in cattle, which leads to the rapid development of crepitating swellings, and, if the animal live long enough, to extensive sloughing (page 46). We have also the bacillus that produces the disease known as malignant œdema, which no doubt must be closely allied to that causing this acute traumatic gangrene. In the case of *malignant œdema*—which, by the way, also attacks man and has occurred in several instances after subcutaneous injections, more especially of musk, in which it seems to grow very readily—the disease is due to a long bacillus which is an anaërobe, and which grows with great rapidity in the subcutaneous tissues, causing enormous swelling of the part from the presence of œdematous fluid, and within a very short time the death of the patient or the animal. This bacillus of malignant œdema can be also obtained from garden earth, and if portions of garden earth be introduced under the skin of guineapigs, a considerable proportion of them will die of malignant œdema after two or three days. (*See* page 46.)

The course of acute traumatic gangrene is generally very rapid, averaging about three days before the death of the patient. Preceding the development of the gangrene the patient does not feel so well, or the shock from which he suffered in the first instance as the result of the accident continues, his pulse becomes irregular, he is very loquacious and very frightened, and soon complains that the wound has become painful, and feels very tense. If the wound be examined the parts around will be found to be hard, œdematous, shining, and tense, the skin being white from deprivation of blood, and very soon becoming dark, livid, or red. At the same time crepitation becomes very marked from the large amount of gas which is present, and the parts may even be resonant on percussion. Once the disease has started it spreads with enormous rapidity, and presents all the ordinary appearances of moist gangrene, bullæ forming on the surface, and the part becoming black and foul-smelling. One may indeed almost see the swelling extending owing to the gas forcing its way through the tissues, and very soon the whole limb will become gangrenous, and the patient will die in about thirty hours after the full development of the gangrene, towards the end having passed into a somnolent condition, lying very quiet, complaining of nothing, with rapid pulse and respiration. There is usually no fever, and the temperature may even be subnormal.

As regards the *treatment*, of course the first point is the prophylactic treatment, which will be discussed in speaking of the treatment of wounds which have not been made by the surgeon (page 226). In the case of such wounds, especially where they are filled with earth, one must bear in mind the great risk of some of these septic diseases taking place, and must therefore be particularly careful to disinfect the wound thoroughly in the first instance. Once the nature of the disease is recognised, the only chance of saving the patient is by immediate amputation far above the gangrenous part; but even at a very early period the chance of saving the patient is very small, only about five per cent. recovering. In amputating, the very greatest care must be taken thoroughly to disinfect the skin around, so as to get rid of any organisms which may be present on the skin, and to avoid soiling the amputation wound with the discharge from the gangrenous part, hence it should be well wrapped up with antiseptic cloths wrung out of strong antiseptic solutions, and this is best done by an assistant, who will afterwards take no part in the operation. The greatest care must be taken not to allow any of the discharge from the wound to run into the part which is being operated on.

(*b*) **Phagedæna.**—The second form of specific gangrene which I need describe is that known as phagedæna, which again is a disease practically never seen nowadays, but formerly extremely common. As I have not myself seen a case, I can only give a very short description from published records. The disease is undoubtedly a parasitic affection, but the nature of the organisms is unknown. It essentially consists in the production in the wound in the first instance of a pseudo-membranous material, beneath which the tissues ulcerate or become gangrenous. The incubation period varies from eight hours to three or four days, and preceding the development of the affection the patient complains of pain in the wound, which becomes swollen, its surface dry, the edges pale, and the granulations of a greyish colour, with commencing formation of a false membrane.

The fully-developed disease is generally described under two forms—namely, an ulcerative and a gangrenous form. In the ulcerative form a pulpy membrane appears on the surface of the wound, and beneath this cup-like losses of substance occur, and subsequently there is rapid ulceration spreading from the part, the surface of the ulcer being covered with a velvety, greyish, fœtid material. In the other form the disease spreads with great rapidity, and the patient dies in from twenty-four to forty-eight hours. Here the wound in the first instance becomes covered with a thicker membrane, which is dark in colour and very pulpy, and which rapidly extends in the planes of the cellular tissue radiating from the wound, leading to rapid sloughing of the skin and muscles, etc., and not uncommonly of vessels, the patient in that case being killed by profuse hæmorrhage. Preceding the development of the affection the patient suffers from malaise. He is in a state of depression, is sleepless, and has nausea; and as the disease develops he has fever, diarrhœa, delirium, and very soon passes into a typhoid condition.

The *treatment* of phagedæna is, of course, in the first instance the prophylactic treatment, which essentially consists in strict antiseptic measures; and, when it is developed, isolation of the patient, and great care must be taken not to infect other persons by the instruments or the attendants. As regards the local treatment, chief reliance is placed on destruction of the pulpy material either by the application of the actual cautery or by the use of perchloride of iron, the pure liquor ferri perchloridi being employed. Of these the actual cautery seems to be the most efficacious, and the parts are very thoroughly destroyed wherever there is the slightest suspicion of this membranous material. Where the perchloride of iron or other caustic is used the surface should in the first instance be thoroughly dried, and then lint soaked in the perchloride of iron should be packed into the wound and left on for twenty-four hours. If the part attacked be an extremity, amputation should, of course, be performed as soon as possible, provided one can get well above the disease, great care being taken not to infect the stump at the same time. In both these cases the general condition of the patient should be attended to, stimulants administered, and every effort made to maintain the strength.

(*c*) **Cancrum oris.**—The third form of specific gangrene to which I need refer is cancrum oris, or noma, which is a disease affecting children, most usually beginning in connection with the mouth, but sometimes on the vulva. This disease generally attacks weakly children of from two to five years of age who are convalescing from some other affection such as measles or scarlatina. In the mouth it usually begins as an ulceration of the gum, the first thing noticed being that the patient complains of pain in the mouth, the breath becomes exceedingly fœtid, and there is an increased flow of saliva, in fact, the saliva runs out of the mouth. On looking in one finds an ulceration about the gums with sloughy edges. In other cases it commences on the inner side of the cheek close to the angle of the mouth, and in the cases where it has begun in the gums it very soon spreads on to the cheek in the neighbourhood. The cheek becomes greatly swollen, intensely shiny, and by-and-by a white spot appears in the vicinity of the angle of the mouth. This spot very soon becomes black, and, in fact, a slough is present, which goes on spreading. In this way it will destroy large portions of the jaws and the skin around the angle of the mouth (Fig. 25). While this is going on the patient is in a very serious condition, the temperature is high, and the pulse very rapid. He is in a semi-comatose state, and usually dies in from four to five days.

This disease is due to long delicate bacilli which are found in large numbers in the tissues at the margin of the slough, and the dependence of the disease on the growth of these bacilli has been thoroughly worked out.

The *treatment* consists in destroying all the affected parts and a portion of the living tissues around in which these bacilli are present, and in order to give the patient a chance at all one has to do what at first sight seems a very barbarous operation. All the parts which are gangrenous must be clipped away, not only the soft parts, but the jaw, and one must go on clipping away tissue till a surface which bleeds in every part is exposed. Having got rid in this way of all the dead tissue, pressure is applied, and the bleeding is arrested, and then strong nitric acid is painted or rubbed into the raw surface. This nitric acid is allowed to act for five or ten minutes, fresh applications being repeatedly made. At the end of that time, if one be thoroughly satisfied that every portion of the disease has been destroyed, the action of the acid may be arrested by the application of carbonate of soda. Anything short of the treatment described will fail in arresting the disease. To leave the sloughs on the part and apply antiseptic washes, or even strong antiseptics, to them is absolutely useless, seeing that the parts where the organisms are growing are the living tissues just beyond the actually dead parts, which cannot be reached if the slough be not in the first instance removed. After removal of the sloughs antiseptic mouth washes are employed, and of the patients treated in this way a considerable number recover, with, of course, very marked deformity, which, however, can be remedied by plastic operation at a later period.

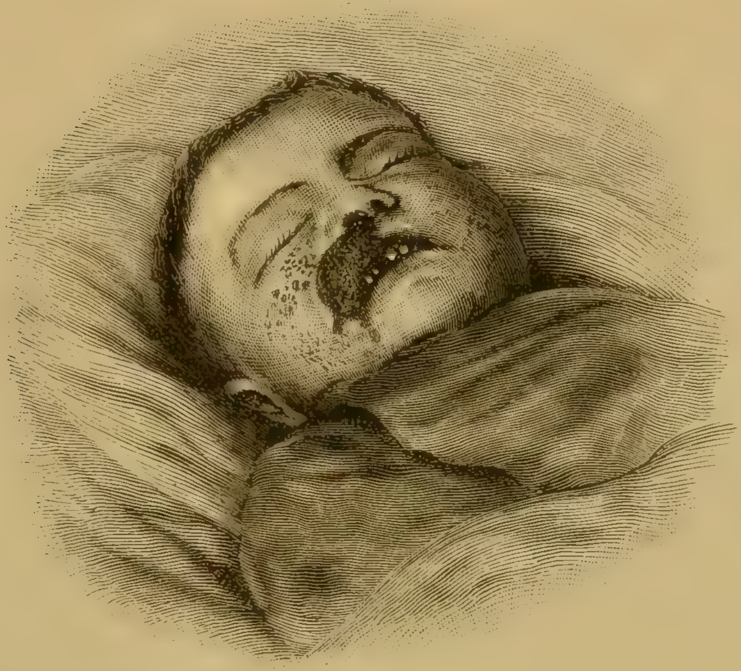

Fig. 25.—Case of Cancrum Oris. Photograph taken after the removal of the sloughs, etc., but before healing had commenced. The patient recovered.

VI. SYNCOPE AND SHOCK

By W. WATSON CHEYNE, F.R.S., F.R.C.S.,

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Syncope. — Syncope and shock must be carefully separated from each other. The actual state of syncope is preceded, when the patient is conscious, by yawning, singing in the ears, vertigo, extreme pallor, nausea and even vomiting, and imperfect vision; but the same condition may appear during the course of an operation, while the patient is unconscious, without, of course, the above premonitory signs, except, perhaps, the vomiting. It is not at all uncommon for a patient during an operation to become pale, with a weak or imperceptible pulse, and very superficial or scarcely perceptible breathing, the condition being temporary, and often ending in an attack of sickness. This more especially arises when the patient is not profoundly under the anæsthetic, and passes off after the sickness, or when complete anæsthesia is again induced. Where the state of syncope is complete the face is pale, the pulse is irregular and extremely feeble or imperceptible, the respiration is very superficial, or momentarily arrested, and the patient is totally unconscious. This condition may last for a few seconds, and then recovery takes place, the first symptoms being sighing, gasping, often sickness, gradual improvement of the pulse, and return of consciousness. In extreme cases, however, syncope may end in immediate death.

The symptoms are essentially due to anæmia of the brain.

Syncope may occur without any injury, as the result of disagreeable mental impressions, or it may follow sudden loss of blood, being in relation not so much with the quantity lost as with the suddenness with which it is shed. Or it may not be dependent upon actual bleeding, but upon a sudden accumulation of blood in one part of the body, as may occur, for example, after too rapid withdrawal of ascitic fluid from the abdominal cavity. Or again, it may follow severe pain or injury to some part richly supplied with nerves, as after a blow over the region of the semilunar ganglia.

The symptoms being essentially due to anæmia of the brain, the first element in the *treatment* is to lower the head, so as to favour the flow of blood to the brain, and then to improve the action of the heart by the sudden application of cold to the chest or head, by the use of pungent inhalations, or by the employment of the interrupted current over the heart.

Shock, on the other hand, is a condition of great depression of the vital activity. It generally occurs after some severe injury, more especially after injury of the abdominal organs, or after the division of large nerves. Shock may occur immediately, or almost immediately, after the injury, or it may come on insidiously, not manifesting itself even for an hour or two, as sometimes happens after railway injuries. The patient who is suffering from shock is usually found lying in a state of complete muscular relaxation, or if he make any movements, they are very irregular and feeble. The face is pale and drawn, the pupils are dilated, there is sweating about the head, the reflexes are very slight, there is diminished sensibility, but not absolute unconsciousness. The patient can answer questions when spoken to, but if not disturbed will generally lie in a semi-conscious condition. The respirations are feeble, irregular, and sighing. The pulse is small, frequent, and dicrotic. At first the pulse-rate is generally slowed, and increased frequency of the heart-beat is regarded by some as a sign of the commencement of reaction. The skin is cold; the temperature sub-normal. The patient does not complain of any pain, and he very often dies in a state of stupor.

In other cases, as I have just mentioned, shock comes on more insidiously, as after a railway accident, where the patient may be able to move about in the first instance, and then gradually passes into a condition somewhat similar to that which has been described.

This condition of shock generally passes off (where recovery is going to take place) in from two to twenty hours, and then *symptoms of reaction* set in, the pulse becomes quicker and stronger, the temperature rises, and may reach even two or three degrees above normal, but unless some complication arises it soon falls again. The marked reactionary symptoms formerly described were essentially septic conditions quite independent of the previous shock, and did not occur where the wounds remained aseptic. The condition formerly spoken of as "prostration with excitement" was probably also a septic condition; at any rate, though I have seen many cases of shock, I have never come across this state, and this is also the experience of other surgeons who operate strictly aseptically.

Pathology of shock.—Numerous theories have been advanced to explain the phenomena of shock, but none of them can be regarded as in any way certain or completely explanatory. One which has been, and is still much in favour, is that shock is due to a paralysis of the vaso-motor nerves, more especially of the splanchnic nerves, resulting in great dilatation of the smaller vessels, especially in the abdomen, which become so distended with blood that but little is left to circulate elsewhere. This view practically implies that the immediate cause of the condition is sudden loss of blood (for the blood is as much lost by its accumulation in the abdomen as by its exit from the body), but the symptoms are distinctly different from those of rapid hæmorrhage. And further, although in some cases it has been found, on *post-mortem* examination, that such accumulation of blood in the abdominal viscera has taken place, in other instances nothing of the kind has been present. The view which seems to me to be most in consonance with the facts, is that which looks on the condition as due to a state of exhaustion of the medulla and the spinal cord, leading to a great reduction in the vital activity generally, and resulting from severe irritation of the peripheral ends of the sensory and sympathetic nerves. Experiments have shown that over-stimulation of sensory nerves will lead to exhaustion and temporary suspension of function of the corresponding centres, and the longer the stimulation is continued, and the greater its severity, the more profound and prolonged is this suspension of function, and the less the probability of recovery.

Treatment of shock.—As to the treatment of shock, no operation should be performed on a patient in this condition unless there be actual bleeding endangering life, and even then chloroform should not be administered. I cannot accept the dictum that complete anæsthesia protects against shock. Where an operation is not immediately necessary, it is much better to check the bleeding, and wait for recovery from the shock, than to perform primary amputation or other necessary operation without delay. Some have advised irritation of the sensory nerves, by enveloping the abdomen in mustard poultices, or by applying strong mustard to the extremities; but if this irritation be severe, it tends rather to increase than to diminish the shock. In any case the head should be kept low, and warmth be applied to the body; in fact, rest and warmth are most essential. If the patient can swallow, stimulants should be given, or, if he cannot swallow, they should be introduced into the rectum. A more immediate effect is produced by the injection of stimulants under the skin, as, for example, the injection of ether (about 40 minims) or of tincture of musk (20 to 30 minims). The latter is very much used in Germany; but it must be either freshly made or the tincture must be disinfected by boiling before use, because bacteria—especially those of malignant œdema—seem to grow with particular rapidity in the musk solution, and in a certain number of cases patients have recovered from the shock and have died of malignant œdema. Strychnine is also of great value in these cases, and is, in my opinion, one of the most important means which we possess of treating this condition. Digitalin, alone or combined with strychnine, is also of service in some instances.

The chief precautions to be observed to prevent the occurrence of shock are the keeping of the patient thoroughly warm, and the employment of strychnine before the commencement of the operation and during its course. Thus, in cases where shock is likely to occur, the operation should be performed in a warm room, and the patient should be placed on a table which is heated by hot water, and at the commencement of the operation about $\frac{1}{30}$th of a grain of strychnine should be injected subcutaneously, and if the condition of shock be manifest during the course of the operation, this, or a smaller dose, combined with ether, should be repeated. Some have advocated the transfusion of saline solutions in large quantities during shock, but I cannot myself think that this is likely to do very much good. The condition of shock being due to exhaustion of the spinal cord, and often accompanied by paralysis of the vaso-motor nerves, the additional non-nutritive fluid introduced by means of transfusion will only accumulate in the already over-distended vessels and will not tend to improve either the nutrition of the cord or to increase the blood-pressure. Where, however, shock is combined with loss of blood, no doubt such treatment may be of advantage, but probably as much good will be done by injecting two or three pints of saline solution into the rectum as by transfusing it directly into the blood, because in these cases of hæmorrhage absorption of fluid takes place with great rapidity from all parts. Where this condition of shock lasts for some hours, coagulation of the blood in the pulmonary vessels is very apt to occur, and thus prevent the recovery of the patient, and no doubt with the view of preventing that complication transfusion may be of benefit.

Secondary shock.—The condition which was formerly described as secondary shock is probably—apart from the delayed form which I have already referred to—really a condition of septic intoxication. The usual history of these cases is that the patient in the first instance suffered from shock after the operation. After he recovered from this shock his temperature rose, and then he suddenly and very quickly passed again into a somewhat similar condition of shock about twenty-four hours later. In most cases this second appearance of shock is coincident with extensive putrefaction of blood contained in the wound, and is no doubt due to septic intoxication. This view is strengthened in my mind by the fact that although I have seen unfortunately a good many cases of shock after operations, I have not met with this condition which was formerly described as being of common occurrence.

Fat embolism.—In connection with certain injuries and wounds, more especially with such as concern the bones, fat embolism may be met with. In this condition, fat enters into the circulation and forms emboli in various organs, particularly in the lungs and in the brain. As a matter of fact, it is now well known that after all fractures fat enters the circulation and is excreted with the urine, being found in it in considerable quantities, two or three days after the injury; but it is only where the fat enters in such excessive quantities as to cause dangerous blocking of numerous minute vessels that the condition of fatty embolism assumes any importance, This condition may come on either immediately after the injury. or (where suppuration takes place) after the establishment of the suppurative process, the pressure in the bone becoming so great that fat is forced into the circulation.

Where we have the condition of fat embolism of the lung, the chief trouble to which it leads is œdema. There is usually more or less sudden and violent dyspnœa, the eyes become prominent, the patient is cyanosed, he coughs, the sputum is frothy and mixed with streaks of blood, or there may be even distinct hæmoptysis, and if the patient lives, this condition may be followed by pneumonia. When the chief embolism is in the brain, the result is delirium, followed by coma; the pulse is small, rapid, and irregular, and the patient generally dies. (*See* Art. XXIX.)

As to the *treatment*, one can only treat the symptoms. In the case of the lungs, dry cupping relieves the patient very considerably; sinapisms to the chest, warmth and stimulants, are also of service.

Traumatic delirium.—After operation we may have the condition known as traumatic delirium, or, where the patient has been a hard drinker, true *delirium tremens* may supervene.

The *symptoms* in the two cases are very much the same, but traumatic delirium is said to differ from delirium tremens in the absence of the trembling of the lips and hands, and in the absence of the history of drunkenness. The symptoms usually set in within twenty-four to forty-eight hours after the injury. The patient becomes excited and extremely loquacious; he imagines that he sees animals and people in the room. He passes rapidly from one topic to another, but generally recurs to the business in which he is employed. He does not complain of any pain; he is very restless, tries to get out of bed, takes off his dressings, and moves his limbs, even though broken. He has no fever, his pulse is regular and normal. The patient may die as the result of this condition, but he usually, after two or three days, falls into a sound sleep and recovers. The great essential in the *treatment* is to make the patient sleep, more especially by the use of opium, and at the same time to see that he takes sufficient nutriment to keep up his strength.

VII. ERYSIPELAS.

By C. B. LOCKWOOD, F.R.C.S.,

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Definition.—Erysipelas (Greek, ἐρυθρός, red; πέλλα, skin) is a diffuse, acute, infective inflammation of the skin or mucous membrane, or of the skin or mucous membrane together with the subjacent cellular tissues, or of the cellular tissues, or of the serous membranes, and is always preceded or accompanied by constitutional symptoms, such as rigors, fever, and vomiting.

Varieties.—Several clinical varieties of erysipelas are met with The classification of Nunneley is here followed.

(1) When the inflammation is confined to the skin, the disease is called *simple cutaneous erysipelas.* Suppuration, except at the point of inoculation, is infrequent in this variety.

(2) When the skin and cellular tissue are both involved, the disease is called *cellulo-cutaneous* or *phlegmonous erysipelas.* In this variety suppuration is the rule. There is no distinct clinical demarcation betwixt cutaneous and cellulo-cutaneous erysipelas. In cutaneous erysipelas there is always some swelling and œdema of the subcutaneous cellular tissue; and in cellulo-cutaneous, or phlegmonous erysipelas, there is always some redness of the skin with vesication or bullæ.

(3) When erysipelatous inflammation spreads in the cellular tissues, with little or no implication of the skin, the disease is called *cellulitis.* In this form suppuration is the rule. By some writers cellulitis is not considered a form of erysipelas, but nevertheless the relationship is close. That cutaneous erysipelas may cause cellulitis there is no doubt. Simple erysipelas, cellulo-cutaneous erysipelas, and cellulitis are all associated with severe constitutional disturbances of a similar type. The exact relationship of these diseases will be further discussed in speaking of their pathology. It is sufficient to say here that as our knowledge of bacteriology increases, it becomes evident that diffuse inflammations of the cellular tissues of an erysipelatous type may be due to various kinds of infection.

Clear and distinct clinical boundaries, as already hinted, do not exist between these three varieties of erysipelas. It is often impossible to say whether a case should be called one of simple erysipelas with slight suppuration, or one of slight cellulitis with cutaneous erysipelas. Difficulties of the same kind occur in separating cellulitis from phlegmonous erysipelas, because in cellulitis there is sometimes redness of the skin with vesication and bullæ.

Frequency.—In sixteen years, from 1878 to 1893 inclusive, 875 cases of simple erysipelas were treated in St. Bartholomew's Hospital, and 800 cases of phlegmonous erysipelas and of cellulitis. The numbers from year to year exhibit great fluctuations. The great prevalence of erysipelas in certain years suggests that it may, like other infective diseases, become epidemic. The spread of it seems also to be favoured by the autumn and winter seasons.

I. SIMPLE CUTANEOUS ERYSIPELAS.

Seat.—Cutaneous erysipelas has a marked predilection for certain regions of the body, and is especially prone to attack the face and head. Here it can usually be shown to have originated in some scratch or abrasion, or in an acne pustule, or from suppuration around the fang of a tooth, and to have spread from the interior of the throat or nose. It is a not infrequent complication of minor operations upon the scalp, such as those for the removal of sebaceous tumours. When the attack is in full career, the point of inoculation may not be found, having disappeared, or become obscured, by the violence of the inflammation.

Incubation period.—The incubation period of cutaneous erysipelas is uncertain, and is variously stated as being eight hours to two days (Ritzmann), or from one to eight days (Pujos). When Fehleisen inoculated cases of cancer and tubercle with the Streptococcus erysipelatis, the incubation period was from fifteen to sixty-one hours, reckoning from the time of the inoculation to the first rigor, which usually coincided with the rash. In injuries and in operation cases the erysipelatous blush usually appears after reaction. When erysipelas complicates wounds it begins at their margins, but may spread to distant regions.

Symptoms.—The **local symptoms** of cutaneous erysipelas are objective and subjective. The most striking objective symptom is the rapidly spreading *redness of the skin.* This is at first a mere blush, which disappears easily on pressure, and then a vivid red, but later it becomes a deep crimson, which is harder to efface by pressure, and which returns as soon as the pressure is removed. The erysipelatous skin is *hot* to the touch, and there are *swelling* and *œdema.* The latter are greatest where the cellular tissue is loose, as in the eyelids, penis, and scrotum. On the other hand, when the skin is bound down, as at Poupart's ligament, the iliac crest, the palms of the hand, soles of the feet, and at the chin, the swelling does not occur, and the disease seems to be arrested.

The swelling which is caused by erysipelas pits on pressure, but is hard, especially at the margin where the disease is spreading. The induration is caused by the effusion of inflammatory lymph into the cutis vera. Cases in which the hard edge is very marked have been called by the name erysipelas marginatum. The margin of erysipelas is not abrupt where the inflammation is receding.

When the swelling increases, and is considerable, the skin becomes smooth, tense, and shining. Its surface is covered with *vesicles*, or studded with bullæ full of yellow serum. At first the serum is clear and transparent, but afterwards it becomes turbid. The bullæ are occasionally absent, but the *vesicles* are said to be invariably present, although a lens may be required to see them. As erysipelas subsides the vesicles and bullæ burst, dry up, and form slight crusts.

In erysipelas the *lymphatic glands* which drain the affected area are always swollen, tender, and inflamed. The deep lymphatics may oftentimes be felt as hard and tender cords running towards the glands, and the superficial lymphatics may be marked by the redness of lymphangitis. When the morbid histology of erysipelas is described, it will be seen that the lymph spaces and lymphatics of the skin are especially involved in the inflammation; hence the disease has been called an infective capillary lymphangitis. Sometimes the inflammation of the lymphatic glands seems to precede the outburst of cutaneous erysipelas.

When erysipelas attacks a wound repair ceases, and its surface becomes dry and glazed. It secretes a little sanious fluid and is devoid of pus or granulations. Granulations which had formed become anæmic and cease to grow, and may disappear. Hæmorrhages sometimes occur, and a layer of ashen lymph, sometimes called that of *wound diphtheria*, may form.

In simple cutaneous erysipelas the skin occasionally sloughs, or portions become gangrenous. These complications are, however, commonest in phlegmonous erysipelas.

Now and then erysipelas is attended with a curious bronzing of the skin.

One of the most characteristic local subjective symptoms of erysipelas is the intense irritation, smarting, or *burning pain* which accompanies it. To this last may be owing its popular name of "*St. Anthony's fire*." Sometimes the pain is of the severest neuralgic type, and I have had under my care a case in which the most violent pain and lymphadenitis preceded by forty-eight hours an outburst of cutaneous erysipelas.

The inflamed parts are stiff and incapable of full flexion or extension.

In *erysipelas of the mucous membranes* the constitutional and most of the local symptoms are the same as when the skin is attacked. The mouth, nose, pharynx, larynx, the female genital tract, and the rectum are its favourite seat. The involved membrane becomes intensely red, swollen, and superficially ulcerated. Bullæ form, but soon break, leaving superficial ulcers. The special functions of the part affected are interfered with; for instance, erysipelas of the larynx causes dsypnœa and aphonia. Œdema of the glottis is one of the gravest dangers in erysipelas of the air-passages.

The constitutional symptoms of erysipelas are very decided. Before an attack the health may have been good, but the rash is usually preceded for some hours by a heightened temperature and feelings of illness. Chilliness is complained of, and *rigors* are frequent, but not invariable. In infants and children convulsions usually take the place of rigors, and in adults epileptiform convulsions are occasionally seen. During the rigor the *temperature* is at its height, but it remains high, 103° to 105° Fahr., throughout the disease. The temperature falls slightly towards morning and rises towards night, as in the other specific fevers—acceleration of the pulse and respiration accompanies the increased temperature. At the beginning of erysipelas the *tongue* is foul and the appetite bad, and vomiting is common. The *bowels* are usually confined, but there may be diarrhœa, with black, offensive motions. Occasionally bile is vomited, and there is jaundice. Those subject to malaria are said to suffer from this bilious type of the disease. Headache and pains in the back, joints, and limbs are usually complained of. Epistaxis sometimes complicates facial erysipelas.

Delirium is exceedingly frequent during erysipelas. It is worst at night, and may be very noisy, and accompanied by troublesome restlessness and delusions. Drunkards may develop delirium tremens. The urine may contain albumen, and nephritis is not an unusual complication of erysipelas. Those with chronic diseases of the kidney seem predisposed to its attacks.

Duration and termination.—The duration of cutaneous erysipelas is uncertain. An ordinary attack seldom lasts less than a week or more than a fortnight. Sometimes the eruption wanders from one region to another (*erysipelas migrans* v. *ambulans*), and when each new area is attacked, there is an exacerbation of the constitutional symptoms. I have seen a case of this severe and dangerous type which lasted nearly three weeks. Although there was profound constitutional disturbance, and the temperature rose to 107° F., the patient, who was young and vigorous, with sound organs, eventually recovered. In erysipelas ambulans the same area is never affected twice.

In erysipelas, a fatal ending is to be apprehended when the strength fails, the respiration becomes quick and shallow, when the pulse is feeble and running, the tongue dry and brown, the mind obscured, with muttered delirium, or when stupor and coma supervene with the involuntary passing of urine or fæces.

A scanty flow of urine loaded with albumen would be of grave import, as indicating acute nephritis.

Erysipelas often ends abruptly, the temperature falling almost suddenly from a high degree (103° F.) to normal, with a corresponding amelioration of the general symptoms. This defervescence is more common in simple cutaneous erysipelas than in the cellulo-cutaneous form, or in cellulitis. It is obvious that no rapid change could occur in the inflamed and pus-containing cellular tissues of the last two diseases. In some subjects erysipelas has an extraordinary tendency to *relapse* after an interval of a few months. This is seldom seen as a complication of wounds, but the existence of such a peculiarity would have to be taken into consideration before performing an operation upon anyone subject to it. Fehleisen, in his inoculation experiments, found that no permanent immunity was acquired. Patients could be successfully inoculated over and over again, provided that an interval of a month or two was allowed to intervene.

Complications.—Some of the complications of erysipelas have been mentioned. Many are purely local, or due to direct extension from the original seat; others are of a more general character. Amongst the local complications may be mentioned suppuration, ulceration, sloughing, gangrene, lymphangitis, lymphadenitis, phlebitis, neuritis, acute inflammation of sheaths of tendons, and of articulations, and acute suppurative periostitis and necrosis. Among one hundred and thirty cases of erysipelas Ritzmann records that the joints of the foot were twice involved, and the knee, elbow, and shoulder each once. The joints named were acutely inflamed, and in some instances suppuration followed.

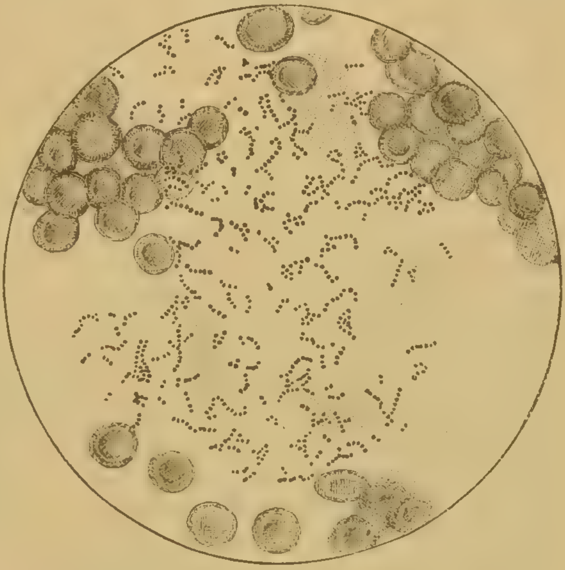

Fig. 26.—Streptococci in the Heart's Blood from a Case which died of Septicæmia after Erysipelas. (From a microphotograph by Mr. Cosens.)

Amongst the later local complications may be mentioned a persistent œdema, which sometimes ensues, and produces permanent thickening or elephantiasis. When the sheaths of tendons have been implicated, limitations of movement in the fingers or toes may result, and ankylosis when the joints have been involved.

When erysipelas attacks the organs of special sense, it may destroy sight, or smell, or hearing, or greatly impair these functions.

Muscular paralyses occasionally follow erysipelas, especially of the pharynx after erysipelas of the fauces (Todd). Many complications are caused by direct extension from the original seat. For instance, in facial erysipelas the inflammation may spread to the eye, conjunctiva, lachrymal apparatus, orbit, nose, pharynx, or larynx, or to the lungs and air-passages. From the pharynx it sometimes extends along the Eustachian tube to the middle ear. In the air-passages it may cause a fatal œdema of the glottis. Perhaps the parotiditis which sometimes complicates facial erysipelas is due to extension along Stenson's duct. Erysipelas of the penis may spread to the urethra and bladder. Erysipelas of the female genitals (erysipelas puerperale) may spread to the uterus, Fallopian tubes, and peritoneum.

From wounds of the abdominal walls erysipelas may spread by extension to the peritoneum, or, after injuries to the head, from the scalp to the meninges.

The streptococcus of erysipelas may enter the blood in such quantities as to cause fatal septicæmia. In a case of this kind I found the streptococcus in the blood of the right auricle (Fig. 26).

Pyæmia is a frequent cause of death in erysipelas. It is more frequent in the cellulo-cutaneous and cellular varieties. It is due to the entrance of pyogenic organisms, especially of streptococci, into the circulation, and their subsequent passage from the circulation into the tissues. In a case of Volkmann's it was thought that a clot in the crural vein had become infected from an erysipelatous wound (Gussenbauer).

Acute nephritis is a common complication of erysipelas. As a rule, it is transient and leaves no ill-effects. It is revealed by the occurrence of a cloud of albumen in the urine. In drunkards it is to be remembered that there may be chronic parenchymatous nephritis. The acute nephritis may occasionally end in fatal uræmia. Congestion of the other abdominal organs may occur in erysipelas, and there may be enlargement of the spleen and of the liver.

Bronchial catarrh, pneumonia, and pleurisy are possible complications of erysipelas. Pleurisy is said to be commonest in erysipelas of the chest wall. Pneumonia is a dangerous complication, and may attack one or both lungs. It may establish itself without cough or pain, and may therefore be overlooked. The pneumonia is of the croupous variety. As in other diseases of the same type, hypostatic congestion and œdema of the lungs are met with in erysipelas. Inflammations of the other serous membranes, of the pericardium, peritoneum, or arachnoid, are occasional complications of erysipelas. The frequency of delirium in erysipelas probably led to the belief that meningitis is commoner than it really is. As a rule, meningitis is due to the inflammation spreading directly through a fracture of the skull, or through the orbit.

Prognosis.—The prognosis of simple cutaneous erysipelas is favourable. Out of 875 cases treated in St. Bartholomew's Hospital, 31 died (3·5 per cent. nearly). The mortality was rather less amongst females than amongst males; 10 out of 391 females died (2·57 per cent.), whilst 21 out of 484 males died (4·3 per cent.). The mortality from phlegmonous erysipelas and cellulitis is far greater than that from simple cutaneous erysipelas. Out of 889 cases of phlegmonous erysipelas and cellulitis treated in St. Bartholomew's Hospital, 99 died (11·1 per cent. about). Out of 685 males, 80 died (11·6 per cent. about); and out of 204 females, 19 died (about 9·0 per cent.). The preponderance of cellulo-cutaneous erysipelas in males is explained by their greater liability to injury. Neglected and lacerated wounds of the head and extremities were the commonest modes of infection in these cases.

The prognosis of erysipelas depends, therefore, to some extent, upon its degree. The age of the patient is also an important consideration in this as in other diseases of similar kind. Infants and aged people are very apt to succumb. In the aged even simple cutaneous erysipelas is dangerous. Amongst infants a variety of erysipelas, called *erysipelas neonatorum*, sometimes attacks the skin of the abdomen during the separation of the remains of the umbilical cord. Suppuration and gangrene of the skin often occur. Erysipelas neonatorum is sometimes complicated by phlebitis of the umbilical vein. It is met with where puerperal fever, puerperal pyæmia, and puerperal septicæmia are prevalent.

Erysipelas is an occasional sequela of vaccination. The streptococcus of erysipelas has been found in impure vaccination lymph (Copeman). Defects of the kidneys, heart, or lungs, add to the gravity of an attack, as do also habits of intemperance. It is obvious, therefore, that the condition of the great organs should always be investigated, and the habits of the patient inquired into.

Diagnosis.—The differential diagnosis of cutaneous erysipelas is sometimes exceedingly difficult. From *simple erythema* it depends upon both the constitutional symptoms and upon the local appearances. In simple erythema there is no rigor or high temperature, nor acceleration of the pulse and respirations, vomiting is infrequent, and the redness has no board-like margin; vesicles and bullæ are wanting, and the lymphatic glands or vessels are not affected. An acute attack of *herpes* is sometimes hard to tell from erysipelas. Both may be accompanied by constitutional symptoms, and in both there is redness and vesication of the skin, with enlargement of lymphatic glands. However, in herpes both the local and general symptoms are less severe, and the eruption is usually confined to the distribution of certain nerves, and does not cross the middle line of the body. Erysipelas might also be confused with *acute eczema* or with acute dermatitis. The latter are sometimes caused by the chemicals used for the disinfection of the skin before operations, or by those contained in dressings. Here, again, the slightness of the constitutional symptoms and the local characters, especially the presence of eczematous papules, crusts, and vesicles, ought to make the diagnosis easy. The early stages of inflammatory wound gangrene are not unlike erysipelas. In acute *spreading traumatic gangrene* there is also redness and swelling of the skin, with the formation of bullæ. But in this most fatal disease there is more profound constitutional disturbance; the bullæ contain blood-stained serum, and there is emphysematous crackling in the subcutaneous tissues.

Pathology.—After death from erysipelas the *post-mortem appearances* are similar to those in other infective diseases. The redness of the skin disappears after death, but the desquamation, vesicles, or bullæ remain. There is also some swelling and œdema of the cutis, with enlargement of lymphatic glands, and occasionally some thrombosis of veins. The fluid in the œdematous tissues is rich in leucocytes, and in sections many cells are found along the course of the vessels. The kidneys and liver and other internal organs are congested, and there is often marked enlargement of the spleen. The lungs are often pneumonic, and the serous sacs contain an excess of blood-stained fluid. The arachnoid, pericardium, pleura, or peritoneum may be inflamed. These inflammations cause fibrinous and purulent effusions. The lymph and pus contain streptococci. The streptococcus of erysipelas should also be sought for in the inflamed skin, in vesicles or bullæ, in the blood, in the fluid of the serous sacs, and in the capillaries of the liver, lungs, and kidneys. During life the blood in erysipelas forms a firm clot, but after death it is thin and watery.

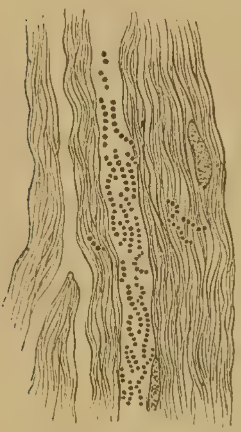
Fig. 27. — Erysipelas: Streptococci in lymph spaces of skin. (After Cornil and Babes).

Great light has been thrown upon the *pathology* of erysipelas by the labours of Nepveu, Fehleisen, Koch, and of many others (page 27). Erysipelas is caused by infection with a streptococcus, which grows along the lymph spaces of the skin (Fig. 27), and causes first a coagulation of their contents, and afterwards excites acute inflammation. The streptococci may be demonstrated by Gram's method of staining (page 51), in skin taken from the extreme edge of the spreading margin. It is difficult or impossible to demonstrate them in the older and more indurated skin. Here they are obscured by the leucocytes and fibrinous exudation which fill the lymph spaces and lymphatics. The streptococcus also grows in the periphery of the hair follicles, which explains the loss of hair which follows an attack (Cornil and Babes). The streptococcus can be grown in culture media by inoculating them with portions of the spreading edge. With such cultures Fehleisen caused typical erysipelas. The streptococcus of erysipelas has been found in the fluid of the bullæ (Orth.), in the blood in fatal cases (*see* Fig. 26), in the pericardial fluid, in the muscular walls of the heart, in the lungs, liver, spleen, and mesenteric glands (Pfuhl). In ordinary cases it is probable that the streptococcus of erysipelas does not enter the blood (Fehleisen). Eiselsberg failed to find them in three cases.

Cornil and Babes give an excellent description of the histological changes in erysipelatous skin. Migratory cells infiltrate the bundles of connective tissues, especially around the blood-vessels and lymphatics, and around the fat lobules. The migratory cells are more abundant in the dermis than in the papillæ. The fixed cells are swollen and undergoing division. There is a sero-fibrinous exudation in the dermis and subcutaneous tissues. The endothelium of the lymph spaces is swollen, and many subcutaneous lymphatics are filled with migratory cells.

Causes.—The *general predisposing causes* of erysipelas may be in the environments, as overcrowding, bad drainage; or in the habits of the patient, especially intemperance. Starvation, debilitating diseases, diabetes, Bright's disease, gout, and malignant disease all predispose to erysipelas, as do also previous attacks. Infants and the aged are probably predisposed.

The *local predisposing causes* are dependent upon the position of the injury in places which facilitate sepsis, such as the scalp, scrotum, female organs of generation, the mouth and anus. The different varieties of erysipelas are also more liable to complicate wounds which are lacerated and contused, simply because they are more likely to be infected, and are harder to disinfect.

The exact relationship of the streptococcus of erysipelas to cellulo-cutaneous erysipelas is still the subject of controversy. There is no doubt whatever but that cellulo-cutaneous erysipelas is caused by a streptococcus. Also that this cannot in most cases be distinguished by its morphology, growth, and effects from the streptococci of simple cutaneous erysipelas. Fehleisen denied that the streptococcus erysipelatis could cause suppuration; but Fraenkel, Kirchner, and others have shown that this is not correct. Those who have hitherto denied that the streptococcus of erysipelas can cause suppuration assert that the streptococcus seen in cellulo-cutaneous erysipelas and cellulitis is the Streptococcus pyogenes. Others, again, think that cellulo-cutaneous erysipelas may be caused by a mixed infection with Streptococcus erysipelatis and Streptococcus pyogenes. However, these two organisms are so nearly alike in morphology, growth, and action that they are indistinguishable (Cornil and Babes, Sternberg). It has been stated that they may be separated by slight differences in their chemical products.

It was at one time a question whether erysipelas was contagious or whether it was infectious. Now that it is known to be due to infection with streptococci, such a discussion is unnecessary. The streptococcus of erysipelas resides in pus, blood, clothing, upon the surface of the body, in the mouth, nose, or vagina (Sternberg), in the excreta, and upon instruments, towels, and sponges. Von Eiselsberg found it in the air of Billroth's wards, Haegler in the air of Socin's wards, and Emmerich found it in the air of a dissecting-room.

II. CELLULO-CUTANEOUS ERYSIPELAS.

Cellulo-cutaneous erysipelas has a close clinical relationship to the cutaneous variety. There are, however, some differences, especially in the local symptoms, due to the infection of the subcutaneous cellular tissues. It occurs as a complication of operations and injuries. I have seen fatal examples after the most trivial proceedings—such as the removal of sebaceous cysts, fatty tumours, adenomata of the breast, after radical cure of hernia, radical cure of varicocele, and amputations of the breast.

Symptoms.—The *constitutional symptoms* of cellulo-cutaneous erysipelas are the same in kind, but more severe in degree than those of the cutaneous form. At the beginning, severe and repeated rigors are the rule. These may simulate intermittent fever. The temperature is high with considerable exaltations during the rigors. The frequency of the pulse and respirations is markedly increased. Delirium is common, and there is an absence of sleep, and great restlessness. The patient's aspect is haggard and anxious. Pneumonia, pleurisy, nephritis, pyæmia, septicæmia, and the other complications of simple erysipelas are all of frequent occurrence. In drunkards delirium tremens is to be feared.

Cellulo-cutaneous erysipelas occurs in broken-down subjects. The aged, the starved, and those with diseases of the kidneys and liver are predisposed to its attacks. It is prone to run an asthenic course, with rapid and feeble action of the heart, quick and shallow respirations, diarrhœa, muttered delirium, with subsultus tendinum, and the formation of sordes or of aphthous patches about the mouth and pharynx.

Suppuration is one of the chief *local symptoms* of cellulo-cutaneous erysipelas. In cutaneous erysipelas, suppuration is the exception. Sloughing, ulceration, and gangrene are also frequent complications of cellulo-cutaneous erysipelas. At first the colour of the skin in cellulo-cutaneous erysipelas is a deep, dusky red, which disappears on pressure; afterwards it becomes irregular and marbled. There is great swelling and œdema, and the whole part looks twice its natural size. The edge of the swelling is not abrupt, nor board-like. The epidermis is raised into bullæ filled with blood-stained serum. The lymphatic vessels are usually inflamed, but may not be felt—owing to the œdema. Irregular red lines may mark the course of inflamed superficial lymphatics. The affected area is hot, and the patient complains of intense throbbing pain. The cutaneous vessels are engorged with blood, and vessels which are ordinarily small bleed smartly for a time after their division.

The subcutaneous fat and cellular tissues are swollen and infiltrated with yellow serum, which gives them a gelatinous appearance. After awhile this fluid becomes purulent, and the skin is separated from the deep fasciæ by tissues soaked with thin pus. This interferes with nutrition and nerve-supply, and leads to sloughing and gangrene of the skin. The sloughing cellular tissues may escape from the apertures with the purulent discharge. They have been compared to wet tow, wet chamois leather, and so forth. True abscesses or ulcers are seldom seen in cellulo-cutaneous erysipelas.

The swelling of cellulo-cutaneous erysipelas is at first soft and œdematous, and pits on pressure. As the tension increases it becomes hard, and the skin over it shining. When suppuration occurs it imparts a characteristic "boggy" sensation; true fluctuation is seldom felt. The pus has no tendency to point, as in acute abscess, but diffuses itself along the cellular planes. It often strips vast areas of skin from off the deep fasciæ. In a case under my care the whole of the skin of the back from neck to nates was separated from the underlying fasciæ. The skin was saved by numerous timely incisions, and the man recovered. When cellulo-cutaneous erysipelas extends beneath the deep fasciæ it may cause extensive destruction of tendons, necrosis of bones, and acute suppurative arthritis.

As a rule cellulo-cutaneous erysipelas does not extend beneath the deep fasciæ. Sometimes, however, the cellular tissues which surround the muscles, and deep vessels and nerves, are extensively involved. I have treated a case in which the muscles of the thigh and the great sciatic nerve were dissected out by the purulent inflammation, which also extended into the iliac fossa.

The scalp is a favourite seat of cellulo-cutaneous erysipelas. Here the suppuration invades the loose cellular tissue betwixt the cranial aponeurosis and the pericranium. It is usually confined by the attachments of the aponeurosis, and of the frontalis and occipitalis muscles.

The post-mortem appearances in cellulo-cutaneous erysipelas and in cellulitis are very similar to those in simple cutaneous erysipelas. The extensive diffuse inflammation and suppuration in the cellular tissues form the most marked features.

III. CELLULITIS.

There is the closest clinical relationship betwixt cutaneous erysipelas and cellulo-cutaneous, and they are both due to streptococcus infection. There is no doubt whatever that streptococci are also the cause of diffuse cellulitis. I have found them in pure culture in pelvic cellulitis, and have seen them filling the lymph-paths in diffuse cellulitis of the neck (cynanche Ludovici). But there is also no doubt that diffuse cellulitis may be caused by other kinds of infection. (*See* Fig. 28.) One of the worst and most familiar kinds of diffuse cellulitis is caused by the extravasation of putrid urine into the cellular tissues of the perineum, scrotum, penis, and abdomen. Severe and fatal cellulitis also follows upon the bites of venomous reptiles and insects, and upon dissection and post-mortem wounds, and poisoned wounds of various kinds (Art. XV.). In many of these there is doubtless infection with streptococci; but at the same time other organisms are present, and are probably equally effectual in the production of the cellulitis. (*See also* page 91)

In cellulitis the cellular tissues of any region may be involved. After parturition and gynæcological operations, for example, pelvic cellulitis may occur. When the cellular tissue of the neck is infected from an acute necrosis or osteomyelitis of the jaw, or from an ulceration of the pharynx, tonsils, or larynx, a dangerous and acute cellulitis (cynanche Ludovici) arises.

Symptoms.—In cellulitis there is profound constitutional disturbance of a type similar to that in cellulo-cutaneous erysipelas.

The absorption of septic materials is, perhaps, greater, and death from sapræmia, septicæmia, and collapse is more frequent.

The *local characters* of cellulitis are extensive, hard, deep-seated and brawny swelling, with heat and burning pain. The skin over the swelling is tense, œdematous, and dusky red. The lymphatic glands are inflamed and enlarged, and the veins are often filled with septic clots.

Gangrene of the skin is a complication of cellulitis. It is more common in some regions than in others, and in cellulitis caused by extravasation of urine the skin of the penis and scrotum are destroyed, unless saved by incisions. The cutaneous gangrene of cellulitis is sometimes mistaken for that rare affection called spontaneous gangrene of the penis and scrotum.

Cellulitis does not necessarily begin at the point of inoculation, but may burst out in cellular tissues at some distance. For instance, in dissection wounds of the fingers, the axilla and chest wall are often affected, whilst the arm is but slightly swollen with some lymphangitis.

The changes in the cellular tissues are the same as in cellulo-cutaneous erysipelas. The purulent fluid is diffused along the cellular planes, and seldom forms collections sufficient to afford fluctuation. Extensive gangrene of the cellular tissues ensues. The characters of the purulent fluid vary according to the causation. In cellulitis due to extravasation of urine, and in cellulitis of the neck, it is often horribly fœtid, because putrefactive bacilli are mixed with streptococci and other bacteria.

Treatment of erysipelas.—The *preventive treatment* of erysipelas requires a proper knowledge of the properties and habitat of the streptococcus of erysipelas, and of the measures required for its destruction. It is destroyed in ten minutes by a heat of 52° to 54° C. (Sternberg), and is killed almost instantly by boiling water or steam. Chemicals kill it in course of time, provided that the conditions are favourable for their action. The streptococcus was dead after two hours' exposure to a solution of mercuric chloride 1 part in 2500 parts of water, or to a solution of carbolic acid 1 part in 300 parts of water. Of course, stronger chemicals act in a shorter time.

In regions of the body where disinfection is difficult or impossible, erysipelas is more liable to follow operations. With care, the transmission of erysipelas is rare. The sister in charge of the erysipelas ward at St. Bartholomew's tells me that only one nurse has acquired the disease during a period of nine years. Scrupulous care is taken in washing and disinfecting the hands and instruments. When erysipelas occurs in a general ward, the case ought to be at once isolated.

Care in the use of antiseptics has practically banished erysipelas. I have not yet had a case after an operation in which no septic focus pre-existed, and can only recall two slight attacks in septic cases. In these the disease was undoubtedly carried by house-surgeons or nurses who had not, perhaps, realised what stringent precautions are required.

An extraordinary number of remedies has been vaunted in the *immediate treatment* of cutaneous erysipelas. Inasmuch as the disease usually runs a short and favourable course, and nearly always ends in recovery, it is obvious that a favourable opinion may easily be formed as to the benefits of particular remedies. It is doubtful whether any internal remedy has any marked effect upon the course of the disease.

By some a 30 per cent. solution of perchloride of iron is painted upon the affected part, and half-drachm doses are given internally. Others administer carbonate of ammonium with tincture or decoction of cinchona.

Cutaneous erysipelas, cellulo-cutaneous erysipelas, and cellulitis are all diseases which make great demands upon the strength. It is therefore necessary, as a rule, to give a generous diet with stimulants, milk, beef-tea, beef or mutton essence, eggs, eggs whipped with wine or brandy, and so forth. The best stimulant is usually that which is taken best; brandy, port, burgundy, champagne, beer, or porter, are all useful. The bowels ought to be regulated, and anodynes given to induce sleep and relieve restlessness.

Locally, the abstraction of blood by leeches or by lancet puncture relieves the pain and tension. Bleeding should be encouraged with hot fomentations.

Nitrate of silver seems to have a decided influence in arresting the spread of cutaneous erysipelas. This is best applied by painting the whole area with a solution made by dissolving twenty grains of solid nitrate of silver in an ounce of the spiritus etheris nitratis. At St. Bartholomew's Hospital the part affected by cutaneous erysipelas is usually covered with an ointment composed of a drachm of prepared chalk and an ounce of lard. This is very soothing, and affords great relief to the burning and tingling. Dusting with starch, oxide of zinc, flour, or bismuth is also soothing and agreeable. It is often beneficial to cover the inflamed area with cotton-wool.

When erysipelas complicates wounds, care should be taken to see that there is a free exit for discharges, and to cover the whole area with a large and warm boracic fomentation. This ought to be renewed every two or three hours, and is very grateful to the patient.

Ichthyol has been greatly advocated by Nussbaum and others for the treatment of cutaneous erysipelas. After a fair trial I have come to the conclusion that the pain which it causes exceeds any benefit which may ensue.

In cellulo-cutaneous erysipelas and cellulitis, to prevent gangrene and to give exit to discharges, a series of incisions, each about two inches long, should be made into the inflamed and tense skin and cellular tissues. These incisions should be made where the skin is most affected, and in the long axis of the limb and parallel to vessels and nerves which are to be avoided. Incisions into the scalp should radiate from the vertex. Incisions usually cause smart bleeding. This is easily arrested either by ligature, or by plugging with antiseptic gauze.

To make the incisions it is in most cases necessary to give an anæsthetic. After an incision has been made great care should be taken not to strip up the undermined skin with fingers or instruments, otherwise its remaining vascular supply may be destroyed, and it may slough. A very large, warm, carbolic or boracic fomentation should be applied. Great benefit is given by immersion in a bath of hot water night and morning. As a rule, this is only applicable to the arm.

In subfascial cellulitis incisions of great extent may be required to give exit to pus, and to permit the cavity to close. I have had to divide the fascia lata from the great trochanter of the femur down to within two inches of the external tuberosity. Collections of pus should be thoroughly drained and the cavity washed out each day with some disinfectant. A solution of a drachm of tincture of iodine to a pint of water is a safe and fairly efficient lotion. If a stronger disinfectant be required, the liquor iodi may be used in the same proportion. If there be great fœtor, 5 per cent. carbolic lotion should be used instead of water for mixing with the liquor iodi. Iodoform and iodoform gauze are suitable dressings for the sloughing or ulcerated wounds in cellulitis.

In Ludwig's angina, or diffuse cellulitis of the neck, the incisions ought to be made in the middle line from the chin to the hyoid bone. It may be necessary to carry them to some depth before a proper exit is given to the putrid fluid.

In cellulitis of the ischio-rectal fossa, free incisions should be made, radiating from the anus to avoid the hæmorrhoidal vessels and nerves.

To prevent adhesions of joints or tendons, passive movements and massage may be required at a later period. Œdema may be treated with elastic bandaging. The other and more serious complications, such as pneumonia, nephritis, and so forth, must be dealt with as they arise.

VIII. PYÆMIA.

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Definition.—The word pyæmia, which we owe to Piorry, is derived from the Greek *πῦον* (pus) and *αἶμα* (blood). It originated in the belief that the disease was due to the entrance of pus into the blood. Although this assumption contains an element of truth, it now requires to be modified. Other names have been given to pyæmia to mark its close connection with suppuration. One of the commonest is *purulent infection*, or pus infection. The word *septicæmia* was applied to those whose blood was supposed to have been poisoned with putrid material.

Since the labours of Ogston, Koch, Klebs, Krause, Rosenbach, and others, it has been understood that pyæmia is caused by the entrance of certain bacteria into the blood, where they live and multiply, and whence they pass into the tissues, joints, or serous cavities, causing purulent deposits. (*See* page 25.)

Thus pyæmia may be defined as a general infection of the blood with pyogenic organisms, leading to the formation of multiple abscesses, and accompanied with intermittent fever and other manifestations of septic disease. More briefly, pyæmia may be defined as septicæmia, with, in addition, the formation of metastatic abscesses.

Mortality.—Pyæmia is becoming a comparatively rare disease. But when it occurs, it is exceedingly fatal. Of 94 cases which were treated in St. Bartholomew's Hospital in sixteen years, 66 died.

Sex and age.—The proportion of the two sexes is almost equal. Among the 94 cases just quoted, 48 were males and 46 females. Males usually die of pyæmia after injuries and operations, or after acute infective osteomyelitis. In females the smaller liability to injury and osteomyelitis is compensated for by the dangers of parturition and of sepsis connected with the reproductive organs. The statistics given by Gussenbauer show that out of 50 cases of pyæmia 44 died. There were 24 men with 1 case of recovery, and 26 women with 5 recoveries.

The tables of Gussenbauer show also that pyæmia is extremely rare in childhood, and hardly occurs during the first five years of life. It is commonest betwixt the ages of sixteen and thirty.

Conditions of occurrence.—Pyæmia is met with as a complication of any wounds and breaches of surface which are exposed to infection, and of any form of sepsis and suppuration. It has occurred during suppuration following fractures, injuries to joints and serous sacs, burns, scalds, and frost-bite, during the course of cystitis, prostatitis, pyelitis, dysentery, gangrene, osteomyelitis, erysipelas, diphtheria, small-pox, scarlatina, and other diseases. Pyæmia is a not infrequent complication of parturition.

As a rule, the way by which the bacteria enter is quite obvious. Sometimes, however, the point of inoculation is difficult to find, being situated in the middle ear, in the mouth, pharynx, or œsophagus, in the rectum or alimentary tract, or about the gall bladder. In some cases the lesion may have healed before the patient came under observation, or may have been so trivial as to have escaped notice. The name of "idiopathic pyæmia" has been applied in the above circumstances. Such a term, however, does not imply that the disease differs from the ordinary type, beyond that the seat of inoculation is obscure or overlooked. The same remark applies to the expression "spontaneous pyæmia," which is sometimes applied to cases in which the mode of infection is not known. The differentiation of pyæmia into *acute* and *chronic* rests upon a sound clinical basis. As a rule, pyæmia is fatal in a few days, but cases are met with in which it lasts months or even years, all the clinical symptoms being the same as in acute cases, but less severe and more prolonged.

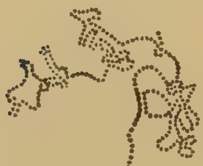

Fig. 28.—Streptococcus Pyogenes, growing from a case of cellulitis. (From a microphotograph by Mr. Cosens.)

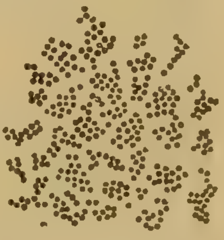

Fig. 29.—Staphylococcus Pyogenes Aureus, grown from pus upon agar-agar. (From a micro-photograph by Mr. Cosens.)

The infecting micro-organisms.—In pure pyæmia it is probable that the infecting micro-organisms are always the pyogenic cocci (page 25). It is not accurately known which kind is the most frequent. In Senn's summary of the investigations of Rosenbach, Pawlowsky, Besser, and Schüller 69 cases of pyæmia are mentioned. In all of these cases either the blood, pus, or fluids of the tissues were examined, and streptococci were found in 37, staphylococci in 23, a mixed infection of staphylococci and streptococci in 5, and Staphylococcus aureus alone in 4. The streptococcus is the same as that ordinarily met with in the diffuse suppurations, especially in cellulitis, and is the Streptococcus pyogenes (Fig. 28). However, there is now little doubt but that more than one kind of streptococcus has hitherto been included under this term. The staphylococcus found in pyæmia is usually the Staphylococcus pyogenes aureus (Fig. 29), and this is occasionally mixed with Staphylococcus pyogenes albus.

Clinical conditions which have a close resemblance to those of pyæmia are sometimes met with during gonorrhœa and typhoid fever. In these the metastatic suppurations contain the gonococcus, or the typhoid bacillus respectively. There is also the closest affinity betwixt pyæmia and acute osteomyelitis (acute suppurative periostitis, acute necrosis, acute panostitis). Indeed, these two diseases are but different manifestations of an infection with the same species of pyogenic cocci. Matters have become simpler since it has been recognised that a single kind of bacterium may produce various results, according to the mode of infection. For instance, the Staphylococcus pyogenes aureus rubbed into the skin causes furuncules (Garré); injected into the cellular tissue it causes an acute abscess (Bumm, Waterhouse); injected into the joints acute suppurative arthritis (Passet); injected beneath the periosteum, acute suppurative periostitis; injected into the serous cavities it causes pleuritis, pericarditis, meningitis, or peritonitis. Conversely, Staphylococcus pyogenes aureus from any of the above affections will, when introduced into the circulation, cause pyæmia. It is probable that the Streptococcus pyogenes and Staphylococcus pyogenes albus play as extensive a rôle as the Staphylococcus aureus.

There can be no doubt but that clinical differences are to be observed in different cases of pyæmia. This is possibly to some extent accounted for by differences in the bacteria that have caused the attack. For instance, it is beginning to be recognised that in streptococcus poisoning the joints are usually affected, whilst staphylococcus poisoning is especially prone to cause suppurative periostitis or osteomyelitis. Wagenmann found streptococci in the emboli which filled the vessels of the eye in puerperal pyæmia.

The circumstances of the infection.—Pyæmia is not, however, simply a matter of infection, as the foregoing might lead the reader to suppose. The mere presence of appropriate bacteria in a wound is, as a rule, not sufficient to cause pyæmia. A proportion of the wounds treated by the most careful antiseptic surgeons contain pyogenic cocci; and yet pyæmia may be unknown in their practice. Furthermore, the mere presence of Staphylococcus aureus or albus, or of Streptococcus pyogenes in the blood is not necessarily followed by pyæmia. Von Eiselsberg and Canon, in cases of ordinary wound fever and of sepsis, found Staphylococcus aureus and albus, and Streptococcus pyogenes in the blood. But as a rule the presence of any of these bacteria in the blood is associated with abscess-formation in some part of the body.

Before the pyogenic cocci can cause pyæmia, certain conditions have to be fulfilled. There are certain kinds of micrococci found in wounds which are, perhaps, capable of causing a local suppuration, but incapable of invading the blood. Many of those which inhabit the skin belong to this class. When the appropriate bacteria are present much depends upon (1) their *virulence;* upon (2) the *dose* of them which enters the blood; and upon (3) the *predisposition* of the recipient. (*See* page 16.)

(1) The variation which the *virulence* of different species of bacteria undergoes is now one of the commonplaces of bacteriology, and in many instances is easily controlled. Cultivated outside the body they become attenuated, whilst passage through an animal restores their potency. Pasteur uses anthrax attenuated by culture without the body for purposes of vaccination. In similar manner the Streptococcus pyogenes soon loses its virulence and dies out when grown in artificial media. The Staphylococcus aureus and albus are more resistant, but even the former after awhile becomes of a paler gold, grows more slowly, and ceases to cause suppuration or general infection.

(2) The importance of the *dose* of bacteria is also exemplified by experimental pathology. Everyone who has studied the infective diseases is aware that it is necessary to introduce a considerable quantity of cocci into the venous system to cause a general pyæmic infection. The healthy organism is able to deal with moderate doses without the occurrence of abscesses in any of the organs. The experiments narrated by Watson Cheyne in his lectures on suppuration and septic diseases show the importance of the dose in a definite manner. 1,000,000,000 of Staphylococcus aureus were required to cause the death of an animal, while 250,000,000 only produced a small circumscribed abscess. It seems probable that Staphylococcus pyogenes aureus and albus and Streptococcus pyogenes are more virulent for man than for animals. It is hardly necessary to point out that fewer virulent bacteria are required to produce results, and more of those which are attenuated.

(3) In pyæmia, as in other diseases, *predisposition* and immunity are both topics that are shrouded in considerable mystery.

We will consider first the question of *general predisposition.* It is a matter of common observation that certain races and individuals are more predisposed to some kind of infection than others. These peculiarities have only been ascertained for certain kinds of infection, of which anthrax is one of the best known. Even here immunity seems to be capriciously bestowed. It is notorious that French sheep die of anthrax, whilst the Algerian are immune. But natural immunity is not absolute. Pasteur found that fowls, which are refractory to anthrax, soon died of it, if, before or after inoculation, their temperature was lowered by placing them in cold water. Similarly frogs, which are naturally immune against anthrax, were rendered susceptible by immersion in warm water. Watson Cheyne also ascertained that natural immunity against bacteria could be overcome by phosphorus, a drug which has a well-known deleterious action upon the protoplasm of the body. There is reason to suppose that the toxins and ptomaines manufactured by bacteria may, when absorbed, predispose to the action of the bacteria themselves (Grawitz and de Bary, Rinne).

On the other hand, Behring claims that he has rendered animals immune against virulent Streptococcus longus by injecting into them the blood of animals that had been rendered immune by repeated inoculations with attenuated cultures of streptococci. This is the same method as that by which Tizzoni and Cattani produced immunity against tetanus. It throws an interesting light upon the comparative safety which demonstrators of anatomy and pathologists enjoy from pyæmia and septicæmia; also upon the rapid healing after amputations for prolonged suppurative arthritis.

It may be inferred that influences similar to the above predispose man to pyæmia. It is recognised that it is more apt to occur after starvation, debility, alcoholism, anæmia, severe hæmorrhage, prolonged shock, and wasting diseases, such as albuminuria and diabetes. Parturition, too, is often accompanied with local and general conditions which predispose to pyæmia. Unhealthy environments may also predispose to pyæmia, and thus it is rife in hospitals or dwellings which are ill-drained, ill-ventilated, or overcrowded. Obviously, most of this class of conditions are also such as would enhance the dangers of actual bacterial infection. Even where they prevail the liability to pyæmia is immensely diminished by antisepsis (Von Nussbaum).

Certain *local predisposing causes* influence the occurrence of pyæmia. Wounds in certain positions are more exposed to infection than others. Such, for instance, as those about the mouth, air-passages, alimentary tract, anus, scrotum, axilla, or scalp. Also wounds which are the result of accident are more likely to be infected than those which surgeons inflict. But after bacteria have been introduced, the local predisposing conditions must be such as (*a*) favour their multiplication in the wound; and (*b*) favour their passage into the circulation.

The local conditions that favour the multiplication of the bacteria which cause pyæmia are the same as those which favour sepsis and suppuration. (*See* page 81.)

Pathology and morbid anatomy. 1. **The condition of the wound.**—In clean-cut wounds in healthy tissues infection makes but little headway, but flourishes in such as are lacerated, bruised, infiltrated with blood, deprived of their blood or nerve-supply, or injured with chemicals. Foreign bodies and blood-clots in the depths of wounds are also suitable nidus for bacteria to grow in, and collections of fluid are especially favourable. Such infected wounds have no granulations or pus, and are usually in a state of acute septic inflammation. The wounds associated with pyæmia usually throb and are painful, hot, swollen, red, with acute lymphangitis and adenitis of neighbouring lymphatic glands; they often gape and display a dry and glazed surface, covered in places with ashen lymph; they may secrete a purulent, sanious, or stinking discharge, which often accumulates in the depths, and ulceration, sloughing, or gangrene may be in progress. Not infrequently there are tenderness and induration along the veins running from the wound towards the heart. When the bones are involved there are pain and tenderness, and, perhaps, thickening along them, and other evidence of acute osteomyelitis.

Pyæmia sometimes occurs when the external appearances of the wound are quite favourable. But I have never seen a case of this kind in which there was not a septic focus in the depths of the wound, with evidence of septic phlebitis or of osteomyelitis.

In the secretions of wounds that are complicated with pyæmia, bacteria of various kinds can easily be seen in cover-glass preparations, and their different kinds can be ascertained and separated by plate cultures. If the secretions of the wound contain nothing but the ordinary pyogenic cocci they are almost odourless, but very often bacilli (the various kinds of Proteus of Hauser) are likewise present, and produce stinking substances. In a case that died of pyæmia after internal urethrotomy, the writer found bacilli and cocci in the wound, which was exceedingly putrid, but only streptococci in the secondary abscesses. In another case of pyæmia following internal urethrotomy, Dr. Prudden found Staphylococcus pyogenes aureus in the blood and internal organs. The presence of other bacteria along with the pyogenic cocci in cases of pyæmia helps to explain anomalous varieties which are met with clinically, and which have been called septico-pyæmia or pyo-septhæmia (Gussenbauer).

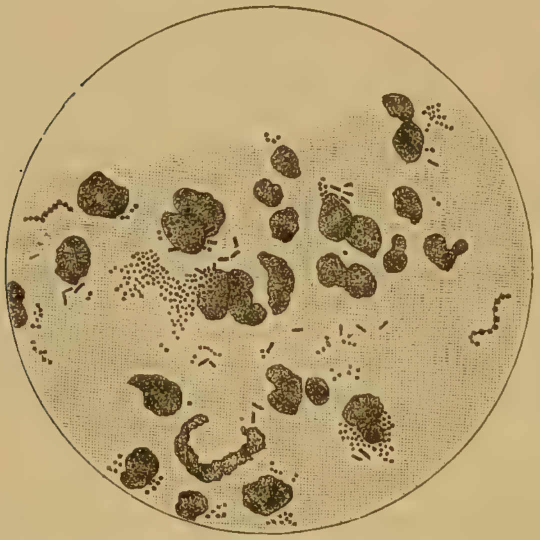

Fig. 30.—Section of the Tissues of a Wound which caused Pyæmia. (From a micro-photograph by Mr. Cosens.)

In the lymph and amongst the pus corpuscles which cover the surface of such septic wounds are various kinds of micrococci. In the specimen shown in Fig. 30, cocci, diplococci, staphylococci, and streptococci could all be discerned, as well as numerous bacilli. The patient died of pyæmia after amputation of the breast.

The tissues around the wound when microscopically examined are œdematous and hyperæmic, and infiltrated with purulent materials.

2. **The condition of the vessels.**—The *lymph paths* in the vicinity of the lesion are choked with pus corpuscles, amongst which are many bacteria, especially streptococci. It is easy to understand that chains of cocci are more adapted to pass along narrow lymph paths than the more bulky masses of staphylococci. The lymphatics, however, are not the main channel by which the cocci invade the blood. As is well known, the lymphatic glands act as filters and prevent their onward progress. The *veins*, on the other hand, play a most important part in pyæmia, and are

the channels by which the blood is usually invaded. The smaller veins near the seat of infection are filled with thrombi, which consist of fibrin, and of white and red blood corpuscles (many of which are disintegrating), and of bacteria. Their endothelial lining is in a state of proliferation, and the surrounding tissues contain many leucocytes. When *septic phlebitis* affects the larger veins, they are distended and of a dark purple colour. Their adventitia may be inflamed and stained with blood pigment, and sometimes abscesses are found along their course. Their walls are thickened and their interior is rough, denuded of endothelium and coated with fibrin. The *septic or infected thrombus* usually begins at the wound, or infecting focus, and extends as far as the nearest valve, or into the nearest great trunk. Here its softened end may float in the blood-stream, so that portions become detached, giving origin to those *septic emboli* which are characteristic of pyæmia.

The clotting of the blood in the veins is one of the consequences of the septic phlebitis. As the latter spreads along the veins, so the clot increases in size. But infective inflammations may also attack healthy veins in which clots have previously formed. It is probable that such clots in the proximity of septic wounds, by the ease with which they become infected, facilitate the entrance of the bacteria. When clots become infected the usual processes of organisation cease, and they soften and disintegrate. Bacteria act upon blood clots within the body in the same manner as they do upon culture media of gelatine or blood-serum. They rapidly peptonise these soft solids and convert them into fluids.

The close relationship of acute septic phlebitis to pyæmia explains the dread which surgeons once had of operations upon, or injuries to, the veins. Septic phlebitis and pyæmia were common after injuries to bones, especially those of the head, in which the veins are capacious, destitute of valves, and incapable of collapsing. Pyæmia is a frequent complication of septic phlebitis of the lateral sinus from suppurative otitis media, and of injuries to the great veins about the neck of the bladder and prostate.

Misled by the appearances of these softened and decolorised clots, John Hunter, and afterwards Cruveilhier, were led to speak of phlegmonous or suppurative phlebitis. In this the so-called pus—in reality the septic and softened clot—was supposed to be secreted by the wall of the vein. Cruveilhier further supposed that this pus was carried away in the blood-stream, and deposited elsewhere to form the metastatic abscesses. The extraordinary rapidity with which pyæmic abscesses form was naturally thought to confirm these ideas. The existence of such a disease as suppurative phlebitis was disproved by Arnott and Dance.

3. **The infarcts and abscesses.**—Virchow threw a new light upon the pathology of pyæmia when he showed that portions of the softened and septic clots became detached, and carried as septic emboli in the blood-stream to the heart, and thence onwards into the pulmonary vessels. Here these septic emboli become arrested in

the smaller arterioles near the edge of the lung, or at the bifurcation of arteries. When once arrested, they excite two kinds of changes in the wedge of lung supplied by the vessel which they plug. The first change is more or less mechanical, and has to do with the interruption in the circulation. It ends in the wedge of lung supplied by the artery becoming filled with blood clot. The exact way in which this occurs is not well understood, but is supposed to be due to regurgitation of blood from the veins. The result is called an "*infarct*," and the process "*infarction*." Thus, whilst the clot is in a vein it is called a venous thrombus, when detached an embolus, and when lodged in a vessel and accompanied with vascular changes it helps to form a hæmorrhagic infarct. Wedge-shaped infarcts are found in the spleen and kidneys as well as in the lungs.

The second set of changes which the septic emboli excite are dependent upon the bacteria that they carry with them. These changes are the same as those which the bacteria cause elsewhere, and are an acute septic inflammation ending in suppuration. It is this final stage which constitutes the *pyæmic abscess.* Not infrequently the suppuration obscures the preceding hæmorrhagic infarction, and doubtless the latter often does not occur at all (Hamilton). In this embolic process the blood clot itself acts a mere passive part in the causation of the pyæmic abscesses, and is by no means essential for their formation.

Since the work of Recklinghausen, Klebs, Hueter, and others the origin of the abscesses in the liver, kidneys, and elsewhere has been clearly understood. The pyogenic cocci circulating in the blood become arrested in the smaller capillaries and vessels and form bacterial emboli, and excite inflammatory changes that end in suppuration. The changes following the arrest of the cocci are, as Weigert and Cheyne have shown, somewhat as follows :—The tissues around the cocci undergo coagulation necrosis, and refuse to stain with ordinary aniline dyes. Beyond this ring of coagulation necrosis is a dense accumulation of leucocytes, which are the beginning of the abscess; beyond these there is acutely inflamed or engorged tissues.

The minute micrococci, which are circulating with the blood in pyæmia, find no obstacle to their passage through such relatively coarse capillaries as those of the lungs. Thus we find that bacterial emboli and pyæmic abscesses may occur in any region of the body to which the blood penetrates. They are commonest in organs and tissues that have the largest supply of blood and the most entensive capillaries, as, for instance, the lungs, liver, kidneys, spleen, brain, muscles, serous and synovial membranes, in the heart, thyroid body, parotid gland, testis, and eye. (*See* page 181.) Until the bacterial origin of pyæmia was understood pathologists found it hard to conceive how emboli, originating in a systemic vein, could elude the capillaries of the lungs, and cause an abscess in an organ such, for instance, as the liver. The perplexity was also increased by the occasional presence of clots in the neighbourhood of the pyæmic abscesses. These, of course, are formed *in loco*, and the mystery is solved now that pyæmia is known to be due to bacteria, which are so small as to circulate everywhere almost without hindrance, and that it can be produced experimentally by the intravenous injection of pure cultures of pyogenic cocci.

Although septic thrombi and emboli are not essential in pyæmia, there is evidence to prove that they are important factors. Pyæmia is caused with much greater ease in animals if, in addition to pyogenic cocci, particles of some inert material are injected into the veins at the same time. Pawlowsky used sterilised cinnabar and Staphylococcus aureus, and produced typical pyæmia; Ribbert injected particles of the potato upon which S. aureus had been grown; Bonomé used elder pith, and so forth. It seems probable that the clot or foreign body acts as a nidus in which the bacteria are able to localise themselves, and multiply without being washed away by the blood stream. Healthy animals can get rid, without much apparent harm, of considerable doses of pyogenic cocci if they be gradually introduced into the circulation in suspension in distilled water, salt solution, or even in filtered or dialysed pus. Besides blood clots, probably many other factors determine the arrest of circulating bacteria in particular places.

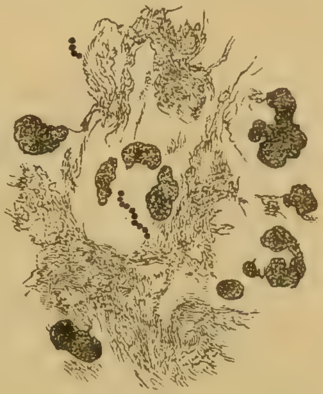

Fig. 31.—Streptococci in Pus from a Pyæmic Knee. (From a micro-photograph by Mr. Cosens.)

4. **The localising of the bacteria.**—Huber found that when he inflamed a rabbit's ear with blistering fluid, and then inoculated the animal with anthrax, the bacilli accumulated in the inflamed area. The effects of traumatism in causing the localisation of circulating bacteria are well known. Rosenbach, Kocher and others have caused osteomyelitis by injuring the bone either before or after the intravenous injection of Staphylococcus aureus. Here the tearing of the vessels, the blood clots, and the lacerated tissues are all conducive to the entanglement and arrest of bacteria. To some extent similar influences are at work in causing the localisation of the bacteria that cause pyæmic abscesses.

In pyæmia the micrococci sometimes localise themselves upon the valves of the heart and cause *ulcerative endocarditis*. This is indicated by the usual cardiac murmurs and clinical signs of valvular disease. Wyssokowitsch caused ulcerative endocarditis by first injuring the valves of the heart, and afterwards injecting pyogenic cocci into the veins.

5. **The character and situation of the pyæmic abscess.**—Pyæmic abscesses are almost always small and multiple, but now and then solitary abscesses of some size are met with. In a case of this kind I found, in addition to the abscess, numbers of bacterial emboli in the liver and kidneys. The pus in the larger abscesses is

thin and watery, with shreds of necrosed tissues in it, and with a slight characteristic odour, which is also smelt when pyogenic cocci are grown by themselves in culture media. The pus contains vast numbers of staphylococci and of streptococci. The largest accumulations of pus are found in the joints or serous cavities, and usually contain streptococci. Schüller found streptococci in twelve cases of pyæmic arthritis following upon child-birth. The pus shown in Fig. 31 came from the knee-joint of a youth who suffered from pyæmia after a printing-machine accident. The hip and ankle of the same leg were also affected. Streptococci were also found in the urine (Fig. 34). Pyæmic abscesses have no distinct walls, but are surrounded by acutely-inflamed tissues which are infiltrated with pus cells, and with streptococci or staphylococci. The accompanying figure was drawn from a part of the wall of a pyæmic abscess that followed a burn (Fig. 32).

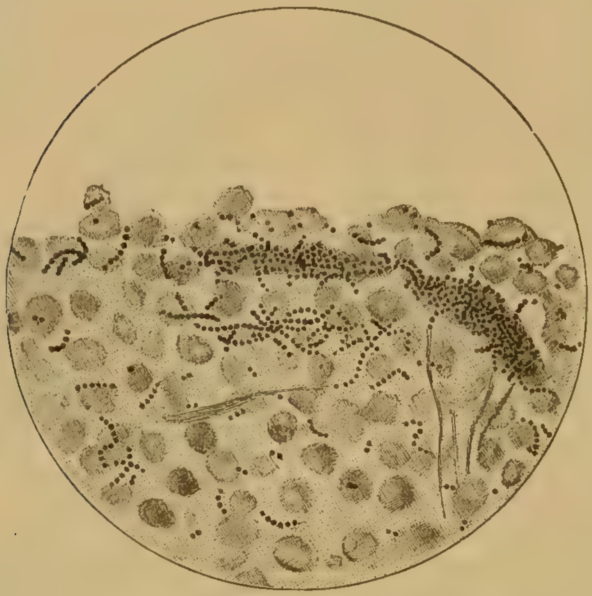

Fig. 32.—Section of the Wall of a Pyæmic Abscess, stained by Gram's method. (From a drawing by C. B. Lockwood.)

The *position* of pyæmic abscesses depends, as we have seen, to some extent upon the kind of lesion in which the disease originated. In the Pathological Society's Report on Septic Diseases (vol. xxx., 1879) a hundred and ten cases of pyæmia are recorded. Abscesses occurred in the lungs in fifty-seven of these, in the joints in twenty, in the liver in twelve, in the spleen in eleven, and in the kidneys in six. The above refers, of course, to gross lesions which could easily be seen with the naked eye. Makins and Abbott have tabulated two hundred cases of pyæmia in which the infection was introduced in all kinds of ways. These figures give a slightly different result. The joints were affected in seventy-six cases, the lungs in sixty-six, the lungs and pleura in seventeen, the pleura in sixteen, the liver in ten, the spleen in twenty-two, and the kidneys in seventeen. Abscesses occurred in the areolar tissues in twelve of the Pathological Society's cases, and in sixty-nine of those of Makins and Abbott.

In pyæmia associated with acute infective osteomyelitis, necrosis, and acute suppurative periostitis, acute suppurations are common in the heart and pericardium. In Makins and Abbott's tables the pericardium was affected in 36·5 per cent., and the myocardium in 26·8. In pyæmia due to other causes the proportion was only 6·5 per cent. for the pericardium, and 2 per cent. for the myocardium. Mr. Stephen Paget says that in eighteen cases of acute necrosis, ten had abscess of the heart, and eleven abscess or infarction of the kidney. Out of one hundred and forty cases of pyæmia after amputation, one had abscess of the heart, and one abscess of the kidney. Mr. Paget has also shown that parotitis is common after injuries to the abdomen and pelvis, and is often pyæmic in origin.

It is an observation as old as Hippocrates that after injuries of the cranium and its venous sinuses pyæmic abscesses are formed in the liver. Out of two hundred and seventeen cases of pyæmia after injuries involving the medulla of bone, fifty-six had abscess of the liver and twenty-eight had abscess of the spleen. In sixty-seven cases of pyæmia after injury of the soft parts, four had abscess of the liver and three had abscess of the spleen. Dr. Wilks believes that the salivary glands are especially affected in the pyæmia which follows typhoid fever, and the joints in that which follows scarlet fever. When pyæmia follows septic phlebitis of the portal veins, the pyæmic abscesses are in the liver.

6. **The post-mortem appearances.**—The body is emaciated and the skin of a jaundice hue. Rigor mortis is slight; bed-sores are often present. A septic focus such as has been described will be found, with the neighbouring veins in a state of septic thrombosis. The lymphatics may be acutely inflamed, and the lymphatic glands swollen or suppurating. Occasionally the seat of infection is hard to find, but its absence cannot be assumed unless every part of the body has been examined, especially the ear, alimentary canal, and genito-urinary system. The blood is dark, thin, and watery, with little tendency to clot, and prone to decompose. Its red corpuscles are diminished and the white increased (leucocytosis—Birsch-Hirschfeld).

The *valves of the heart*, especially those of the left side, may be inflamed and ulcerated. The inflammation may affect a large extent of the endocardium. The *serous cavities* may contain serous, blood-stained, purulent or fœtid fluid, and the surface of the heart, lungs, or abdominal viscera may be covered with lymph. On mobile organs like the lungs or heart this is often rough. Cerebral or spinal meningitis may be found.

The viscera are usually engorged with dark blood, especially the spleen, kidneys, and lungs. The changes found are as follows:—

The *lungs* are usually engorged with blood, especially towards their bases, and often have areas of broncho-pneumonia. Beneath the pleura a number of small deep-red or opaque white elevations can be seen and felt. Their size is variable, but the largest may have a diameter of an inch. An incision shows that the deep-red patches are wedge-shaped hæmorrhagic infarcts. The base of the wedge is towards the surface. They are devoid of air, solid, and sink in water, and look like damson cheese; the expression "pulmonary apoplexy" is applied to lung in this condition. The remains of a septic embolus may be found in a pulmonary vessel at the apex of the wedge. The lung tissue around the infarct is inflamed, and the whole has undergone changes which end in abscess.

In the opaque whitish patches the various stages of an acute broncho-pneumonia ending in suppuration may be traced. In the centre is thin pus, then a zone of more solid greyish white lung, and next a zone of acute broncho-pneumonia. The pleura over these infarcts and abscesses is sometimes inflamed; the pleural sac may contain pus or blood-stained fluid, and its surface may be covered with rough lymph full of streptococci or staphylococci.

Infarcts and abscesses are commonest at the surface of the lungs and around the interlobular fissures. Small miliary abscesses are frequently found in the substance of the lungs.

When pyæmic abscesses occur in the *liver* they are nearly always small and multiple, and scattered over its surface. Tropical abscesses, on the other hand, are usually single and of large size. Dr. Wilks is of opinion that pyæmic abscesses are commonest at the back of the right lobe of the liver, which, as he points out, is the most dependent part when the patient is on his back. In pyæmia abscesses seldom occur in the liver alone but are met with together with abscesses in the lungs, joints, muscles, and other organs.

The *spleen* is usually enlarged to nearly a half more than its usual size. It is dark purple and studded with infarcts and abscesses, similar to those in the lungs.

The *kidneys* are swollen, mottled grey and red, and engorged with blood, so that the stellate veins, glomeruli, and vessels of the medulla stand out as if injected. Small hæmorrhages are common in the cortex. Towards the capsule and at the junction of the cortex and medulla are round or linear patches of suppuration, surrounded by zones of acute nephritis. Pus is sometimes met with in the interior of the kidney. Disseminated suppurative nephritis may be extensive without presenting much change to the naked eye (Ziegler). In this the vessels are dilated, and some of the capillaries filled with micrococci. Around the glomeruli and smaller veins numbers of leucocytes are extravasated, and are the beginning of abscesses (Fig. 33). The renal epithelium is granular, degenerated, and detached. The lumen of the tubules contains granular débris, albuminous coagulum, leucocytes, and often bacteria. Extravasation of red corpuscles is also found between and within the tubules.

The suppurations which occur in the cellular tissues, muscles, bones, brain, testes, ovaries, eyes, and elsewhere call for no special description.

In *pyæmic arthritis* the joint cavity is distended with thin pus, which usually contains streptococci. (*See* Fig. 31.) The synovial membrane is swollen and inflamed. The articular cartilages are ulcerated, especially round their edges. In advanced cases the bones may be carious, and periarticular abscesses may form. As a rule several joints are affected in pyæmic arthritis; the knee, elbow.

shoulder, ankle, wrist, hip, and sterno-clavicular are those usually involved, the knee the most often, and then the others in the order in which they are placed.

The clinical phenomena of pyæmia.—The clinical history of acute pyæmia accords with the account given of its pathology.

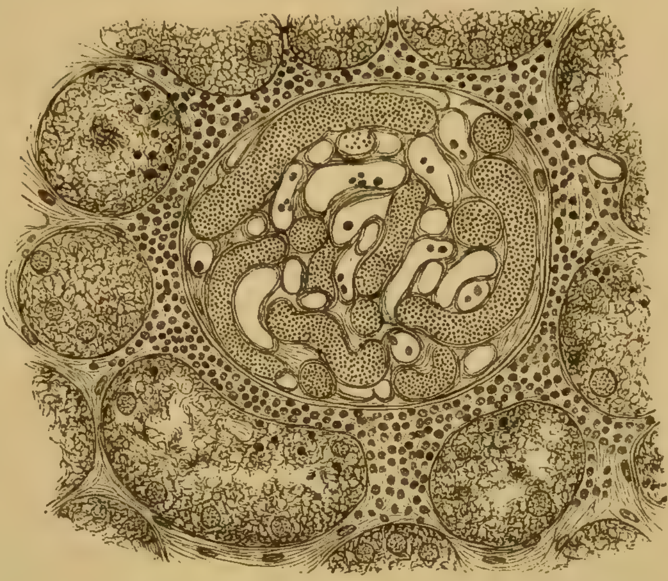

Fig. 33.—Acute Suppurative Nephritis in Pyæmia. Glomerular vessels full of micrococci, with surrounding infiltration of leucocytes. (After Ziegler.)

When the infective material which causes an acute pyæmia is introduced into a fresh wound an interval, which is in reality *a period of incubation*, elapses before the general symptoms begin. During this interval the shock of the injury disappears, reaction is established, and the bacteria multiply in the wound. The temperature of reaction, however, seldom falls to normal, but maintains the rise which marks traumatic fever. At the same time the pulse and respiration are accelerated. After this interval the symptoms, which are pathognomonic of pyæmia, may be expected to begin. A violent *rigor*, which shakes the bed, occurs, and whilst it is at its height the temperature rapidly increases, and may reach some unusual degree—say 106° to 107° F. The rigor is followed by profuse sweating, which makes the bedclothes wringing wet, and the temperature falls as rapidly as it rose, but seldom falls to normal. During the rigor the pulse and respiration are accelerated, and the patient is greatly distressed.

In pyæmia the rigors recur at irregular intervals. The irregular and rapid alterations in the temperature, with the accompanying rigors, are typical of pyæmia, and help to distinguish it from ague, malaria, acute rheumatism, typhoid fever, and septicæmia. In the last there is usually a single rigor at the commencement, and it is not repeated. In pyæmia a continuance of the rigors renders the prognosis hopeless. A single rigor soon after an operation has no particular import. It may merely be due to cold and exposure, or to the severity of the operation, and will not recur after reaction is established. Rigors, too, are comparatively common after operations upon the urethra, prostate, or bladder, and have no especial gravity if they are early and unaccompanied with other signs of septic infection. The rigors which characterise ague or tropical fevers are also precipitated by operations, and might be a source of alarm if their origin were unsuspected.

The symptoms attending *pyæmic abscesses* vary greatly. A rigor usually marks the occurrence of a fresh infarction or of a new purulent deposit. These may be painless, but as a rule they cause a sudden and acute stabbing pain. In the lungs this is accompanied with shortness of breath, the formation of an area of dulness, and the expectoration of blood-stained or rusty sputum; in the liver by swelling of the organ and by jaundice; in the spleen by enlargement; in the kidneys by albuminuria, sometimes by hæmaturia, and by bacteria in the urine; in the eye by pain and blindness and increased tension; in the brain by delirium, coma, and cerebral symptoms; in the heart by rapid and irregular action and endocardial murmurs. Abscesses which form in accessible positions appear with great rapidity, and are accompanied with the usual signs, as are also empyema, pyo-pericardium, and suppurative peritonitis. The joint suppurations which complicate pyæmia simulate acute rheumatism. As several joints may be involved, and as they have all the clinical features of acute arthritis, the mistake is easily made. However, doubt can easily and safely be set at rest by aseptic puncture with an aspirator should the other clinical signs not point to a correct diagnosis.

In some instances no local symptoms attend the filling of the joint with pus.

In pyæmia the *patient's aspect* betokens the gravity of the disease. The complexion is pale or jaundiced, the eyes are sunken, the cheeks are hollow, and the lips and tongue dry or covered with aphthæ, ulcers, or sordes. Wasting of the tissues is rapid, and bed-sores soon form. The mind is full of apprehension and very despondent. Delirium is common. The bowels may be confined, but towards the end diarrhœa is not infrequent. The urine is scanty, dark-coloured, and of high specific gravity. The breath and exhalations are supposed to have a peculiar sweet odour in pyæmia. They merely smell the same as old cultures of pyogenic cocci.

As we have already seen, *the wound* is, as a rule, in a state of acute septic inflammation. It is painful, throbbing, hot, swollen, red, with inflammation of the nearest *lymphatic vessels and glands*. The *veins* may be tender to the touch, hard, and plugged with clot. The discharges, the blood, and the urine should be examined bacteriologically. The urine should be obtained fresh in a sterilised test-tube. A little of it boiled with acetic acid gives a trace of albumen. Cover-glass preparations of the sediment yield cocci or streptococci. I believe this to be an almost constant sign if properly sought for. The accompanying specimen was obtained from the urine of a youth whose leg was amputated for a machine accident (Fig. 34). He also had streptococci in the pus in his joints (Fig. 31).

Recent investigations (Sherrington) tend to show that the bacteria do not escape until the kidneys have been injured by their presence; or, in other words, that normal kidneys do not excrete bacteria. I have always found acute disseminated nephritis and often abscesses when the pyæmic urine has contained cocci.

During pyæmia various *skin eruptions* occur. These in the order of their frequency are erythema migrans, purpura, papules, pustules, vesico-pustules, urticaria, milium, and sudamina. The erythema may resemble erythema nodosum or be like German measles. Recently I have seen a case in which it was mistaken for scarlatina. Makins and Abbott mention a case of the same nature. The rashes of pyæmia are similar to those of septicæmia, of which Aulas mentions the following varieties: zona purpura, simple or papular erythema, urticaria, vesico-pustular, and miliary eruptions. Some of these are very rare, but in neither pyæmia nor in septicæmia are mistakes likely to occur if a thorough examination of the local and general symptoms be made.

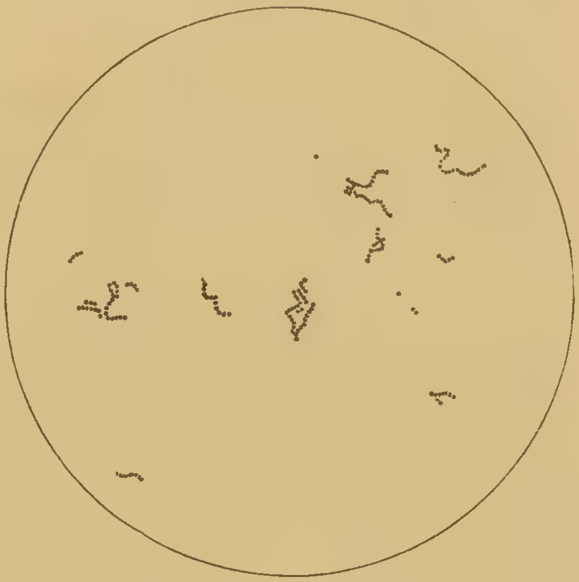

Fig. 34.—Streptococci in Pyæmic Urine. (From a microphotograph by Mr. Cosens.)

Prognosis.—Death from pyæmia is seldom delayed beyond the fortnight. The average duration of forty-one cases complicating acute infective osteomyelitis was twelve days. In a hundred and thirty-three cases of ordinary pyæmia it was 10·15 days (Makins and Abbott). Recovery from pyæmia is a slow and anxious process, liable at any time to be complicated with rigors, which portend recrudescence of the disease. Recovery only ensues when the pyæmia, which has had an acute beginning, has been chronic for a long period. The prognosis, therefore, is more favourable as time elapses. The average duration of four cases which recovered was one hundred and thirteen days, and in four others the duration was thirty-one days, one hundred and forty-three days, one hundred and ninety-three days, and eighty-six days respectively (Makins and Abbott). The prognosis is more favourable when the pyæmic abscesses attack the limbs, or are confined to the areolar planes. The prognosis is most unfavourable when the abdominal and thoracic viscera are affected. The only difference betwixt acute and chronic pyæmia consists in their duration and in the severity of the symptoms.

Treatment.—The treatment of pyæmia is preventive and curative. The *preventive treatment* of pyæmia mainly consists in the correct practice of antiseptic surgery. The only case of pyæmia which I have had was after an amputation for an acute osteomyelitis of the femur. In this it had undoubtedly arisen before the operation. Cases of suppuration of any kind are to be isolated as far as possible, and all who are in contact with them taught the dangers of the conveyance of infection from them. These remarks apply with greater force to cases of pyæmia.

Some of the dangers of infarction and embolism may be obviated by keeping the patient absolutely at rest. I have seen a fatality which seemed directly caused by neglect of this precaution.

The *curative treatment* of pyæmia is exceedingly unsatisfactory—drugs are of no avail. Full doses of quinine help to diminish the severity of the rigors. Every effort should be made to sustain the strength with concentrated and peptonised foods, and with alcohol. The latter may be administered in considerable quantities in the form of brandy, port, burgundy, or champagne. If pain be severe, anodynes may be given to relieve it and induce sleep. Abscesses should be opened early whenever they occur in accessible places. A pyæmic abscess is a new focus from which a fresh invasion of micrococci and of ptomaines may enter the blood. The most rigorous attempts should be made to open up and disinfect the focus of infection. When this is in the tympanum or mastoid antrum the internal jugular vein should also be exposed in the neck and ligature if there be evidence that there is a septic clot in the lateral sinus. Mr. Clutton has published a case of this kind which recovered, although abscesses had formed in some of the joints. When the pyæmia is due to acute infective osteomyelitis, amputation ought, in some cases, to be performed, especially if there be no evidence of abscesses or infarcts in internal organs.

The suppurating joints ought to be incised and drained. This is usually followed by relief, and does not necessarily end in ankylosis should the patient eventually recover. Should the suppurative arthritis have damaged the joint irretrievably it would be better to amputate the limb than to run the risks of prolonged suppuration. To prevent pathological dislocations and displacements of the joints, the limbs ought to be put in suitable splints and apparatus. Whatever incisions or operations are performed in pyæmia, should be done under the usual antiseptic precautions. Although the abscesses contain pyogenic cocci, yet they are capable of being infected with other bacteria.

In chronic pyæmia similar principles are to be followed. Abscesses should be opened, but there is less need of haste in performing amputation or incision. The further danger of damage to the kidneys, as evidenced by albuminuria, and of amyloid degeneration of them and other organs, has also to be reckoned with. In chronic pyæmia sea-air sometimes works wonders.

IX. TETANUS AND TETANY.

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Definition.—*Tetanus* (Greek, *τείνω*, I stretch; *τέτανος*, a stretching) is one of the traumatic infective diseases in which the poison, acting through the nervous system, causes violent, continuous, painful contractions of the voluntary muscles of the body.

The muscular spasms which are characteristic of the disease are of the *tonic* kind, being continuous; they are, however, liable to exacerbations marked by violent intermittent *clonic* contractions, followed by periods of comparative rest, though very seldom by complete relaxation. Special groups of muscles are affected in an almost constant order, and the disease runs a rapid and fatal course.

When muscular spasms begin gradually, persist evenly for some time, and then gradually subside, they are called "tonic spasms." But when the onset is sudden, the contractions violent and intermittent, with sudden cessation, they are called "clonic."

Ætiology.—Tetanus is now a comparatively rare disease in European hospitals. During sixteen years, from 1878 to 1893, only thirty-one cases of it were treated in St. Bartholomew's Hospital.

Climate.—In the tropics it is a common disease, especially among negro infants; indeed, all the dark races seem to possess a special proclivity to tetanus. Generally speaking, the inhabitants of warm countries suffer more than those of cold or temperate climates. Nevertheless, more cases occur in England in winter than in summer. Military surgeons have observed that those who were wounded during inclement weather were more likely to die of tetanus than those injured when the weather was salubrious.

Sex.—Tetanus is much commoner in males than in females. The proportion usually given is four to one. Out of thirty-one persons treated for tetanus in St. Bartholomew's Hospital, three were females, and twenty-eight men. The greater liability of males to injuries will go far to explain this disproportion, although the excess might be expected to have been reduced if the lesions incident to parturition be taken into consideration.

It has not been shown that females enjoy any especial immunity, nor is the mortality of the disease less high in that sex.

Constitutional condition.—This has little to do with the liability to tetanus, except that those who are young and debilitated are more easily affected by a given dose of the poison than those who are mature and strong. But not only the quantity of the dose, but also its quality, and the manner of its introduction, have to be taken into consideration.

Injury.—When accompanying a wound, tetanus is called "traumatic." In the absence of any obvious wound or puncture, it is called "idiopathic," but since the infective nature of the disease has been better known, and the point of inoculation has been more diligently sought for, idiopathic tetanus has become a rapidly-decreasing disease.

It may follow the most trivial injuries. The prick of a thorn or of a hypodermic needle, the bite of an insect, the impaction of a fish-bone in the fauces, a slight graze upon the pinna, the piercing the ears for ear-rings, circumcision, and a slight wound of the tongue, have all sufficed to inoculate the disease. Anomalous cases have been described by competent observers. In these tetanus has followed subcutaneous injuries, such as a contusion of the back (Morgagni), a dislocation of the finger (Richelot), the effort to prevent a fall (Verneuil), or the blow of a whip-lash upon the face, the skin being unbroken (Watson). The minute wound by which the inoculation may have occurred is often healed long before the symptoms of tetanus commence, and thus may easily have been overlooked. The records of cases prior to our knowledge of the infective nature of tetanus are not very trustworthy. As a rule, the word "idiopathic" merely means that a lesion has not been looked for or has not been found.

In traumatic tetanus the starting-point is most often a wound of one of the extremities. This is merely because wounds of the extremities are by far the most frequent. Tetanus may follow any kind of wound, but is commonest after those which are punctured, lacerated, or septic.

As actual examples may be mentioned three consecutive cases that were caused respectively by a stick driven into the face, a splinter of wood driven into the finger, and a rusty nail driven into the foot. In each instance the implement had been in the earth. Wounds and compound fractures caused by the wheels of vehicles are also apt to be followed by tetanus; also gunshot wounds, especially those which occur in war, and which do not have immediate and proper attention. Tetanus has occurred after wounds inflicted by the surgeon, and has been met with after all kinds of amputations, excisions, herniotomies, ligature of arteries, and even after trivial operations—such as tenotomy, ligature of hæmorrhoids, ligature of varicocele, or evulsion of toe-nails. All these accidents betoken want of care in antiseptic precautions. One surgeon is said to have lost ten major operations from tetanus before he discovered that his hæmostatic forceps were infected (Roswell Park). In the tropics tetanus is exceedingly liable to follow severance of the umbilical cord (trismus neonatorum). Mr. Mandy suggests that this is due to earth contamination, as all the nurses are field-labourers. Wounds that are followed by tetanus are, as a rule, septic. Suppuration, ulceration, sloughing, and phagedæna are frequently associated with the sepsis. An ulcer caused by an ill-fitting plaster of Paris case, which had been applied for tuberculous disease of the ankle, was the starting-point in a case in St. Bartholomew's Hospital, and foul and sloughing burns sufficed in two others. A dustman was inoculated through a chronic ulcer of the leg which had existed fifteen years, and two other men with chronic ulcers of the legs died with most acute tetanus. Evidently the wound need not be a recent one.

An account of the bacillus of tetanus will be found on page 44.

Period of incubation.—After inoculation the onset of tetanus is always preceded by a period of incubation, during which the patient may seem quite well, although careful inquiry usually elicits that indefinite symptoms, feverishness, epistaxis, or shivering have preceded the attack. No definite limit can be assigned to this period of incubation. In the Peninsular War, during which tetanus was very prevalent, it was never longer than the twenty-two days. In a child aged 4 years tetanus began on the fourth day, in a youth of 14 on the tenth day, and in a strong young man on the sixteenth. The period of incubation seems to be shorter in childhood, but the second week is the period during which the attack is most likely to occur. Those cases, in which the convulsions supervened directly after the injury, are probably spurious. Trismus neonatorum is popularly called ' nine-day fits," from the time after birth at which the disease usually commences. In such cases as those of burn, chronic ulcer, and septic wound it must obviously be very difficult to say exactly when the bacillus was introduced.

Degrees of tetanus.—Tetanus is called *acute* or *chronic*, according to the severity of the symptoms. In acute tetanus the incubation is, as a rule, short. The onset of the symptoms is quick, and they soon culminate and run a rapid course. The end is almost invariably fatal. In chronic tetanus the incubation is, as a rule, a long one. The symptoms appear gradually, are mild and prolonged, and gradually disappear. Recovery is comparatively frequent. However, this distinction of acute and chronic tetanus is decidedly artificial. Intermediate degrees of severity are frequently met with.

Symptoms of tetanus.—It has been already stated that tetanus is a disease of the nervous system, manifested by tonic spasms of the voluntary muscles. Various groups of voluntary muscles may be affected. Those supplied by the motor division of the fifth nerve, by the seventh, by the spinal accessory, and nerves of the cervical plexus are usually the first to suffer wherever the original injury may have been. Very often the muscles of the neck are the first to contract, so that the patient thinks he has got a stiff neck from sitting in a draught, or that he is suffering from a slight attack of rheumatism. The muscles of mastication are involved at an early stage. The contracted temporals and masseters stand out in bold relief, and the jaws are tightly clenched. This very constant symptom has given rise to the popular name of the disease—"lockjaw." The scientific designation of this symptom is *trismus*. At first the spasm is incomplete, and can be increased voluntarily; but it afterwards increases, and at times the muscular spasms clash the teeth violently together, causing them to fracture, and sometimes lacerating the tongue or cheeks. The symptoms may begin with difficulty in swallowing, as in the case of a man who cut the tip of his finger off with a pair of shears. This symptom is due to spasm of the muscles of the fauces and pharynx.

When the tetanic spasms affect the muscles of expression, a characteristic feature of the disease, the *risus sardonicus*, is produced. In this, as Sir Thomas Watson forcibly says, "The forehead is corrugated and the brow knit, the orbicular muscle of the eye rigid, the eyeball motionless and staring, the nostril spread, the corners of the mouth are drawn back, the set-teeth exposed, and all the features fixed in a ghastly grin." When the tetanic contractions seize the muscles of the trunk, those of the abdomen and back suffer most. The powerful extensors of the back may act with such violence that the trunk becomes rigid and is thrown from the bed with the back arched and the abdomen projected into the air, so that the patient is supported upon his head and his heels. This condition is called *opisthotonos*. The abdominal muscles are nearly always contracted in tetanus, and usually at an early stage. Their rigidity gives a peculiar board-like feeling to the abdomen, which is very characteristic. When violent clonic spasms supervene upon the tonic contraction of the abdominal muscles the trunk may be bent forwards, so that the chin touches the knees. This condition, which is the reverse of opisthotonos, is called *emprosthotonos*. The diaphragm and the other muscles of ordinary and of extraordinary respiration may all undergo spasmodic contractions, and respiration becomes impossible, the chest being held as if in a vice (Watson). Sometimes the body is bent to one side by the muscular contractions. This is one of the rarer manifestations, and is called *pleurosthotonos*. Should the muscular spasms balance one another, so that the body remains stiff and rigid like a statue, the condition is called *orthotonos*. The muscles of the limbs seldom escape. Their contractions are sometimes so violent that the muscles are torn across, and the thigh bone even has been broken. As a rule, the limbs are extended, with stiffness of the joints, those nearest the trunk being more stiff than the others. Oftentimes the hands and feet are quite unaffected although I have seen the hands tightly clenched. When many groups of muscles are implicated at once the whole body is convulsed. These tetanic convulsions precede the fatal ending.

In tetanus produced experimentally in animals, the spasms often begin in the groups of muscles nearest the point of inoculation.

A child under my care ran a stick into his face whilst playing in the garden. Four days afterwards the facial muscles began to twitch, and this was followed by trismus and opisthotonos. Similar instances are mentioned by Fagge and others. According to Rose, of Zürich, *facial paralysis* may accompany tetanus, especially when the original lesion is in the distribution of the facial nerve. In these circumstances an ascending neuritis is supposed to ensue, and to cause swelling with compression of the nerve within its bony canal.

The *tetanic muscles* feel as hard as stones, and their bellies stand out in knotted masses. They are the seat of horrible pain, comparable to that of violent cramp. Oftentimes their fibres rupture, causing bruising and extravasations of blood. Even such muscles as the psoas and rectus femoris have been found to be torn across. The affected muscles are usually painful when manipulated.

The *paroxysms of clonic spasm*, which supervene upon the tonic spasm, do not occur spontaneously at the beginning of the disease, but are usually excited by some trifling irritant acting through any of the senses. For instance, they may be caused by sounds, such as the banging of a door, the moving of furniture, or by loud voices. They are also excited by strong light, by vibrations, by joltings, by currents of air upon the skin or the contact of bed-clothes, by the patient's own efforts to eat or drink, or even by his emotions.

The difficulty in swallowing, and the dyspnœa caused by efforts to drink, simulate hydrophobia. Indeed, the resemblance is sometimes so close that a *tetanus hydrophobicus* has been described. In this the muscles of the face, as well as those of the larynx and pharynx, are especially involved. However, the resemblance betwixt tetanus and hydrophobia is very slight in ordinary cases.

From the above it will be noted that the spasms of tetanus are to no small extent reflex in their nature. A slight peripheral efferent stimulus, acting upon the over-irritable nerve-centres of the medulla oblongata and spinal cord, produces through the efferent nerves violent tonic and clonic spasms. The nature of the poison which causes the exalted reflex activity of the nerve-centres will be discussed later. It is to be noted that as the disease progresses the spasms become more spontaneous, and recur with augmented violence, frequency, and pain.

In tetanus the *general condition* suffers less than might be expected from the severity of the symptoms. The mind is clear, though full of apprehension. There may be slight delirium towards the close. Sleeplessness prevails, although patients have been known to fall asleep spontaneously, the muscles becoming soft and yielding for the time.

Verhoogen and Baert mention a *rash* like erysipelas which frequently precedes the acute symptoms. What was thought to have been an erysipelas preceded tetanus in the case of a youth who was in St. Bartholomew's Hospital with a septic and lacerated wound of the buttock.

Fever is not a necessary accompaniment of tetanus. The temperature is sometimes normal throughout an attack. As a rule, however, a moderate rise of temperature is met with, the thermometer registering 100° F. to 102° F. Just before death, as Wunderlich pointed out, hyperpyrexia is common. Thus a temperature of 110° F. has been seen before death, and immediately after has risen to 112° F.

As a rule, *profuse perspiration* accompanies the tetanic convulsions. The exudation may be sufficient to soak the garments of the patient and the bed-clothes. It has been supposed by some that this free secretion of sweat has a favourable influence upon the course of the disease, and, therefore, it has been promoted by vapour baths, and by the subcutaneous injection of pilocarpine. Both of these remedies have enjoyed some repute.

In tetanus the *pulse* only undergoes such alterations as might be expected to be caused by the violent exertions of the muscles. At first it is natural, and merely increases in frequency during the convulsions. When exhaustion supervenes, it beats feebly, and with uncountable rapidity. By some this last symptom has had importance attached to it, and it has been considered a sign of paralysis of the vagus. Many patients die from sudden cessation of the heart's beat. It is much disputed whether this is due to cardiac paralysis or to cardiac spasm.

Respiration is unembarrassed so long as its muscles are unaffected. It merely becomes deeper and quicker during the convulsions. But when the abdominal muscles become rigid, they cease to act in expiration, and as the muscles attached to the thorax become tetanic, the chest is held "as if in a vice," and dyspnœa becomes an urgent symptom. The diaphragm, without doubt, participates in the tetanic spasm, and its forcible contractions help to account both for the dyspnœa and for the pain which is so often complained of in the sternum and pit of the stomach. The fear of impending suffocation is one of the most painful features of this horrible disease.

The *spasm of the respiratory* muscles may be so extreme as to cause sudden death, and sometimes this occurs at a very early stage. It is also probable that the muscles of the glottis may be involved in the spasm, the patient becoming deeply cyanosed during the closure of that aperture. In the case of a man aged 27, who was inoculated through an ulcer caused by a plaster case, tracheotomy was performed without any relief of the dyspnœa and cyanosis.* But according to Bauer, the danger of dyspnœa is always obviated by the muscular relaxation spontaneously caused by the carbonic-acid poisoning.

The *urine* is usually normal in tetanus, although albuminuria and hæmaturia are said to have been observed. Both of these conditions are met with in other infective diseases. Perhaps a more careful record would show that they occur oftener than is suspected. When the sweating is very profuse, the quantity of urine is diminished, and inability to swallow liquids brings about the same result. Owing to spasm of the compressor urethræ and the rigidity of the abdominal muscles, there may be retention of urine. It is important, therefore, that the state of the bladder be always ascertained.

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* St. Bartholomew's Hospital Reports, 1884.

Obstinate constipation is constant in tetanus. The tongue is foul and the bowels refuse to act for days together. The constipation cannot be altogether explained by attributing it to the condition of the expiratory muscles or to spasm of the sphincter. It is seen at the beginning of an attack, and long before those muscles are involved. Ordinary mild purgatives are powerless to overcome it, and such drugs as croton oil do not always act, unless in full doses. The motions are of a dark colour, and sometimes abominably fœtid.

Diagnosis of tetanus.—The differential diagnosis of traumatic tetanus ought to offer few difficulties.

(1) *Trismus*, and even opisthotonos, are sometimes simulated by hysterical females. However, the other features of the disease are wanting, and an anæsthetic at once puts any doubt at rest. The impaction of a wisdom tooth, or inflammatory affection of the jaws, may also cause a spurious kind of trismus. The finger would promptly detect this occurrence. The retraction of the head is much alike both in tetanus and in cerebro-spinal meningitis. The cephalalgia, high temperature, vomiting, optic neuritis, paralysis, stupor and coma which accompany the latter disease suffice for its discrimination.

(2) It has been already mentioned that there is a resemblance betwixt tetanus and *hydrophobia;* but in hydrophobia there is no trismus, and in the intervals between the convulsions there is a complete absence of spasm.

(3) In *strychnia poisoning* the convulsions are tetanic in character, and there may be pronounced opisthotonos, with spasms of the muscles of respiration. The suddenness of the onset of the muscular spasms, the rapidity with which they become general, the absence of muscular rigidity during the intervals, the discovery of strychnia and brucia in the urine, the rapidity with which death or recovery occurs, and spasm of the muscles of the hands (Fagge), may all be adduced as aids to differential diagnosis.

(4) *Tetany* is dealt with on page 200.

Prognosis of tetanus.—The prognosis of tetanus is most unfavourable. At Guy's Hospital 72 cases were treated between 1825 and 1857, inclusive, and 60 of them died. At St. Bartholomew's Hospital 31 cases were treated between 1878 and 1893, inclusive, and of these 20 died. It is also to be noted that nearly all the cases which recover are either chronic or "idiopathic."

The so-called idiopathic tetanus seems less fatal and more chronic than traumatic tetanus. For obvious reasons the first class has hitherto been admitted into the medical wards, and I find that at St. Bartholomew's Hospital, out of eight such cases, five recovered;

step, and obtained the first cultures of tetanus bacilli uncontaminated with any of the bacteria of putrefaction or sepsis, with which they are almost invariably associated.

Tetanus bacillus.—This micro-organism (Fig. 35) has been described elsewhere (page 44), and it is only necessary to mention here those peculiarities which are of particular importance to surgeons. A feature of especial interest is its extreme abhorrence of free oxygen. It is an obligatory anaërobe, and the presence of air, and probably of oxygenated fluid like the blood, is inimical to its propagation. Thus it is prone to live and grow in wounds which are deep and punctured. A suitable absence of oxygen may also be brought about by the simultaneous growth of oxygen-loving bacteria, such as those which cause putrefaction. Thus the tetanus bacillus is favoured by sepsis. Indeed, Vaillard and Vincent go so far as to say that the presence of septic organisms is indispensable, and that apart from them the tetanus bacillus cannot form its toxins. This is much disputed. The next characteristic of the tetanus bacillus is its great resistance to the two most common forms of disinfection—namely, heat and chemicals. The spores and spore-bearing bacilli resist the temperature of 80° C. for an hour, but are killed by five minutes' stay in the steam steriliser at 100° C. (Kitasato).

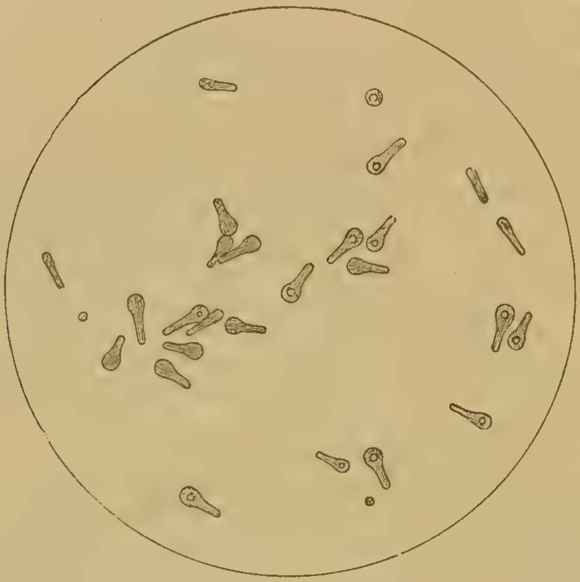

Fig. 35.—Bacillus of Tetanus. (After Fraenkel and Pfeiffer.)

Chemicals act somewhat feebly upon the bacillus of tetanus. Kitasato ascertained that they were still virulent after ten hours' immersion in 5 per cent. carbolic lotion, but that fifteen hours' killed them. A solution of perchloride of mercury, one part in one thousand parts of water, with 5 per cent. of hydrochloric acid, killed them in thirty minutes, although the same strength of perchloride without the acid took three hours.

Almost any surface soil or dust will produce the so-called "earth tetanus," and it has been caused by dust, mud, or soil from gardens, fields, roads, and buildings, especially churches. Further, anything which has been contaminated with such dust, mud, or soil becomes capable of transmitting the disease. Verneuil has attributed this to the admixture of horse-dung. The equine origin of tetanus has had much credence given to it in France. Of late years cases of tetanus have been recorded there as being of exceptional interest, because no contact with horses or horse-dung could be discovered. Horses and those who work amongst them are somewhat liable to tetanus. Poland relates—quoting Hurtel d'Arboval—that twenty-four horses were castrated on the same day, and afterwards led four times a day through a pond of water supplied by a very cold spring. Sixteen of them died from tetanus between the tenth and fifteenth day after the operation. In this brief account the cold spring is especially mentioned to support the "cold theory" of tetanus. Verneuil gives a great number of instances of the transmission of tetanus by veterinary tools, and amongst those who had had to do with horses. Park asserts that although tetanus is rare amongst sailors, yet when it does occur it is almost exclusively on board of ships used for the transportation of horses. The following is an example of earth tetanus in a human being:—A boy aged four years was under my care for a fatal attack of tetanus, which had been caused by a punctured wound of the face. Whilst playing in the garden he ran a piece of firewood into the subcutaneous tissue of the face. A drachm of the garden soil was placed in a pocket beneath the skin of a rabbit. It died of tetanus, as did also mice which were inoculated from the tenacious white lymph which formed where the earth had been inserted. It is recorded by those who took part in the last Stanley expedition that the natives poisoned their arrows with juices mixed with earth, and that many of those injured with them died of tetanus.

Within the body the comportment of the tetanus bacillus is not well understood. Its presence at the seat of inoculation is beyond doubt, and can be ascertained by inoculation experiments and by microscopical examination. Both the typical drumstick spore-bearing and non-spore-bearing bacilli are found in the pus or discharges from the wound. A cover-glass preparation stained with carbol fuchsin gives the best results. The tissues in the proximity of the wound are, at early stages, infective, and Beumer and others have caused tetanus with them; but, like other observers, I have failed to find the tetanus bacillus in the tissues of the wound.

Tetanus bacilli cling to splinters of wood or foreign bodies, such as are found in wounds. Many observers have caused tetanus with such splinters, and some have procured from them the bacilli. The tissues in the neighbourhood of these foreign bodies are highly infective.

It has been shown by very large numbers of inoculation experiments that the blood is not infective in tetanus.

After animals have been inoculated with pure cultures of tetanus bacilli, the microbes are found during the first twenty-four hours at the seat of inoculation and in its immediate vicinity; but they then disappear from all parts, except, perhaps, from the pus of the wound, and are said not to be found in any of the fluids or tissues of the body. Moreover, it is said that none of the fluids or tissues of animals experimentally inoculated are infectious, except the pus and, for a time, the recently inoculated tissues. However, it may be found that this assertion needs modification. Buedinger discovered that very small bits of the lymphatic glands in connection with the wound would cause tetanus when transplanted into rodents. Schnitzler has confirmed this observation, and found sporeless tetanus bacilli in the substance of the lymphatic glands.

Shakespeare induced tetanus in rabbits by subdural inoculation with an emulsion of the spinal cord or medulla of horses and mules that have died of that disease. I myself grew an impure culture of tetanus bacilli from the spinal cord of a man who had died of tetanus, and caused tetanus in mice by inoculating these impure cultures. Moreover, I was of opinion that tetanus bacilli could be seen upon the pia mater and arachnoid of the man's spinal cord. If it could be shown that the tetanus bacillus has its habitat within the body upon the pia mater and arachnoid, this would go far to explain the clinical features of the disease.

After its passage through a series of animals such as guineapigs the tetanic poison becomes attenuated as was shown by Bossano. This is one of the facts which bears upon the question of the varying severity of the disease.

Animals that have recovered from an attack of tetanus are for a long period protected against further inoculation, and it is probable that this immunity is produced in human beings. Tizzoni and Cattani have made the curious observation that in animals the immunity is transmissible from the mother to her offspring; possibly through her milk, which contains antitoxin.

Up to the present it has been thought that when the tetanus bacillus has been inoculated, it multiplies for a time at the point of inoculation. Here it manufactures that most potent toxin which Brieger has isolated and called *tetanin*, and which Faber is of opinion is of the nature of a diastase, as it is destroyed by a heat of 65° C. This toxin, whatever its exact nature may be, is supposed to become absorbed from the wound, and by its action upon the nervous system to produce the symptoms which have been described. Fermi and Celli say that this poison has no action when introduced by mouth or rectum, and is not acted upon by bile, pancreatic juice, saliva, or by bacteria of various kinds, but is destroyed by the gastric juice.

Treatment of tetanus.—The treatment of tetanus is local and general. With regard to the *local treatment*, the wound may contain infective pieces of wood, or glass, and therefore it ought always to be carefully searched. This procedure may require an anæsthetic. If small, the whole of the wound may be excised, although I have never seen any actual benefit result from this measure. In any case it should be thoroughly exposed and disinfected, and any damaged tissues removed. In addition to the ordinary disinfectants, such as have just been mentioned, a ten per cent. solution of peroxide of hydrogen may be used. When the wound is upon an extremity, amputation has frequently been performed. The results of this practice have, however, been too equivocal for it to have become a recognised mode of treatment. At St. Bartholomew's Hospital I am unaware of a limb having been sacrificed in the forlorn hope of curing an attack of tetanus. Success might ensue in a certain number of cases, because, as I have already shown, a fair proportion of the less severe end in recovery. A finger or toe might reasonably be sacrificed. It would also be reasonable to remove any obviously inflamed nerves or lymphatics. The division of nerve trunks or their stretching has hitherto given no trustworthy results.

The *general treatment* of tetanus affords as little satisfaction as the local. The remedies that have been used almost exhaust the pharmacopœia. The following is the routine which is usually adopted at St. Bartholomew's Hospital, and which has afforded fair results. The bowels are thoroughly moved with a minim of croton oil. For children a less violent purge suffices. Then the patient is kept under the influence of a mixture consisting of equal parts of bromide of potassium and of chloral hydrate. A usual dose is twenty grains of each every six hours. Chloroform is administered when the convulsions are violent and distressing, and may be increased or diminished as is found requisite. A light and nourishing fluid diet is administered. If trismus prevent the mouth from being opened, food may be given by a tube passed behind the teeth or through the nose. At the same time, as Verneuil recommends, the patient may be wrapped in cotton-wool, and isolated in a warm, silent, darkened room. Amongst the other remedies which have been given may be mentioned oxygen gas (from this I have seen a slight benefit), conium, belladonna, cannabis indica, opium, jaborandi, calabar bean, tobacco enemata, nicotine, and other remedies too numerous to mention, and all equally dubious in their action.

The *preventive treatment* of tetanus is fully attained by adherence to the tenets and practice of aseptic surgery. It is necessary to remember, however, that this bacillus, owing to the extreme vitality of its spores, offers great resistance to heat and chemicals. Instruments of any kind, and especially those which may have been used for a case of tetanus, should be boiled in washing soda and water for at least twenty minutes. All recent wounds into which earth, mud, or dust may have entered ought to be thoroughly explored and cleansed by the removal of fouled tissues, and thoroughly disinfected with solutions of chemicals, such as a solution of perchloride of mercury—one part in five hundred parts, with the addition of 5 per cent. of hydrochloric acid; or a lotion of carbolic acid, one part in ten. The wound should afterwards be dusted with iodoform powder and packed with iodoform gauze. An outbreak of tetanus in a hospital ought to be traced to its source, and thorough disinfection practised under the strict superintendence of a competent person. All the dressings and discharges from a tetanic patient should be burnt.

But the treatment of tetanus cannot be dismissed without alluding animals experimentally inoculated are infectious, except the pus and, for a time, the recently inoculated tissues. However, it may be found that this assertion needs modification. Buedinger discovered that very small bits of the lymphatic glands in connection with the wound would cause tetanus when transplanted into rodents. Schnitzler has confirmed this observation, and found sporeless tetanus bacilli in the substance of the lymphatic glands.

Shakespeare induced tetanus in rabbits by subdural inoculation with an emulsion of the spinal cord or medulla of horses and mules that have died of that disease. I myself grew an impure culture of tetanus bacilli from the spinal cord of a man who had died of tetanus, and caused tetanus in mice by inoculating these impure cultures. Moreover, I was of opinion that tetanus bacilli could be seen upon the pia mater and arachnoid of the man's spinal cord. If it could be shown that the tetanus bacillus has its habitat within the body upon the pia mater and arachnoid, this would go far to explain the clinical features of the disease.

After its passage through a series of animals such as guineapigs the tetanic poison becomes attenuated as was shown by Bossano. This is one of the facts which bears upon the question of the varying severity of the disease.

Animals that have recovered from an attack of tetanus are for a long period protected against further inoculation, and it is probable that this immunity is produced in human beings. Tizzoni and Cattani have made the curious observation that in animals the immunity is transmissible from the mother to her offspring; possibly through her milk, which contains antitoxin.

Up to the present it has been thought that when the tetanus bacillus has been inoculated, it multiplies for a time at the point of inoculation. Here it manufactures that most potent toxin which Brieger has isolated and called *tetanin*, and which Faber is of opinion is of the nature of a diastase, as it is destroyed by a heat of 65° C. This toxin, whatever its exact nature may be, is supposed to become absorbed from the wound, and by its action upon the nervous system to produce the symptoms which have been described. Fermi and Celli say that this poison has no action when introduced by mouth or rectum, and is not acted upon by bile, pancreatic juice, saliva, or by bacteria of various kinds, but is destroyed by the gastric juice.

Treatment of tetanus.—The treatment of tetanus is local and general. With regard to the *local treatment*, the wound may contain infective pieces of wood, or glass, and therefore it ought always to be carefully searched. This procedure may require an anæsthetic. If small, the whole of the wound may be excised, although I have never seen any actual benefit result from this measure. In any case it should be thoroughly exposed and disinfected, and any damaged tissues removed. In addition to the ordinary disinfectants, such as have just been mentioned, a ten per cent. solution of peroxide of hydrogen may be used. When the wound is upon an extremity, amputation has frequently been performed. The results of this practice have, however, been too equivocal for it to have become a recognised mode of treatment. At St. Bartholomew's Hospital I am unaware of a limb having been sacrificed in the forlorn hope of curing an attack of tetanus. Success might ensue in a certain number of cases, because, as I have already shown, a fair proportion of the less severe end in recovery. A finger or toe might reasonably be sacrificed. It would also be reasonable to remove any obviously inflamed nerves or lymphatics. The division of nerve trunks or their stretching has hitherto given no trustworthy results.

The *general treatment* of tetanus affords as little satisfaction as the local. The remedies that have been used almost exhaust the pharmacopœia. The following is the routine which is usually adopted at St. Bartholomew's Hospital, and which has afforded fair results. The bowels are thoroughly moved with a minim of croton oil. For children a less violent purge suffices. Then the patient is kept under the influence of a mixture consisting of equal parts of bromide of potassium and of chloral hydrate. A usual dose is twenty grains of each every six hours. Chloroform is administered when the convulsions are violent and distressing, and may be increased or diminished as is found requisite. A light and nourishing fluid diet is administered. If trismus prevent the mouth from being opened, food may be given by a tube passed behind the teeth or through the nose. At the same time, as Verneuil recommends, the patient may be wrapped in cotton-wool, and isolated in a warm, silent, darkened room. Amongst the other remedies which have been given may be mentioned oxygen gas (from this I have seen a slight benefit), conium, belladonna, cannabis indica, opium, jaborandi, calabar bean, tobacco enemata, nicotine, and other remedies too numerous to mention, and all equally dubious in their action.

The *preventive treatment* of tetanus is fully attained by adherence to the tenets and practice of aseptic surgery. It is necessary to remember, however, that this bacillus, owing to the extreme vitality of its spores, offers great resistance to heat and chemicals. Instruments of any kind, and especially those which may have been used for a case of tetanus, should be boiled in washing soda and water for at least twenty minutes. All recent wounds into which earth, mud, or dust may have entered ought to be thoroughly explored and cleansed by the removal of fouled tissues, and thoroughly disinfected with solutions of chemicals, such as a solution of perchloride of mercury—one part in five hundred parts, with the addition of 5 per cent. of hydrochloric acid; or a lotion of carbolic acid, one part in ten. The wound should afterwards be dusted with iodoform powder and packed with iodoform gauze. An outbreak of tetanus in a hospital ought to be traced to its source, and thorough disinfection practised under the strict superintendence of a competent person. All the dressings and discharges from a tetanic patient should be burnt.

But the treatment of tetanus cannot be dismissed without alluding to the more hopeful methods which we owe to the labours of Tizzoni and Cattani, and of Roux and Vaillard. Tizzoni and Cattani first ascertained that susceptible animals could be protected against tetanus and rendered immune by the injection of minimal doses of cultures attenuated by filtering, by heat, or by the addition of mineral acids. They have since discovered that this immunity cannot be obtained when the spleen has been previously removed. Next they found that the blood or blood-serum of an immunised animal—usually a dog or rabbit—rendered a filtered tetanus culture inert when mixed with it for twenty hours. The next step was to inject the serum into animals before inoculating them with tetanus. They were found to have been rendered immune. Then it was discovered that the serum also antagonised the tetanic poison when it was injected after inoculation, and even when tetanus had begun. The antitoxin was found to reside in the albuminous constituents of the serum. These can be precipitated by alcohol and dried in vacuo. In this form the tetanus antitoxin can be preserved like any other drug. At present it can only be obtained from Italy, or from M. Roux, of the Institute Pasteur, and from some chemists.

These brilliant investigations were soon applied to man, and with promising results. The cases which have now been reported are too numerous to mention separately. About eleven subcutaneous injections—each consisting of fifteen grains of the dried serum dissolved in one hundred and fifty grains of distilled water—are usually required. Clarke, of Leicester, who seems to have been the first to treat a case successfully in England, gave a hundred grains during the first twenty-four hours. In cases by Gattai, Morgagni, and Finotti, the treatment lasted a fortnight. Tizzoni and Cattani have also vaccinated horses against tetanus, and Lesi has used the serum of one of them to cure a man of acute tetanus. Kanthack, however, has pointed out that in the earlier cases treated with antitoxin, twelve out of thirty were fatal. Also that those which recovered were not of the worst kind. Also Escherich reports that out of four cases of trismus neonatorum treated with tetanus antitoxin, three died. Of late, however, the cure of acute cases has been reported, and it seems safe to say that no remedy which has yet been used has given such promise of ultimate success.

Tetany.—Tetany is a rare disease, which has sometimes been mistaken for tetanus. Very little is known as to its pathology. It is evidently an affection of the nervous system characterised by tonic spasm of the muscles, and has nothing to do with infection of any kind. The spasms are tonic in character, but liable to exacerbations. In severe cases the muscles may become as hard and rigid as in tetanus. Similar groups are also affected. The muscles of the face, larynx, eyes, and jaws, the sterno-mastoids, the pectorals, abdominal muscles, and muscles of the back, may all be rigidly contracted, and cause symptoms closely simulating tetanus. The muscular spasms may be accompanied by much pain, so that the patient cries out. The spasms may be produced or increased by mechanical or electrical stimuli, and by compression of the great arterial or venous trunks, or by tapping the motor nerves. The spasms of the facial muscles, caused by tapping the facial nerve, are said to be pathognomonic of tetany (Chvostek's sign), as is also the spasm of the limb-muscles caused by pressure upon the main vessels (Trousseau's sign). The slight attacks of tetany which are seen in children are often relieved by cold applications. In surgical practice, tetany is most often met with after complete removal of the thyroid gland, and is then exceedingly fatal in its effects. After partial extirpations it is almost unknown. Women are most prone to its attacks, but men are sometimes affected. Tetany occurred twelve times in fifty-three cases operated upon by Billroth, and of these twelve no less than eight died. The onset of tetany may be delayed for weeks, or it may occur directly after the operation. It may be fatal in a few hours, or last for many months. During its course, owing to the muscular spasms, the face may be distorted so as to assume a *risus sardonicus;* the eyes may squint; the jaws may be clenched, so as to interfere with speech, mastication, and deglutition; there may be dyspnœa, due to spasm of the larynx, or of the thoracic and abdominal muscles; and lastly, the limbs are flexed at nearly all their joints, and the hands are clenched, with the thumb flexed and adducted into the palm of the hand, or amongst the fingers. The temperature in tetany is usually elevated, but not immoderately.

After death from tetany no characteristic morbid change has been found.

Experimental pathology shows that tetany is common, and can easily be studied, after the removal of the thyroid gland of carnivora, such as cats and dogs. In addition to a notable fall of temperature, many of the animals treated in that manner show characteristic signs of tetany. There is reason to suppose that the symptoms may be abated by massive doses of thyroid juice, or of thyroid extract.

Hitherto *treatment* seems to have had little influence over this disease in the aggravated form in which it is sometimes met with in surgery. General measures directed towards the maintenance of the strength by nourishment and stimulants are indicated. Also chloral hydrate, bromide of potassium, or morphia to relieve the pain and spasms. Perhaps something may be ultimately hoped for from treatment with thyroid juice or extract.

X. WOUNDS AND CONTUSIONS.

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THE HEALING OF WOUNDS.

There are various ways in which wounds may heal, namely—

1. Healing by first intention;
2. By granulation;
3. By union of granulations;
4. By blood clot;
5. By scabbing.

1. Healing by first intention.—The simplest and, therefore, the first method to be considered, is healing by first intention. When a wound is inflicted, the knife, in passing through the tissue, sets up the early stages of inflammation in a microscopic layer of the living tissue in the immediate vicinity. This inflammation, as can be readily seen in a frog's foot, does not extend more than a line or two beyond the cut surface, but the extent will vary according to the degree to which the tissues have been injured by manipulation, sponging, etc. The inflammation so set up goes on to the first stage, namely, in the first instance there is dilatation of the blood-vessels and an increased flow of blood, followed by slowing, and ultimately stasis; while at the same time exudation of liquor sanguinis and blood corpuscles takes place from the inflamed vessels, and thus the surface of the wound becomes in a few hours glazed over with exuded material—what is termed lymph. (*See* page 59.) If at the time of the operation all foreign material, such as blood clot, etc., be removed from between the cut surfaces, and they be placed in accurate apposition, this lymph will glue them together. If no further causes of inflammation come into play, the inflammatory process ceases at the point of exudation, and healing changes commence.

In a very short time, this lymph which glues together the cut surfaces becomes infiltrated with large numbers of cells, in part leucocytes which have emigrated from the blood-vessels, and in part also larger plasma cells which are derivatives of the fixed connective tissue cells. The function of the white corpuscles seems to be to remove the coagulated blood and lymph which previously glued the edges together, while the plasma cells very soon become spindle-shaped, acquire connections with the cells on either side of the cut surfaces, and, developing into fibrous tissue, thus unite the cut edges by a layer of young fibrous tissue. Very soon this fibrous tissue becomes more fully developed, and the union is, in the course of a few days, comparatively firm. In addition to the development of this fibrous tissue, young blood-vessels are also formed, though in the case of union by first intention this does not go on to any very great extent. As the fibrous tissue becomes older and more perfect, these new blood-vessels again disappear. At the same time that these processes are occurring between the cut surfaces, the epithelium at the margins of the wound spreads over the intervening gap on the surface of this cellular mass and completes the union. This process of healing by first intention varies in rapidity chiefly according to the age of the patient, and also according to his general condition, the union being fairly firm in the case of young children after three or four days, and in older people within a week. The process is the same in the various tissues of the body, and the fibrous tissue which is developed tends to approximate in character to the normal fibrous tissue of the part. Thus in the dermis, the fibrous tissue becomes firm, resembling that of the skin; in the subcutaneous tissue it becomes loose, and fat cells develop in it; in the case of tendon again, it resembles tendon, and so on.

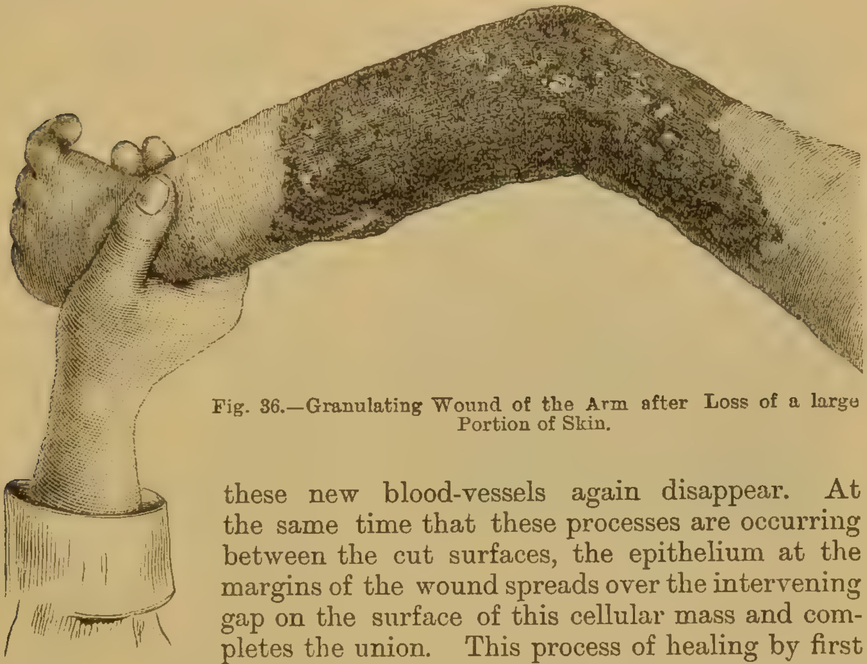

Fig. 36.—Granulating Wound of the Arm after Loss of a large Portion of Skin.

2. **Healing by granulation.**—In this process the edges of the wound are not brought together in the first instance, or, if they are, further causes of inflammation coming into play prevent the occurrence of union by first intention, and they subsequently become separated. Perhaps the simplest way of studying healing by granulation is to suppose that a portion of the skin and subcutaneous

tissues has been completely removed, and that no attempt has been made to bring the edges of the wound together. In such a case, the first effect of the operation is the same as in union by first intention, namely, that as the result of the injury done to the tissues by the knife and by the manipulations, the early stages of inflammation occur up to the point of exudation, and the surface of the wound becomes covered with lymph.

In healing by granulation, further causes of inflammation now come into play and keep up the process, the most common being the development of pyogenic organisms. Other causes, however, which produce a certain amount of granulation, are the irritation of the dressing or of chemical substances applied to the wound. Whatever be the cause, the result is that after a few hours the lymph, which in the first instance glazed the surface of the wound, becomes liquefied and disappears, and the inflammation over the whole surface continues up to the stage of destruction of the tissue and its replacement by granulation tissue (Fig. 12).

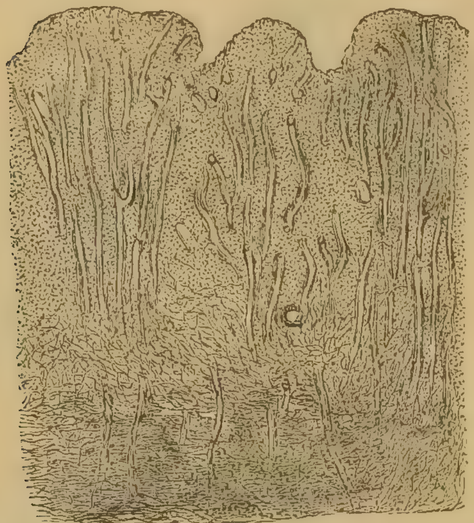

Fig. 37.—Granulations from the Surface of a Wound, showing the Buds, the numerous young Blood-vessels shooting up into each, and the Organisation of the deeper Layers into fibrous Tissue.

After the wound has become covered with this tissue, granulations appear, the young tissue being heaped up in the form of little buds over the whole surface, so that the wound instead of being smooth, is covered with little red warty projections or *granulations* (page 61) (Figs. 36, 37). These granulations go on increasing in size, till by-and-by the whole wound becomes filled up, at the same time the older granulation material at the deeper parts of the wound being protected from further irritation by the layer of granulations above it, organises into fibrous tissue, which like all young fibrous tissue, tends to contract, and thus while on the one hand the depth of the wound is being filled up by granulations, the extent of the wound is being diminished by the contraction of this young fibrous tissue formed in the deeper parts. When these granulations have nearly or quite reached the level of the skin, epithelium begins to spread over the surface from the margins of the wound (Fig. 38), and as soon as they become covered with epithelium and are thus protected from irritation, the cells in the granulations develop into fibrous tissue, which again contracts. This process of growth of granulations, of contraction of the wound, and of growth of epithelium over it, goes on till by-and-by the wound is completely healed, and we have a scar which is considerably smaller in area than the original wound.

While healing is going on, one can easily distinguish two or three layers around the central granulations, the outer layer being a white ring, where there are considerable masses of epithelium which have become macerated by the discharge ; inside this a bluish layer, where there is a moderate thickness of epithelium ; and the innermost layer of all being a pink layer, where there are only one or two strata of epithelial cells through which the red granulations show (Fig. 38). This pink layer generally indicates that healing has occurred within quite a recent period, usually within 24 or 48 hours, and it is of great importance as indicating whether the process is actively going on or not.

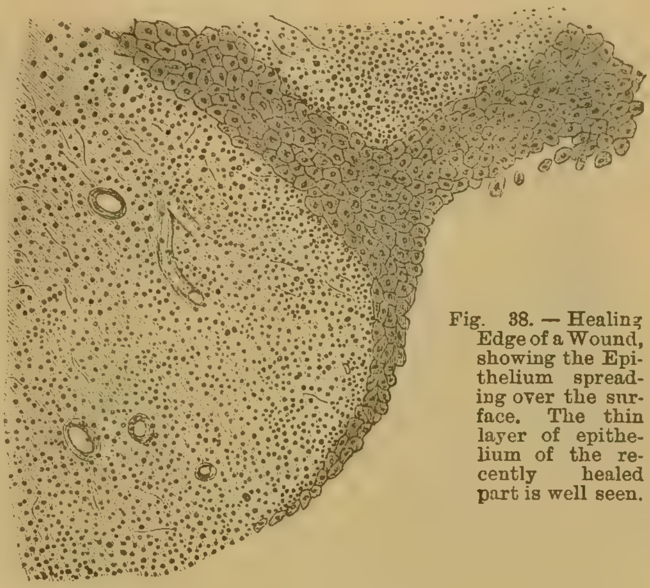

Fig. 38. — Healing Edge of a Wound, showing the Epithelium spreading over the surface. The thin layer of epithelium of the recently healed part is well seen.

When the scar is first formed it is red on account of the large number of new vessels which have been formed in the granulation tissue, but as time goes on it becomes paler, and ultimately is whiter than the surrounding skin, because the new vessels have become compressed and disappear as the result of the contraction of the fibrous tissue (Fig. 39).

3. **Healing by union of granulations.**—About the beginning of the century it was very much the fashion in some quarters not to attempt in any circumstances to obtain union by first intention, on account of the very serious consequences that so commonly followed the occurrence of suppuration in the deeper parts of wounds, the skin edges of which had been brought together. Hence the treatment often adopted was to introduce charpie or other dressing between the edges of the wound till granulation had completely occurred, and then, after washing the pus away, the two surfaces were allowed to come into contact, and immediate union often occurred over a large area. In this case the two granulating surfaces protect one another from further irritation, a slight amount of lymph is poured out after the washing of the wound, and then union occurs between the granulating surfaces at once, the cells present

in the granulations developing into fibrous tissue and joining the cells on the opposite surface of the wound.

This is not a method of healing to be aimed at at the present time, because, apart from the fact that we can obtain healing more rapidly in other ways, pus is apt to accumulate at some part where the granulations have not been actually in contact, or where the suppurative process is going on more actively, and consequently the wound may have to be opened up. It is, however, well to bear in mind that granulating surfaces will adhere in this way under suitable conditions, and if proper provision be made for the escape of discharge from parts of the wound where the surfaces do not come properly in contact, it is a plan which may be employed in certain instances.

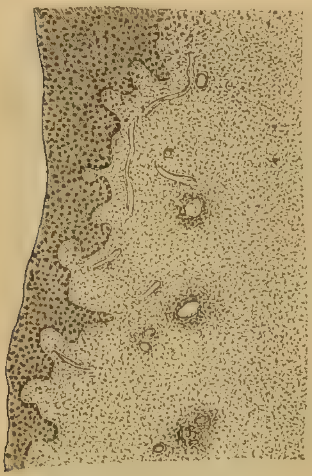

Fig. 39.—Cicatrix, showing the great Decrease of Vascularity, and the fact that there is no Regeneration of any of the complex Structures of the Skin such as Hair and Glands.

4. **Healing by blood clot.**—This is a method of healing which only occurs in aseptic wounds, and which was, therefore, undescribed and but seldom observed before the Listerian era. In this case the edges of the wounds are not brought together in the first instance. A cavity is left into which bleeding goes on, this blood accumulating and filling it up with clot. As the result of the operation itself, lymph is poured out as usual on the surface of the wound between it and the clot, and no septic organisms coming into play, and the wound being kept at rest, the clot remains in it, and no further exudation takes place on the cut surfaces. Hence, in the course of a few hours, leucocytes which have passed out of the blood-vessels begin to penetrate into the clot, and, as shown by the researches of Ballance and Sherrington, collect around the nodal points of the fibrine, and proceed to destroy the red corpuscles in the clot. Following them, there is an emigration into the clot of the larger plasma cells derived from the connective tissue corpuscles, which in their turn prey upon and remove the white corpuscles, and at the same time become elongated and spindle-shaped, and develop into fibrous tissue. Hence, after a few days, those portions of the clot in the immediate neighbourhood of the wound are infiltrated with cells which are developing into fibrous tissue, and very soon the whole thickness of the clot becomes similarly attacked. During this process the clot itself is absolutely inert, and simply acts as a mould into which the cells penetrate, and as a pabulum, more especially for the white corpuscles. After a time, when this process has gone on to a considerable extent, and reached to or near the surface of the clot, epithelium begins to spread from the divided edges of the skin over this organising material, usually under a thin

layer of clot on the surface which does not become infiltrated with cells, and in the course of a few weeks, if this thin layer of clot be lifted up and peeled off, a cicatrix is found beneath.

This process of healing by blood clot is, of course, not limited to open wounds. Where a wound, in which the edges of the skin are united at the time of the operation, is deep and extensive, it is very seldom that the whole of the cut surfaces are in actual contact as is requisite for union by first intention; and in the parts where these surfaces are not in actual contact blood clot is formed, and the process just described takes place. In subcutaneous injuries also this healing by blood clot is almost the only form in which healing takes place.

5. **Healing by scabbing.**—As I have already said, where a portion of skin is removed, or where the edges of the skin are not brought together, the surface of the wound becomes in the first instance glazed with lymph, as a result of the early stage of inflammation set up by the irritation of the operation. In certain cases, where the wound is exposed to the air, this lymph, which in such circumstances would be somewhat excessive in amount, dries up on the surface, and a scab forms in which micro-organisms cannot grow, and which thus protects the actual surface of the wound from further irritation. That being the case, the processes which go on under the scab are essentially the same as those which go on between the cut surfaces of a wound which is healing by first intention, with the single exception that these surfaces are not in accurate apposition. The lymph originally poured out, the surface of which has dried and formed a crust, remains on the surface of the wound, and becomes infiltrated with cells, which organise in the same manner as between the cut edges, while after a time epithelium spreads in beneath the scab over the surface of the organising material, and leads to complete cicatrisation, so that when the scab is picked off a cicatrix is found beneath. This method of healing by scabbing is, however, not to be recommended unless in very small wounds, because it is seldom that the scab is complete over the whole surface, or that it forms quickly enough to prevent the entrance of micro-organisms; and if micro-organisms have entered, suppuration is very apt to occur beneath the scab which confines the pus, and thus lead to the occurrence of ulceration or inflammation of the wound.

General symptoms attending the healing process.—We have now to note the constitutional condition of the patient during the process of healing in the various ways which have been described. Where healing takes place by first intention, or by blood clot, or by scabbing, the general state of the patient remains good, and unless the operation has been a severe one, there is no disturbance of the temperature or pulse, while after the effects of the anæsthetic have passed off, the patient is as well as before the operation.

In cases where the operation has been extensive, or where much blood has been lost, we may have during the first twenty-four hours the condition spoken of as **aseptic fever.** In the first instance in such cases the temperature generally falls below normal, as the result of the shock and loss of blood. Following this fall there is a reactionary rise of temperature, so that within twenty-four or thirty-six hours after the operation the temperature may have reached 100° or even 101°. Where the wound is aseptic the temperature does not remain at this point, but immediately afterwards falls again to normal, and during this period of slight elevation of the temperature the patient does not suffer from any other symptoms of fever. This is what is spoken of as the aseptic temperature, and the aseptic course of the wound is where there is no inflammation in it. Whether the wound be united throughout its whole extent or be left open, where there is no general disturbance and no elevation of temperature beyond that mentioned just now, it will be found that after the effects of the anæsthetic have passed off the patient is to all intents and purposes quite well, unless where the operation has been so extensive, and where the blood lost has been so great as to cause excessive weakness.

On the other hand, where the wound is not aseptic—for example, in wounds where the healing takes place by granulation—there will be more or less constitutional disturbance. In the first instance the temperature rises very soon after the operation, and reaches its highest point within twenty-four to forty-eight hours—usually 103° or 104°. It remains at about this height for three or four days, and then pretty rapidly falls to normal. During this period the wound is painful, the edges are red and swollen, and there is a considerable amount of discharge from it, while the patient suffers from the various symptoms of fever—namely, quick pulse, loss of appetite, arrest or diminution of the excretions, and the various symptoms described in speaking of acute inflammation. This is the condition known as **traumatic or inflammatory fever,** and is due to absorption from the wound of septic products which have been developed by the bacteria growing in it, more especially by the pyogenic bacteria.

Where the wounds are very large and contain a quantity of decomposing blood, a large amount of the chemical substances may be rapidly absorbed, producing the condition known as *septic intoxication* or *sapræmia.* In this condition there is usually a rapid rise of temperature with a very rapid, feeble pulse, and great exhaustion of the patient; indeed, in very bad cases, after the preliminary rise of temperature, it again falls, and the patient passes into a state of collapse and may die. Formerly, this condition was not recognised as being due to septic absorption, and was spoken of as secondary shock; but we now know that the great majority of the cases of so-called secondary shock are in reality cases of acute septic poisoning.

Further, during the early period of these septic wounds the initial temperature of traumatic fever may not fall when the wound has granulated, or, having fallen, may again rise, and the condition of *septicæmia* sets in, where an intermittent high fever is developed, and the patient passes into a state of asthenic fever. This condition of septicæmia is probably due to the penetration of pyogenic organisms into the body, and possibly into the blood, and the poisoning of the blood with the products of their growth. Or, again, even after the complete subsidence of the traumatic fever, the patient has a severe rigor, accompanied by a rapid rise of temperature, and followed by an equally rapid fall, these rigors being repeated at lessening intervals, and the condition of *pyæmia* developing.

Conditions which interfere with healing by first intention or blood clot.—It is thus evident that wherever it is possible one should strive to obtain healing by first intention or by blood clot, and to avoid healing by granulation and suppuration. By doing so, the patient recovers much more rapidly, without having suffered any pain or constitutional disturbance, and without having run the risks which I have mentioned as incidental to the process of healing by granulation, and at the same time the resulting scar is much less evident, becoming, in fact, after some months, but slightly visible. It is necessary, therefore, to consider more in detail the conditions which interfere with healing by first intention or by blood clot.

In order to obtain healing by first intention or by blood clot, the essential point is to place the wound completely at *rest*, whether it be from mechanical disturbance or from the action of chemical substances. Mechanical rest of the wound may in most cases be obtained by bringing the edges accurately together and by placing the part at absolute rest, if necessary, on a splint, while at the same time care is taken that the dressings shall not irritate either by rubbing over the surface of the wound or by direct contact with it. The most important point, however, is to provide against the irritation of chemical substances, and these chemical substances may be either various applications which are made to the wound, or substances developed in it as the result of the growth of certain bacteria. The irritation of chemical lotions is, of course, readily avoided, and lotions that are irritating to the wound should not be applied to it during the after-treatment. Above all, where a wound is aseptic there is absolutely no necessity for injecting antiseptic solutions into it; on the contrary, to do so will be to irritate it and prevent the desired result of union by first intention.

The essential causes which lead to the failure of healing by first intention or by blood clot, and which expose the patient to the risk of various septic diseases, are the *growth of micro-organisms* in the material on the surface of the wound, and subsequently in the tissues themselves. The organisms that act in this way are generally the pyogenic organisms, more especially the various kinds of pyogenic cocci that have been already referred to in speaking of suppuration (pages 25 and 81). Where a wound is freely open to the entrance of organisms, however, these are not the only ones which may grow in it. The early disappearance of the blood clot and the occurrence of traumatic fever and of septic intoxication are often associated with and, no doubt, in part, due to the growth of ordinary saprophytic and putrefactive bacteria in the fluids and blood of the wound, and although these organisms are not parasitic and cannot penetrate into the living body, their chemical products are intensely poisonous and irritating, and if absorbed may cause very serious effects. I have already described, in speaking of suppuration (page 82), how it is that these organisms produce an acute abscess, and the process is exactly the same where, instead of causing the formation of pus in a closed cavity, they lead to granulation and suppuration from the surface of the wound.

I have already discussed the conditions under which these organisms can act in the tissues, and the same facts hold good with regard to open wounds. More especially it must be borne in mind if these organisms are likely to enter, that the manipulations of the surface of the wound should be particularly gentle, so that as healthy a tissue as possible is opposed to their entrance. Where this point is not attended to, these organisms are particularly likely to gain a foothold in the tissues and cause suppuration, and thus prevent healing by first intention; while if parts of the wound are much bruised, actual sloughing may take place.

As to the mode in which these organisms enter wounds, they may fall in along with the dust of the air, but most usually they are conveyed to it from the instruments or the operator's hands, or grow into it subsequently from the skin of the patient himself. These pyogenic organisms seem to be normal inhabitants of certain parts of the surface of the skin, more especially parts that are moist, for example, the axillæ, perineum, the space between the toes, etc., and unless special care be taken to destroy those which are present on the surface of the skin, they may, after the operation, spread into the wound and grow there, producing the various effects to which I have referred. In fact, it is but seldom that these organisms gain entrance to the wound along with dust, because they die soon if they be allowed to dry, and consequently, unless when one is operating in hospitals, or in places where there are patients with septic wounds, and where therefore the drying of the discharge containing the organisms has only recently taken place, living organisms are not likely to be introduced from the air.

We must also consider the various modes in which the life and action of these organisms may be interfered with. They grow very readily on a variety of soils outside the body, but one very essential condition of their growth is that the nutrient material shall contain a large quantity of water, at least 90 per cent. Where the nutrient material is too concentrated bacteria cease to grow, or grow only with great difficulty and without any marked degree of virulence, and this is a point of great importance to bear in mind in the treatment of wounds, because if it be not possible to exclude bacteria from wounds, one way in which their action may be interfered with is by permitting evaporation of the fluids contained in the wound, and thus producing too concentrated a medium for their growth. Another point which the pyogenic bacteria share in common with a considerable number of others is that when grown in the presence of air they do not produce such a quantity of poisonous materials as when air is absent, and it was on these two points that the good results which were formerly obtained by the open method of treatment depended, for on the one hand plenty of air was furnished to the part, and thus the amount of poisonous materials produced by the bacteria was diminished, while evaporation was promoted, and the fluids tended to become so concentrated that the bacteria did not readily grow.

The most important point, however, with reference to the life history of these bacteria, more especially in relation to the present methods of wound treatment, is their behaviour with regard to heat and various chemical substances. Bacteria vary greatly in their relation to heat, according to the stage of their existence. Those forms which grow rapidly, more especially the micrococci, are very readily destroyed at comparatively low temperatures; but those forms which produce spores, if they have already passed into the spore stage, resist heat to an extraordinary degree. Indeed, the spores of bacteria may be exposed to dry heat considerably above the boiling-point of water for hours without losing their vitality; while, on the other hand, the same bacteria in the adult stage will be destroyed by short exposure to a very moderate degree of heat; indeed, a thorough and prolonged drying, even without the action of heat, will lead to their death. The same differences are observed between adult bacteria and spores in their relation to various chemical substances, Fortunately, the organisms which for the most part come into play in surgery belong to the non-spore-bearing bacteria, and are thus much more readily killed; indeed, with the exception of the anthrax bacillus, the tetanus bacillus, the tubercle bacillus, and one or two others, we have essentially to do with cocci, which are very readily destroyed by comparatively dilute chemical substances. The pyogenic cocci, for example, are destroyed in a few seconds by the action of a 5 per cent. watery solution of carbolic acid, and even by a 2½ per cent. solution; and they are also destroyed, though perhaps not quite so rapidly, by a 1 in 2,000 solution of bichloride of mercury, and, of course, much more rapidly where the solutions are more concentrated. While this is the case with regard to watery solutions of these antiseptic substances, it is very important to bear in mind that oily solutions are not nearly so active; in fact 1 to 20 solution of carbolic acid in oil is practically inert as a means of killing bacteria; indeed, a 1 to 5 solution of carbolic oil will not do what a 1 to 20 watery solution will. Apart from the destruction of the vitality of these organisms by antiseptic solutions, we have in the treatment of wounds to remember also that it is sufficient if we prevent their growth, and that very much smaller quantities of carbolic acid, and more especially of corrosive sublimate, will interfere with the growth of the organisms, although they may still remain alive; and if the growth be prevented, they cannot penetrate into the tissues, or if they do come in contact with living cells, they are quickly destroyed.

It is also an important point to remember that the tissues themselves have a very great power of resisting the entrance of bacteria, and hence it is only under certain special conditions, which have been in part referred to in speaking of suppuration, that they can really obtain a foothold in the body. How it is that the tissues act as destructive agents of bacteria is still a matter of dispute. It is supposed that the products of certain bacteria have an attractive power on white blood corpuscles, what is termed chemiotaxis, and that it is in consequence of this attractive action on the white blood corpuscles that the enormous migration of cells which takes place in inflammation is brought about. The products of other bacteria, on the other hand, have the opposite effect, so that with them, instead of an active migration of leucocytes, their field of action remains comparatively free from cells. Whether there is such an effect on the white corpuscles by the products of the bacteria or not, it is certain that in cases where death is not occurring with extraordinary rapidity, that is to say, where the bacteria are not extremely virulent, there is enormous heaping-up of leucocytes in their neighbourhood, and it is held on the phagocytic theory that these leucocytes or other free cells seize on the bacteria by means of their amœboid action, take them into their own protoplasm, and there cause their destruction. (*See also* page 23.)

On the other hand, a large amount of evidence has been brought forward to show that there is present in the serum of the blood in certain animals and in certain circumstances substances that have a destructive action on the bacteria, and hence some hold the view that the first thing which occurs is a weakening or destruction of the vitality of the bacteria by means of these chemical substances, and that it is only when the organisms are so injured that the cells are able to take them up and destroy them. It is most likely that the truth lies between these two theories, if indeed these are the only possible explanations. However that may be, we have the broad fact perfectly clearly established as the result of clinical evidence that the tissues when in a healthy condition have a great power of resisting the entrance of bacteria, and even of destroying them when present under certain conditions, such as where the number of the organisms is not too great, where their products are not too concentrated, where their virulence has been diminished, and so on.

THE TREATMENT AND VARIETIES OF WOUNDS.

The treatment of wounds must vary in different circumstances, and we shall have to consider the following classes of wounds from the point of view of treatment, namely—

1. Those made by the surgeon through unbroken skin.
    - (*a*) Where the edges of the skin can be brought together.
    - (*b*) Where union by first intention fails.
    - (*c*) Where the edges of the skin cannot be brought together.
    - (*d*) Wounds of mucous membrane.
2. Accidental wounds.
3. Contused wounds.
4. Lacerated wounds.

The importance of these distinctions concerns the question of sepsis, which, as I have already indicated, is the main point to be considered in the treatment of wounds. Where the wound is made through unbroken skin by the surgeon the avoidance of sepsis practically entirely depends on his care. Where mucous membrane is involved in the wound it is impossible for the surgeon completely to exclude bacteria, and he must do his best to diminish their action.

Where the wound has been made accidentally the problem is, on the other hand, a totally different one, because here the bacteria have already entered the wound, and the surgeon's efforts must be directed, if possible, to getting rid of them again.

1. **Wounds made by the surgeon through unbroken skin.** (*a*) **Where the edges of the skin can be brought together.**—In such wounds the great aim must be to obtain healing by first intention, and in order to get this we have to follow out the principles which have been already indicated—namely, to provide rest for the part, to avoid any mechanical or chemical irritation by applications to the wound, and, above all, to secure the absence of pyogenic organisms. This is best done by paying the fullest attention to the details of antiseptic or aseptic surgery, which is the best and most certain method of treatment. With the view of obtaining healing by first intention, and avoiding the various dangers of wounds, I shall describe the method I now carry out—namely, *aseptic treatment on the Listerian principle*, which aims, as far as possible, to destroy all organisms which may obtain access to a wound, and to prevent their entrance subsequent to the operation.

In taking measures *to prevent the entrance of bacteria* into a wound we must in the first place look to the sources from which the bacteria may come. As I have mentioned, they may come from the dust either in the air, or deposited on surrounding objects, or from the skin of the patient, or from that of the operator, on which they may not only be lying, but actually growing. In the foremost place the greatest attention must be paid to the *skin*; in fact, it is essential that the pyogenic organisms should be thoroughly removed both from the skin at the seat of operation, and over a sufficient area around to allow ample overlapping of the dressing, and also from the hands of the surgeon and his assistant. On the skin these bacteria especially inhabit the surface epithelium and hairs, and penetrate to a slight extent into the sebaceous and hair follicles where these are large. Care must be taken that the antiseptic substances employed shall thoroughly reach and act upon the bacteria contained in these parts. It may be sufficient, in order to avoid the introduction of bacteria at the time of the operation itself, to destroy the bacteria which are present on the surface of the skin, and that is probably all that need be done with regard to the surgeon's hands; but in the case of the skin in the neighbourhood of the wound this is not always sufficient, because, in spite of the antiseptic dressings applied afterwards, bacteria which may have been left behind in the deeper parts in the hair and sebaceous follicles, may grow in the epithelium, and very shortly reach the wound and develop in it. Therefore it is of importance that the disinfection of the skin of the patient should be so thorough as to destroy, if possible, all the bacteria present in it, and certainly all the pyogenic cocci. In order to do this we have to get rid of the surface epithelium, to get rid of all the oily material on the surface of the skin, to remove the hairs, and then to saturate the part thoroughly with strong antiseptic solutions for a sufficient length of time. Hence the method which I adopt to disinfect the skin is in the first instance to wet it thoroughly with turpentine, with the view of getting rid of the fatty materials (the turpentine also is an antiseptic). Following the application of the turpentine the part is well washed with soap and carbolic acid lotion. I generally use a five per cent. lotion of carbolic acid containing a five-hundredth part of corrosive sublimate in solution. By means of soap and this solution and a nail-brush the surface epithelium and fatty material are thoroughly removed from the surface of the skin, and the part is then shaved in order to get rid of the hairs. A further application of this same "strong mixture" is then made, and where time will permit, a cloth saturated in the solution is wrapped round the part for some two or three hours before the operation, and then at the time of the operation the whole process—turpentine, nail-brush, etc.—is repeated.

As regards the *surgeon's hands*, the main point to be attended to is the disinfection of the nails and the parts under the nails and around their bases, and here also the same measures are employed. The hands are first sponged over with turpentine, then they are thoroughly washed with soap and the "strong mixture" and a nail-brush, and then the nails are carefully cleaned with a knife so as to remove all the dirt and dead epithelium from beneath and around them. The hands disinfected in this manner are not dried afterwards, but a basin containing 1 in 2,000 sublimate solution is kept close by in which they are repeatedly dipped during the progress of the operation.

All the *instruments* which will come in contact with the wound should also be thoroughly disinfected, and this is generally quite satisfactorily done by immersing them in 1 to 20 carbolic acid solution for at the most about half an hour before the operation. This is quite satisfactory except in the case of toothed instruments, such as forceps, where care must be taken to clean the teeth thoroughly, and not to leave any greasy material behind. It may be accepted as an axiom that a 1 to 20 watery solution of carbolic acid will destroy bacteria—at any rate, cocci—in a few seconds, provided it can penetrate into all the recesses of the material. It can certainly do so in almost any material in a few hours, and many of the precautions which are taken in asepticising instruments, etc., are quite unnecessary, and are only too often employed with the most scrupulous care while equally important precautions are totally neglected. No doubt the boiling of the instruments renders their disinfection perfectly certain, and it is strongly to be recommended where instruments have been previously employed for a septic case, more especially for cases where bacillary diseases are present, such as tetanus, but in the majority of instances it is unnecessary, and it is certainly not so rapid in its action as undiluted carbolic acid, while it is only applicable to instruments that are made entirely of metal. Where some instrument has been overlooked in arranging for an operation, and is called for during its course, it is most rapidly disinfected by dipping it in undiluted carbolic acid for a few seconds. This is then washed away by a 1 to 20 carbolic acid lotion, when the instrument is ready for use. Simple immersion in boiling water—which is as long as one could give if the instrument were required in the course of an operation—would not disinfect it. It must actually remain in the boiling water for some minutes. On the whole, therefore, I prefer to adhere to the old plan of employing a 1 to 20 solution of carbolic acid for the disinfection of the instruments, but in cases where very septic wounds have been treated, I think it is well to boil them before laying them aside.

Not only must the instruments be disinfected before the operation has begun, but care must be taken not to infect them again during the course of the operation. Thus reinfection might readily occur if the instruments were laid on a blanket or dry towel, or allowed to drop on the floor ; and it is therefore necessary so to arrange matters that such an accident cannot occur, and this is done by placing over the blankets and clothes and table, for a considerable area around the seat of the operation, towels which have been soaked for some time in a strong aseptic solution, either of carbolic acid or of corrosive sublimate. These towels are squeezed out of the solution, but should not be wrung dry ; it is well to have them fairly saturated with the fluid. They are then placed on mackintoshes and arranged around the area of the operation, so that if instruments are laid down, or if the hands are rested at any time, they can only be placed on this aseptic basis. As a further precaution, I always have before me a small basin containing a 1 to 2,000 sublimate solution, in which I dip my hands from time to time as well as forceps and other instruments. It is just possible that one might accidentally soil one's hands or the instruments during the course of the operation, and without such a solution at hand, mistakes might be made which would lead to septic infection of the wound.

A very important question is whether it is necessary to *irrigate the wound* during the course of the operation, that is to say, whether it is necessary to take means to destroy organisms which may have fallen in from the air. In hospital practice I think that this is necessary. In places where there are wounds, dried-up discharges, dried-up blood, numbers of people—many of whom have come from dissecting-rooms—and so forth, I cannot but think that the air in an operating theatre must contain pathogenic organisms, and therefore I think it is necessary to take means to prevent their taking foothold in the wound. Formerly this was done by means of a spray of carbolic acid, and it was supposed that the spray acted by destroying organisms which were floating in the air. This has now been clearly proved to have been erroneous, but in addition to the spray passing through the air over the wound, the wound itself was constantly bedewed with the antiseptic solution—in other words, it was gently irrigated—so that although the organisms were not disinfected while floating in the air, they fell on to a wound, the surface of which was covered with a layer of antiseptic fluid, which could at once act on them. The objections to the spray as an irrigator were, however, numerous, more especially the strength of the carbolic acid was very disadvantageous for the healing of the wounds, and therefore it has been given up almost entirely, and its place taken by irrigation. As a material for irrigating wounds I use a weak sublimate solution, certainly nothing stronger than 1 to 2,000; and in cases where a joint is opened, I generally employ 1 to 4,000 or 1 to 6,000. These sublimate solutions do not irritate the wound to anything like the degree that carbolic acid does, and in fact I see no reason to suppose that they materially interfere with the healing process, while for the reasons I have mentioned I believe that they are an advantage, more especially in hospital practice. That they are not absolutely essential in the great majority of cases we know, but what one aims at, especially with this method of treatment, is to obtain certainty and not probability as regards the results. As a rule I do not employ any special irrigator. In the majority of wounds I simply from time to time squeeze a sponge saturated with 1 to 2,000 solution over the wound, and this is more especially done while the wound is being stitched up. In cases, however, where I am operating on a healthy joint, such as the knee joint, I feel happier when there is a constant gentle stream of weak sublimate solution flowing over the wound from the time that the joint is opened.

As regards the use of *sponges*, for my own part I still employ them. Some surgeons have given them up, and use instead pads of absorbent wool, but these are to my mind very objectionable, especially in the way in which I have often seen them employed. It is not uncommon to see a dishful of dry pads of wool brought into the operating room, and these are simply wrung out of the solution before being handed to the operator. In such circumstances, no time is allowed for proper disinfection of the pad, and the result must be, in a considerable number of cases, that infective material is left behind in the wound. Further, these pads are not so absorbent as sponges, and, in addition, portions of wool are apt to become detached and remain behind in the wound. That, of course, would be a matter of no consequence if the wool were really aseptic; but, as I have said, in many cases the immersion in the antiseptic is so short that these portions of wool left behind not infrequently contain bacteria, and form a source of subsequent infection of the wound. Sponges are, on the other hand, much more satisfactory for the purpose of absorbing blood, and are perfectly reliable if only they are properly seen to. After an operation I always have the sponges thoroughly washed in cold water, and then subsequently with soap and water containing a little soda, and when they are thoroughly cleansed they are placed in a jar containing 1 to 20 carbolic acid solution, and this solution is renewed every three or four days. The result is, that at the end of a week—and the same sponges are not used again before that—they are satisfactorily disinfected, and care must simply be taken not to infect them during their transit to the surgeon. This is, however, what is very often done. The nurse often rinses the sponges in water, then dips them in an antiseptic solution, squeezes them dry, puts them in a dish which has not been rendered aseptic, and from the dish hands them to the surgeon, either directly or very often after rubbing blankets, coats, etc., in their transit. What I do is, after removing sponges from the 1 in 20 solution, to rinse them thoroughly in 1 to 2,000 sublimate lotion, and then to place them in a basin containing the same solution. This basin is held within reach of the surgeon or his assistants, and when a sponge is required they squeeze it out for themselves. Thus the errors which may be committed by the nurse during the transit of the sponge are completely avoided, and there is practically no loss of time or increase of trouble to the surgeon in squeezing the sponge; in fact, I very often squeeze the sponge over the wound, thus at the same time employing it for the purpose of irrigation. After the sponge has been used it is returned to the nurse, who again washes it in sublimate solution and places it in a fresh vessel of the same.

As regards *ligatures*, I believe that catgut is still the most universally applicable material when prepared in the way last described by Sir Joseph Lister, by the employment of sulphurous acid and chromic acid. As a matter of fact, one does not require to ligature many vessels in a wound now that we have such excellent pressure forceps; but where it is necessary, the finest sulpho-chromic catgut forms a trustworthy ligature. Objections have been urged as to the asepticity of catgut, and these objections no doubt hold good with regard to the old method of preparation in carbolic oil. In hospital we always keep our catgut in 1 to 20 carbolic acid solution for at least a week before using it, and it is handed directly from that solution to the surgeon, and used in this way it may be relied on as being thoroughly aseptic. Silk also acts perfectly well for ligatures, but one must be quite satisfied as to its asepticity. In order to ensure this, some surgeons take excessive precautions, the most essential of which is that they boil the silk very carefully before use. This precaution is, I believe, quite unnecessary. As I have said before, twenty-four hours' immersion in 1 to 20 carbolic acid solution will ensure the asepticity of almost anything which can be thoroughly penetrated by the solution, and, therefore, it seems to me quite sufficient to keep the silk in it for a sufficient length of time before it is used. We, therefore, keep the silk, like the catgut, for about a week in the 1 to 20 solution before employing it.

The operation having been concluded the wound must of course be stitched up, and in connection with this point the question of *drainage* arises. Formerly, great stress was laid in antiseptic work on the importance of drainage of the wounds, and undoubtedly it was a matter of clinical experience that if the wounds were not satisfactorily drained, one frequently had interruption of the healing process from the large amount of serum which was poured out after the operation. This was, I believe, to a great extent due to the constant irritation of the wound with carbolic acid, more especially with the spray, and the consequent excessive amount of serum which was exuded. I certainly have found that since employing weak sublimate solutions for irrigation, this great quantity of serum is not poured out, and consequently one of the chief reasons for employing drainage is done away with. In fact, I very seldom employ drainage of a wound unless in certain circumstances, using instead of it pressure by means of sponges incorporated in the dressing in the manner to be immediately described. Where, however, pressure cannot be satisfactorily employed, I think that it renders the rapid healing of the wound more certain, and adds to the comfort of the patient, to insert a drainage-tube for two or three days. Such cases, for example, as the stump after amputation, where the flaps are thin and cannot be subjected to pressure, removal of a bronchocele, where pressure cannot be applied on account of the presence of the trachea, the removal of the breast in very thin women, where the flaps of skin are thin and might slough, in very fat people also, where a quantity of oil is poured out into the wound, I believe that it is always best to employ drainage. Of course, in cases where the asepticity of the wound is doubtful—as, for example, where there has been an open sore or a sinus in the region operated on—it is essential to employ drainage for two or three days in case the wound should not be aseptic. In the great majority of cases, however, the wound may be sewn up without the use of drainage. Some surgeons stitch together the deeper planes of the wound with catgut so as to avoid the occurrence of a cavity, but except where one wishes to unite muscular or fibrous tissues in order to give support to the part, I think this is quite unnecessary, because the same result will be obtained by suitably applied pressure.

As regards *the union of the edges of the wound*, one must as far as possible avoid all traction on the immediate edges. If there be a difficulty in bringing the sides of the wound together, the stitches that are employed for the purpose must be inserted at some distance from the edge; in fact, where the tension is very great it is well to employ the button stitches that were introduced by Sir Joseph Lister. In applying the button stitch, a needle threaded with stout silver wire is inserted at a considerable distance from the edge of the wound, and the end of it is fastened to a lead button, so that a considerable area of pressure is exerted at the base of the flap. The needle is then carried through the opposite side of the wound, also far away from the edge, and when the wire is pulled as tight as possible, another button is attached in a similar manner on that side. Two or three pairs of buttons will generally suffice in the case of a large wound, and will enable one to approximate the edges much more closely. As a rule, where much skin has been taken away, it is necessary, in addition to these button stitches, to employ stitches of thick silver wire at various points in the course of the wound, these stitches being also inserted at some little distance from the edge, so as not to constrict the actual healing edge. Where there is much tension these stitches should be numerous, so as to distribute the pressure over a considerable area. Finally, the actual edges of the wound are brought into contact without any tension by means of fine suture of silk, fishing-gut, horse-hair, catgut, or any material which the surgeon fancies. For my own part, I generally employ a continuous stitch of fine silk, the stitch being inserted in the same manner as in making button-holes, that is to say, the needle always passing *inside* the loop of the silk, so that on one side there is practically a continuous line of silk. These stitches may be left in for a week or ten days. As a rule, however, it is well to remove the button stitches after four or five days; otherwise they are apt to cut through or even to cause a slough at the point of greatest pressure. Where it is desirable to have as little scar as possible, as, for example, in the face, it is very important to avoid the stitch marks, which are often much more disfiguring than a linear scar, and this can be done by inserting stitches of very fine catgut by means of a Hagedorn's needle between the deepest layers of the dermis on each side. These stitches remain in and gradually become absorbed, and there is no puncture through the skin itself. The actual epidermic line can then be brought together by strips of gauze fixed on the skin on each side of the wound by means of collodion.

Having stitched up the wound, it should be thoroughly squeezed, so as to press out all the sublimate solution which has entered during the irrigation, and also to expel any fluid blood which may be present, and then the *antiseptic dressings* are applied. I believe the most universally applicable and most satisfactory dressing is the double cyanide gauze (mercury and zinc) introduced by Sir Joseph Lister. This should be wrung out of either 1 to 40 carbolic lotion, or 1 to 4,000 sublimate solution, and applied over the wound, and in a considerable mass over a large area around; and among the layers of this gauze I place one or more sponges which have been thoroughly wrung out of carbolic acid solution, so arranged as to press over all the recesses of the wound. Outside these sponges fresh layers of gauze are applied, overlapping them in all directions, and outside of all a mass of salicylic wool. This is then firmly bandaged on, and, unless where a drainage-tube is employed, the dressing need not be changed for about ten days, when it will be found that the wound has healed and the stitches can be removed.

It is an occurrence of the very greatest rarity, where proper precautions are taken, for suppuration to occur in a wound; and, if it does occur, we must at the present day accept the view that it is due to some oversight on the part of the surgeon or his assistants. Where a drainage-tube has been used and the wound remains aseptic, the dressing should be changed after about three days in order to remove it; and, no doubt, the patient is more comfortable after the first dressing, because the bandage can be applied more in accordance with his sensations; but, on the whole, I think where a drainage-tube is not employed that it is best to leave the dressing on till the wound is completely healed, otherwise, in removing the dressing, one is very apt to disturb the union which is occurring in the deeper parts, because the dressings applied as I have described very soon dry up, and the deeper layer adheres firmly to the skin and the edges of the wound, and, therefore, in removing it, these parts are apt to be pulled upon.

In *changing the dressings* similar precautions must be employed as have been described in making the wound. For example, a wet towel should be arranged in the neighbourhood of the wound, so that if by any accidental movement of the patient the clothes should shift, what would come in contact with the wound would be an aseptic towel, and not the dirty blankets. The lotion I generally use is 1 to 2,000 sublimate solution, but in parts such as the axillæ, or after operation for hernia, where there may be a certain amount of bacteric growth in the neighbourhood of the wound, I generally wash the surrounding skin with 1 to 20 carbolic lotion. For the purpose of washing the skin I use salicylic wool taken from the layer of wool removed in the dressing which has been next the cyanide gauze. This layer of wool is aseptic, having been protected from dust by the superficial layer next the bandage, which is not employed for this purpose. Of course, it need hardly be pointed out that in washing the parts after the removal of the dressing one must not wash the surrounding skin, and then with the same cloth wash the wound subsequently, because one might quite readily transfer septic material from the surrounding skin to the wound in this way; and in order to avoid any risk of this kind it is well, as soon as the wound is uncovered, to lay a piece of the soaked wool over the line of incision before proceeding to clean the parts. One may then set to work and thoroughly cleanse all the skin in the vicinity without any risk of involuntarily transferring septic material to the wound itself. As a matter of fact, it is not necessary to wash the wound, and, above all things, it is not necessary to inject antiseptic solutions into it, if, indeed, a cavity were present into which one could inject; and therefore, after having washed the skin around, one may uncover the wound, cut the stitches, possibly wipe the neighbourhood of the stitches with a little lotion, and then re-apply the dressing as before.

Of course, while one is in this way arranging for the absence of sepsis from the wound, one must also attend to the other details of wound treatment, more especially to securing mechanical rest to the part; and in the case of the extremities this is best done by placing the limb on a splint after the operation. In the case of operations about the neck, for example, the large masses of wool and dressing which are applied generally act in the first instance as a thoroughly efficient splint.

As regards the *general treatment* also, the patient should for a day or so, till he has recovered from the effects of the anæsthetic, be kept on slops; but in two or three days—if the wound be following an aseptic course—he should be allowed generous diet, and, in fact, he may be permitted to take his ordinary food. As a rule, about the end of the second day it is well to administer an aperient Stimulants are not necessary, unless in cases where a very extensive wound has been made, and where there is consequently exhaustion of the patient from loss of blood and shock.

In Germany, more especially, it is becoming the fashion at the present time to avoid entirely the use of antiseptics in contact with the wound, and there, after thorough disinfection of the skin and of the instruments (the latter by boiling), no further antiseptic is employed; and the dressings which are applied are composed of materials which have been sterilised by heat, and which do not contain any antiseptic substances. Theoretically, such a method is an ideal one, and practically it can be carried out in certain circumstances by persons who have had large experience in bacteriological work; but in ordinary practice the difficulties in the way of securing asepsis of the wound in these circumstances are so great that the surgeon cannot have any certainty as to his results. The precautions needed to prevent the entrance and action of micro-organisms are so irksome that it is practically impossible to carry them out satisfactorily, more especially where one is dealing with an operation which requires the exercise of much skill or thought. In my opinion, it is much better to use antiseptics which, although they no doubt irritate the wound to some slight extent, correct involuntary mistakes on the part of the surgeon, and thus add a much greater amount of certainty to the results. Such being the view which I take of this plan of treatment, I need not enter into any details with regard to it. The method which I have described will secure union by first intention practically in all cases, unless some error has crept in during the operation.

(*b*) **Where union by first intention fails.**—In some instances, however, union by first intention does not take place throughout the whole extent of the wound, sometimes because drainage has been omitted where it ought to have been employed, and serum has therefore collected and prevented the approximation of the deep surfaces; at other times because the incisions through the skin have been made obliquely, leading possibly to a narrow slough at the edge; or because the stitches have been much too tight, and have set up a certain amount of irritation; or because movement of the part has been permitted to an excessive degree.

The great cause, however, of failure of union by first intention is the growth in the wound of pyogenic organisms, and these organisms must have entered from outside, either at the time of the operation or subsequently, and they can only enter if some error have been committed in carrying out the details previously described.

Where this cause of failure of union by first intention comes into play, various symptoms arise which indicate what has occurred. In the first place, the temperature rises rapidly to a much higher degree than takes place in the case of the mere reactionary temperature where the wound is aseptic, usually reaching 103° or 104° within twenty-four hours. This rise of temperature is also accompanied with other symptoms of fever, as distinguished from the aseptic rise where no other febrile phenomena are present. The patient presents all the characteristics which have been previously described in connection with inflammation, that is to say, the pulse becomes rapid, the skin hot and dry, the tongue furred, the excretions diminished, and possibly there is some delirium. The temperature in these cases of *traumatic or inflammatory fever* generally begins to fall about the second day, and in uncomplicated and not very severe cases will again reach the normal on the fifth or sixth day; at the same time the patient complains of great pain in the wound, which becomes swollen, the edges of the skin red, and presently pus is found in it. (*See* page 65.)

In other cases after the traumatic fever has passed off, or towards the end of its course, other symptoms arise, indicating grave *septic complications*, such as septic intoxication, acute septicæmia, or pyæmia, or erysipelas, conditions which are described in other Articles. In ordinary uncomplicated wounds, where these graver forms of septic disease do not arise, the general course is that the edges of the wound in the first instance become glued together, subsequently separate more or less completely, pus exudes from the deeper parts, which become covered with a layer of granulation tissue, and the wound gradually heals from the bottom. Where these symptoms arise, the first point in the treatment is to provide for free exit of the discharge; and, therefore, when a patient's temperature rises and remains high, and when he complains of pain in the wound, the dressing should at once be changed, some of the stitches removed, the edges of the wound separated at that part with sinus forceps, and a large drainage-tube inserted. In these circumstances, I do not think it advisable to wash out the wound with any antiseptic solution, because by that means there is no chance of getting rid of the septic condition, and the injury to the surface of the wound may actually enable the organisms to penetrate into the body and give rise to graver local and general phenomena. It is only in cases where wounds are large, and where the symptoms are due to septic intoxication, that I think washing out the wounds is good practice. There, of course, it is essential that the putrefying material in the wound should be got rid of as soon and as completely as possible, and in such cases the washing out of the wound is merely employed to remove these poisonous substances mechanically; and, therefore, it may be carried out with plain water or boracic lotion quite as effectually, and with much less disadvantage to the patient, than by the use of strong antiseptic solutions.

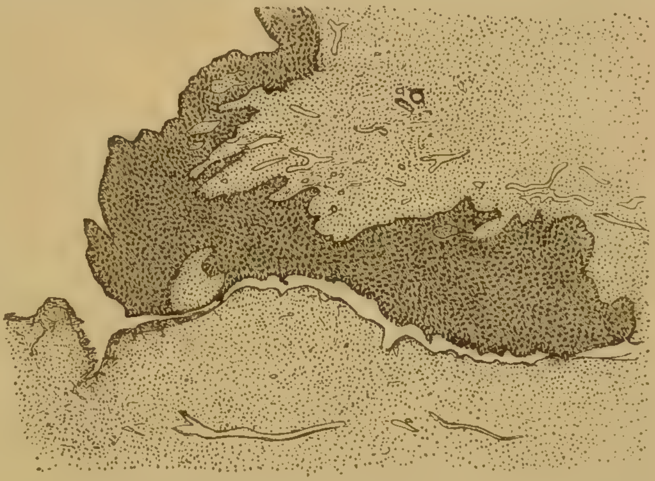

Fig. 40.—Section through a small Sore which had remained unhealed for many months. It will be seen that epithelium has spread in along one side of the sore. On dissecting away the invested part and uniting the edges the sore healed at once.

As to the subsequent dressing of a wound which has become septic, I think, unless there be much pain and swelling, that it is well to continue the sort of dressings already described; but where there is much swelling and pain relief will be obtained by employing dressings which partake of the character of fomentations. The antiseptic material—whether it be cyanide gauze or boracic lint, or other substance—is wrung out of warm lotions, and outside this a layer of mackintosh overlapping it in all directions is applied, with a layer of wool outside. I think it is well in most cases to employ antiseptic material for the purpose of these fomentations, or for the dressings, because, although bacteria have already entered, it is possible that the kind which has got in is not so virulent as others which might get in, and while by continuing the antiseptic dressings we do not influence those which have already entered, we may possibly prevent the entrance of worse forms.

It is of importance to remember, where the edges of the wound are not at the same level, that one side may be drawn in, or that epithelium may spread down one side, and when this occurs the raw surface in contact with the epithelial-covered surface will not heal (Fig. 40). In such a case it is necessary to dissect away the inverted edge, or the epithelial-covered surface, so as to have two raw surfaces opposed to each other.

(c) **Where the edges of the skin cannot be brought together.**—It is not always possible to bring the edges of the skin together after operation. For example, in many cases it is necessary to remove such a large amount of skin that an open wound must be left, and in these circumstances the best method of healing to aim at is healing by blood clot. After the operation has been finished, and the wound closed as far as possible, the remainder should be allowed to fill with blood. If the bleeding has been too freely arrested, some parts should be scratched so as to furnish the blood, care being taken not to pour fluids into the wound, otherwise the coagulation of this blood might be interfered with. Hence, when the wound has been inflicted and stitched up with the various precautions that have been described, a little bleeding should be allowed to go on, and a piece of protective which has been dipped first in 1 to 20 carbolic lotion, and then subsequently in weak sublimate solution, is placed over the open part of the wound, overlapping the skin all around. Outside this the ordinary gauze and wool dressings are applied, and the wound is left at rest for ten or fourteen days. The result is that underneath the protective the blood coagulates, and this coagulum is not further irritated; and here, of course, especial care must be taken to prevent any movement of the part. Cells very quickly penetrate into this clot, and in the manner previously described (page 206) develop into fibrous tissue, and if organisms be carefully excluded and the dressing be changed very seldom, and irritation of the surface of the clot be avoided, it will be found after a few weeks that the place of the clot has been taken by organising fibrous tissue, and that towards the surface epithelium has spread across and formed a cicatrix.

In cases where wounds are very large, however, healing by blood clot would imply a long period of time, and in such circumstances it is best to get immediate healing by means of skin-grafting. If Thiersch's method of skin-grafting be employed in the manner previously described (page 117), these grafts adhere apparently quite as readily to the freshly-cut wound as they do to the scraped surface of an ulcer, and, therefore, on the termination of an operation the wound may at once be completely covered with skin grafts in the manner already detailed.

If these wounds become septic from some accidental occurrence during or after the operation, the symptoms are somewhat the same as those where sepsis has taken place in an incised wound; but unless the wound is large, or unless some specific virus, such as that of erysipelas, has entered, they are not nearly so severe. The temperature rises to some extent and only falls gradually, while the patient complains of pain in the wound, which becomes red and swollen, and very quickly covered with granulation tissue and exudes pus. In such circumstances it is not usually necessary to do more than wash away the pus that accumulates on the surface, and continue the same sort of antiseptic dressings, the wound being skin-grafted, if large, after granulation is complete, in exactly the same manner as has been described in speaking of ulcers.

Some surgeons use iodoform very largely, both in recent and in septic wounds; but it seems to be now clearly established that iodoform as an antiseptic is quite untrustworthy, in fact, it has almost no antiseptic power. Consequently, to depend on this substance as a method of keeping bacteria out of wounds in the first instance, or of interfering with their growth in the wound should they enter, is quite an untrustworthy plan. Indeed, it has been shown in a certain number of cases that sepsis has actually been conveyed to the wound by the iodoform itself, when the vessel in which it has been contained has become contaminated with septic material, which has dried up and mixed with the iodoform, and been subsequently powdered over the surface of the wound. As an application, however, in wounds that have already become septic, iodoform is apparently of some value, and it seems from experiments that it acts more especially by breaking up the poisonous products which have been already formed by the bacteria, and that while doing so it is decomposed, and free iodine liberated, which may possibly exercise a certain inhibitory effect on the bacteric growth. Hence the use of iodoform in these cases may be of some advantage, and, provided it has been properly sterilised and has not been used in too large quantity, it can do no harm.

The too free use of this drug may lead to the phenomena of iodoform poisoning, such as an erythematous eruption, fever, rapid pulse, vomiting, and excitement or delirium.

As regards the lotions for washing out these open wounds, I should strongly dissuade from the use of carbolic acid or of strong sublimate solutions, because these can only irritate and injure the granulation layer, and thus enable bacteria to penetrate into it. If the wound must be washed out, it should be done either with some weak sublimate solution (1 to 4,000 or 1 to 6,000 is what I always use) or with boracic lotion, or sanitas, or some other non-irritating antiseptic. Where the wound has become entirely covered with granulations, the use of various antiseptic ointments sometimes answers very well, more especially boracic ointment of half the pharmacopœial strength, or eucalyptus ointment, and outside the muslin which has been spread with the ointment some antiseptic dressing such as boracic lint should be applied.

(*d*) **Wounds of mucous membrane.**—In cases where the wounds involve the mucous membrane, or have been made entirely on mucous surfaces, strict antiseptic treatment is, of course, out of the question, and here the aim must be to place obstacles in the way of the growth of the bacteria, more especially to strengthen the resistance of the body. Hence, before doing operations in these situations where there is no immediate hurry, steps should be taken as far as possible to place the patient in a good state of health. For example, in operating for cleft palate, one should see that the patient has had a good holiday, and that his general health is good; secondly, one must perform the operation as neatly as possible, and more especially avoid any bruising of the surface of the wound, or any application of antiseptic substances to it; then thirdly, the wound must be accurately united throughout the whole extent, and not merely at its mucous surfaces, and care should be taken that blood, saliva, or any septic material is thoroughly removed from the surface of the wound before the stitches are fastened. Further, as regards the stitches themselves, they should be of some soft material which will not cause irritation, which will yield to the movements of the part, and which is not porous, such as horsehair or fine catgut. Finally, the part should be kept as much at rest as possible, and great attention paid to the strength and general condition of the patient. Where union by first intention fails in such cases, the use of iodoform is very good, the part, of course, being placed as completely at rest as may be.

2. **Accidental wounds.**—Finally, we must consider now the treatment in cases where wounds which have been recently inflicted have not been made by the surgeon. Examples are afforded by suicidal wounds, wounds caused by the wheel of a vehicle, etc. In such cases we usually have not to deal with a clean-cut wound, but with a contused or lacerated one; and I shall presently speak of the characteristics and treatment of these wounds. But the great point with regard to the treatment in these cases is that dirt has almost certainly entered the wounds, and, if it be not neutralised, will give rise to sepsis; hence, the first factor in the treatment is thorough disinfection, and this is more especially imperative where the wound has been caused by the wheel of a vehicle, and where, therefore, a large amount of dirt and earth has been ground into the part. In such circumstances it is often necessary to anæsthetise the patient, to lay the wound freely open and thoroughly scrub it with a nail brush and the strong mixture (page 214) till the dirt and grease have been as completely removed as possible. In badly soiled cases I think it is best to sponge the whole surface of the wound with undiluted carbolic acid. Although this produces a minute layer of slough, still it more effectually destroys the organisms than any other method. In these wounds, also, no attempt should be made to secure union by first intention along their whole extent. Apart from the fact that the edges are contused, and will not in all probability properly unite, it would not do to stitch the wound closely together, because in spite of the most careful attempts at disinfection, one sometimes fails in attaining the object, and one must therefore always provide for free exit of discharge by means of drainage-tubes if necessary. The utmost that one should do in the way of stitches in the first instance, till the asepticity of the wound is clear, is to put in one or two so as to hold the flaps in position. This is an essential element

in the treatment of a cut head, for example, where pieces of hair are almost certain to enter, and these must be carefully removed; and even when removed with the greatest care one can never be certain that the wound will remain aseptic. Hence, in the case of a cut head, I recommend disinfection of the wound in the way I have described; but it should be left open, or, at most, supported by one or two stitches, and a large drainage-tube should be passed into it, the external dressings employed being the same as those already described.

I need not here enter into the special treatment of compound fracture, wounds of joints and large serous cavities, as these will be discussed under the proper headings. So far as asepsis is concerned, the essential point is to enlarge the wound, if necessary, so as to be able to get at all the recesses, and then to remove thoroughly any foreign bodies or dirt, and saturate the tissues with some strong antiseptic solution, more especially with a 1 to 20 carbolic acid solution.

3. **Contused wounds.**—So far I have been speaking of the treatment of incised wounds. Where the wound is not inflicted by the surgeon, it most usually, however, is not a clean incised wound, but either a contused or a lacerated wound.

In a contused wound the skin has given way over a contused part, and the condition of the wound is such that the edges are very much bruised and infiltrated with blood, and if excessive inflammation occur in these edges sloughing is very apt to take place. Besides, if the wound become septic, the organisms have a large amount of weak tissue in which they can grow luxuriantly, and very serious suppuration and general septic effects will probably result. Hence, in cases of contused wounds, it is particularly desirable to asepticise the wound thoroughly, and if the part be seen soon after the accident, as a rule there is no great difficulty in doing so. If the wound in the skin be small, it should be enlarged, so that the whole contused subcutaneous area is exposed, and, the edges being held aside, the injured tissues are thoroughly cleansed and scrubbed with strong antiseptic solutions, more especially with a 1 to 20 carbolic acid solution, and also with sublimate solutions, the skin around being very carefully disinfected in the manner previously described (page 213). No stitches should be employed, but the wound should be left freely open. In fact, in some cases it is well to stuff the wound with gauze for a few days. If after that time it be found that asepsis has been attained, and that the edges of the skin have retained their vitality, one may then insert a few stitches with a view of getting more rapid healing.

When suppuration occurs in contused wounds, and where the wound is extensive, probably the best method of treatment is constant irrigation with some weak antiseptic solution. Irrigation acts essentially by washing away the albuminous fluids as soon as they are poured out from the wound, and thus removes the pabulum on which the micro-organisms would live; and if a small amount of some antiseptic be added to the water employed for the irrigation, a certain inhibitory effect will be produced as regards the growth of these organisms. Irrigation is carried out in various ways, and as a matter of fact it is almost only applicable to injuries of the extremities. Where it is used the bed should be covered with mackintosh, which is carefully fastened round the upper part of the affected extremity, so as to prevent the fluids from running into the bed, and, if it be possible, the limb should be laid in a trough in order to catch the fluid. Where this is not possible, one must simply arrange the mackintosh so that the stream of fluid is conducted into a vessel at the side of the bed. Various irrigators are employed, probably the simplest being a piece of worsted thread, one end being placed in a vessel containing the solution to be employed, and the other end lying on the centre of the wound. By capillary attraction, there is a constant, though slow, stream running into the wound. Where more rapid irrigation is required, this must be carried out by means of a proper irrigator, with tubing and a nozzle with stopcock ; but in most cases the worsted thread will supply enough fluid.

In employing irrigation, the parts around should be thoroughly soaked with vaseline or other fatty substance, so as to prevent the skin from becoming sodden, and the fluid must not be allowed to drop on to the wound. Whether it be the worsted thread or the nozzle of an irrigator, the supply of the fluid must be in actual contact with the surface, there being nothing more painful than a continual dropping of fluid on to a wound. As regards the fluid to be employed for irrigation, it should be about the temperature of the body, and contain some mild antiseptic, more especially some antiseptic which does not coagulate albumen. Boracic acid, for example, is an excellent material for irrigation, weak iodine water has also been much employed, as also weak solutions of perchloride of iron. Sanitas and permanganate of potash are, next to boracic acid, probably the best materials. The irrigation must be interrupted after a few hours, the part in the interval being wrapped up in wet antiseptic gauze, with mackintosh outside. Continuous irrigation renders the wound too œdematous and interferes with the healing process. As soon as the wound has become covered with granulations, the irrigation should be intermitted and recourse had to various antiseptic ointments or other dressings which have already been described in connection with ulcers and wounds (pages 115 and 225).

4. **Lacerated wounds.**—By a lacerated wound we mean one which has been inflicted by a blunt instrument which has torn the skin rather than contused it. Very often these wounds are produced by machinery in movement, and the skin is often torn off for a very considerable distance. The result is that if the wound be left to nature, violent inflammation and suppuration occur in the part, and extensive portions of these torn tissues slough, and here again it is very essential that the wound should be made aseptic if possible. This is done in the same way as has been previously

described, by thoroughly scrubbing the parts with a nail-brush and strong antiseptic solution, the patient naturally being under chloroform. In lacerated wounds it is very often more difficult to complete the disinfection, owing to the fact that the instrument which has produced them may have been covered with grease or dirt. No attempt should be made to stitch up a lacerated wound. If an extensive flap of skin has been torn off, it may be held somewhat in position by one or two stitches, but union by first intention is quite out of the question, and the wound should therefore be left open to permit a free escape of the discharge. Where the wound is aseptic, healing occurs usually by blood clot; but where the skin has been extensively torn off, it is well—when once the clot has become partially organised—to remove the half-organised surface and cover the part with skin grafts; otherwise a long time may elapse before healing is complete, and excessive contraction of the neighbouring parts may take place. Where the attempt at asepsis has failed, constant irrigation is again the most favourable method of treatment.

DISEASES OF CICATRICES.

While in cases where union by first intention has taken place the cicatrix is usually quite satisfactory, this is not by any means the case where cicatrices have followed wounds in which the edges have not come together. In such cases we may have (1) weak cicatrices which are constantly tending to break down, (2) painful cicatrices, (3) contracting cicatrices, or (4) a cheloid condition, the latter also occurring in union by first intention.

1. **Weak cicatrix.**—Where a cicatrix constantly tends to *break down*, the cause is generally to be found in the large size of the original sore and in the incomplete contraction which has taken place during the healing processes. Hence these cicatrices are generally found after very extensive burns, towards the centre of the part injured, or where the scar is attached to bone, etc.; and the treatment in these cases must consist in the first instance in the prophylactic treatment, namely, the avoidance of these cicatrices, or if they have occurred, one must attempt to remedy them by permitting a proper amount of contraction.

Where we have a large wound we may feel pretty certain that if the healing of the wound be left to nature, the centre of the scar will be weak, and therefore in these large wounds it is well before healing has progressed to any great extent to employ skin grafting, even although the wound affects the trunk or the upper extremity. By doing so a much sounder scar will be obtained and much less contraction will be necessary. Where, however, we have to do with an already-formed cicatrix which is constantly giving way, if the condition be due to inefficient contraction one must attempt to remedy this by freeing the scar by tenotomy, by plastic operations, etc. As a rule, skin grafting at this period does not produce a completely satisfactory result, and probably the best method is to cut away the whole of the weak part of the scar and then to turn a flap over it from some convenient part in the neighbourhood. Thus where the scar is on the side of the chest, one may fasten the arm to the side and turn a flap of skin from the arm on to the centre of the scar. In such a case a long flap would be required, and if it were detached at once at one side it would very probably slough, hence the method which has been advocated, more especially by Mr. Croft, is to be recommended. He marks out a flap of the proper length and makes lateral incisions of the necessary breadth, undermines the flap of skin, and then introduces between this flap and the deeper tissues a piece of protective to prevent adhesion, and leaves the flap attached at each end till the under surface has completely granulated. He then detaches one end and turns the flap on to the granulating surface of the sore, and after ten days or a fortnight, when adhesion has thoroughly occurred, the other end is also divided and the flap remains *in situ*.

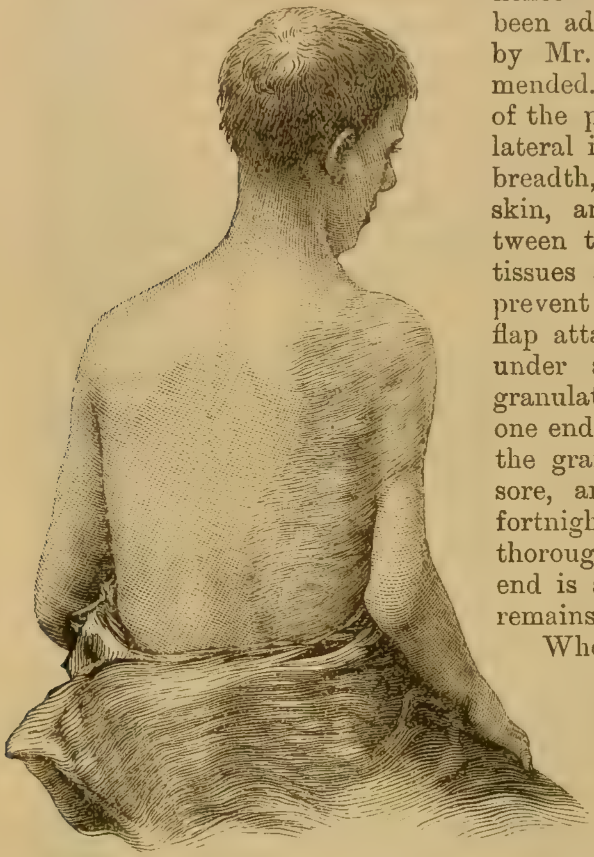

Fig. 41.—Arm bound to Side by Contraction of a Sore after extensive Burn.

Where the weakness of the scar is due to adhesion to the bones, one may also attempt to separate the scar from the bone, or even to excise the scar and turn in the flap from the side. In very bad cases, especially where contraction is present as well, it may be necessary to remove the subjacent portion of the bone in order to permit the scar to contract soundly. For example, where we have such a condition over the elbow joint, cure will be obtained by excising the elbow; and by such a method of treatment two advantages will be gained, not merely that a sound scar will result with the possibility of contraction, but that a movable elbow will be obtained. In the case of the leg it has been found necessary to remove an inch or two of the tibia and fibia, and thus shorten the leg in order to obtain healing of the wound; but as I have previously said, these troubles can usually be entirely avoided if during the course of the healing of the wound skin grafting according to Thiersch's method is employed (page 117).

2. **Painful cicatrix.**—Where we have to do with painful cicatrices, the cause is generally the implication of the nerves in the contracting fibrous tissue and the consequent occurrence of a certain amount of neuritis; and the best remedy is to dissect away the scar altogether and employ immediate skin grafting.

3. **Contracting cicatrix.**—Where we have contracting cicatrices, the cicatrix should be divided and the gap either closed by plastic operation, or much better by Thiersch's skin grafts (Figs. 41 and 42).

4. **Cheloid.**—A cheloid condition of a scar is one in which the scar begins to thicken, the thickening commencing at the point where it joins the skin. After a time these scars form irregular and unsightly hard projections, which often show dilated veins on the surface which are not very vascular and which creak under the scalpel. The surface is smooth and glistening and raised, and from the major mass nodules or claw-like processes may extend over the adjacent integument. The skin around becomes pulled on, and in this way great deformity results. This condition of cheloid seems to be dependent on some constitutional state, and is supposed to be more especially associated with tuberculous disease. It is often noticed in wounds which have been irritated unduly. Certain it is that in some persons every scar, even the minutest, tends to thicken and become cheloid in this way. These scars do not, as a rule, cause any pain, but they are heavy and unsightly, and are very apt to ulcerate and bleed. They are often the seat of great itching and there may be neuralgic pains in them. On examining the structure microscopically, there is found to be marked proliferation of the fibrous tissue, and in the growing parts there are numerous cells. They

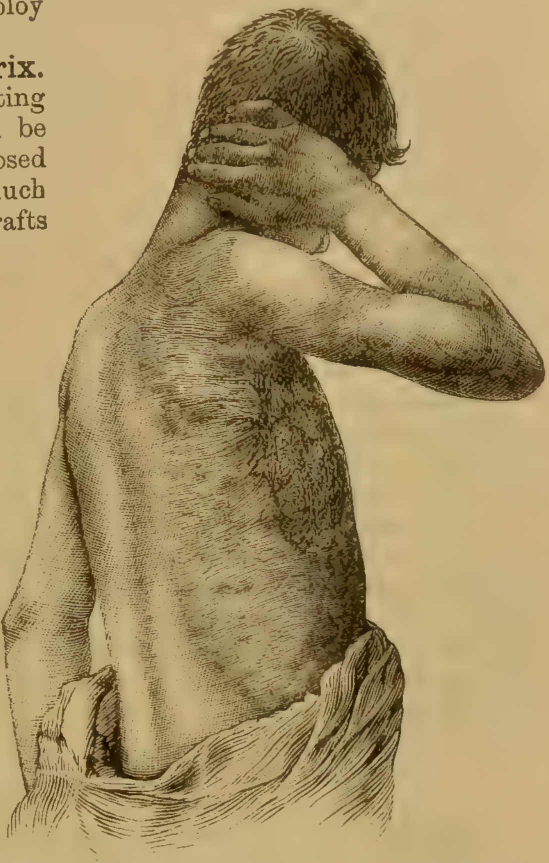

Fig. 42.—Same Case as Fig. 41, after the Arm had been separated from the Side and the raw Surfaces made to heal at once by Skin-grafting.

do not usually penetrate below the skin, keeping where they affect the skin itself to the surface of the dermis, leaving the papillary layer intact.

As regards the treatment, the condition is an excessively difficult one to get rid of. At first sight one is often tempted to cut out these cheloid scars; but as a rule the condition is simply reproduced in the scar so formed, and not only along the line of incision, but also in the stitch tracts, and the resulting deformity may be equally bad with the original. Nevertheless, I think that in some cases where one has to do with a large broad cheloid, and where it is possible to bring the edges of the skin together, one may very much improve matters, even although one does substitute a linear cheloid scar for the original mass; and if an attempt be made to dissect out the cheloid with the view of getting a linear cicatrix, one should avoid the use of stitches altogether unless there is great tension on the parts. The edges of the skin may be approximated by buried stitches as described under wound treatment, and the approximation completed by the use of strips of gauze fixed with collodion which bring the actual epidermic surfaces into contact (page 219). Where, however, it is not desirable to dissect out the cheloid scar, the essential point in the treatment is to interfere with the blood supplied to the part, and this is done by employing pressure in various ways. In some cases improvement is obtained by painting the surface of the cheloid with ordinary collodion. As the collodion dries it contracts and compresses the parts beneath. The disadvantage is that in some cases it leads to ulceration. Various plasters have also been applied with the view of causing pressure, and formerly attempts were made to destroy the cheloid by means of scarifications or by the use of the actual cautery; but as a rule, these attempts have been given up. At the same time, the use of cod-liver oil internally seems to be of considerable disadvantage.

CONTUSIONS.

Effects.—By contusion one means the result of a severe blow on the soft parts without a breach of the surface. The effect of the blow is to bruise and tear the cellular and muscular tissues, and very probably the skin itself, leading to hæmorrhage into the torn parts. Where the skin is unbroken, and the patient is healthy, this deep-seated wound generally recovers without any suppuration or other trouble, healing occurring by blood clot just as in the open wound; but in some cases the contusion of the skin is so severe as to lead to its ultimate sloughing, and in that instance, if the part which has been contused has not been rendered aseptic previously, we may have a suppurating wound with the separation of sloughs of cellular and muscular tissue, which may be a long time in healing, and which may even be a considerable source of danger to the patient. In some cases the blood which is poured out into the cellular tissue after a contusion is large in amount, and leads to the formation of a distinct tumour (*hæmatoma*). This blood may not coagulate properly, or, if it does, may not be completely organised, the result being that around the exterior a layer of fibrous tissue is formed, in the interior of which is clear serum; in other words, we have a *blood cyst* which may remain indefinitely, or even may become inflamed and suppurate. As a rule, subcutaneous bruises do not suppurate, unless in drunkards or unhealthy persons.

Symptoms.—As to the characters of a contusion, the patient generally suffers a good deal of pain in the first instance from the bruising of the nerves, which, however, passes off in the course of twenty-four hours. The part may become swollen even to a very large extent, the swelling being due partly to hæmorrhage from the torn vessels and partly to œdema from the early stages of inflammation which occur around the contused parts. In the course of a few hours, if the contusion be superficial, the blood stains the skin, and we have a dark colour of the part; but very shortly the blood pigment undergoes alterations, so that the skin changes from a black to a blue or yellowish colour before it ultimately regains its normal appearance. As the result of the œdematous condition of the tissues this blood pigment is also carried for a considerable distance through them, and thus the yellow and green stain is generally much more extensive than the original seat of injury, and is usually more marked towards the most dependent parts, owing to the gravitation of the œdematous fluid towards these parts.

Where much blood has been poured out we have the condition known as hæmatoma, and where a blood cyst is about to form we find around the margin of the collection of blood a hard rim, while the interior is quite soft. When these hæmatomata are situated on the skull this peculiar condition has often given rise to the mistaken view that there was a fracture of the skull with depression, because the finger in passing over the swelling feels the hard edge of what is apparently the bone, and then inside that edge passes much deeper, apparently into a cavity. This condition is more especially evident where the bruise has affected the periosteum, and where, therefore, the blood is caught in the meshes of the periosteum and does not diffuse itself so readily as in an ordinary wound.

Treatment.—As regards the treatment of contusion, the most rapid method of getting rid of the effused blood is by gentle massage, carried on in the first instance very lightly and only for a few minutes, but gradually increasing both in time and in vigour as absorption goes on. In such circumstances the parts will generally be well in the course of a week or ten days. Of course, this treatment is not necessary unless in large contusions, or where the contusion affects parts—more especially joints—where stiffness may subsequently result if they be kept too long at rest. In other circumstances the best treatment is in the first instance the employment of cold, with the view of checking the subcutaneous hæmorrhage, and afterwards rest and pressure with a mass of cotton-wool and a bandage, so as to lead to absorption. Formerly one was told not to interfere by operation with hæmatomata; but now, with the protection of asepsis, there is no object in losing time by leaving large effusions of blood to become absorbed or converted into blood cysts, and therefore, where we have to do with a large effusion of blood which has not properly coagulated, the best treatment is within a day or two, after asepticising the skin, to make an incision into the part, turn out the fluid and soft clots, and insert a drainage-tube. The same treatment will usually be sufficient with blood cysts, though in some cases it is perhaps more rapid and more advisable to cut them out altogether.

XI. MILITARY SURGERY.

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The wounds met with in warfare may be classed as follows :—

I. Gunshot wounds: (1) From massive projectiles; (2) from musketry and rifle and pistol fire.

II. Bayonet and sword wounds.

III. Arrow wounds.

I. GUNSHOT WOUNDS.

These include all those resulting from the action of "missiles that have been projected by force derived from explosion" (Longmore). They may arise *directly* from shell or bullet, or *indirectly* from objects such as pieces of stone set in motion by the above missiles. They are in their nature contused and lacerated wounds. In civil life we have chiefly to do with injuries produced by "shot" or pistol bullets. At a very short range, before the shot has had time to scatter, a severe lacerated wound may be caused. Pistol bullets cause a wound possessing more or less the character of that caused by a rifle bullet, though less damaging in their effects.

Variety of bullet.—The injuries produced by musketry fire vary with the nature of the missile. We will first consider the difference in the effects caused by *round* and *conoidal* bullets. As regards (1) *shape*, the conoidal bullet acts chiefly as a wedge, having great penetrative and splitting powers, whilst the round bullet causes chiefly local effects. The round bullet also frequently lodges, whereas the pointed shape of the conoidal bullet tends to prevent this result. In its (2) *smaller dimensions*, the conoidal affects a narrower zone of tissue, and foreign bodies are less likely to be carried into the wound. The round bullet has greater stopping-power. In respect to (3) *velocity*, the round bullet quickly loses its initial velocity after leaving the barrel, whilst that of the conoidal is retained; and inasmuch as the destructive power of a bullet is increased proportionately with the square of the increase of its velocity, greater disturbance of the parts traversed is caused by the conoidal missile. But although the destructive action is more intense, the area laterally affected is more limited, as Kirker has pointed out. Hence, the wound caused by the conoidal will be narrower, have more defined walls, but contain more thoroughly disintegrated tissue. Finally, with regard to (4) *rotation*, the round bullet revolves on an axis at right angles to its line of flight, and this can be easily deflected by any resistance in its course; whereas the conoidal bullet, rotating on an axis identical with its line of flight, penetrates the tissues more easily, and so will destroy them in a more limited degree than the round. Hence, it would seem that the wound of the conoidal being narrower, containing less of the foreign substance, and being more sharply defined from the surrounding tissues, has a greater tendency to heal. The exception occurs where the bones have been much splintered.

The round bullet, on meeting with a hard-resisting substance, such as the compact tissue of the thigh-bone, becomes *splintered* and altered in shape. The expansion undergone by the Snider bullet and its splintering were so great that it was practically an explosive one. With the Martini-Henry missile, the bullet being composed of lead mixed with tin, the deformity on contact was much less.

At the present time every important Power is armed with the magazine rifle. The bullets are smaller, harder, and lighter. In the British Army the Martini-Henry, with its calibre of ·450 inch, has been superseded by the Lee-Metford rifle, with a bullet of a calibre of ·303 inch (Fig. 44). The following table shows the more important characters of the various bullets used by the European Powers at the present time:—

Power.	*Weight.*	*Calibre.*	*Composition.*	*Velocity (Muzzle).*
Great Britain				
a Snider ..	480 gs.	0·577″	Pure lead	1,240 ft. per sec.
b Martini-Henry	480 gs.	0·450″	Lead and tin	1,315 ft. per sec.
c Lee-Metford ...	217 gs.	0·303″	A central core of lead and antimony, with a covering of copper and nickel	2,000 ft. per sec.
Austria	242 gs.	0·315″	Covering of steel	1,968 ft. per sec.
France	216 gs.	0·315″	Covering of copper, nickel, and zinc	2,073 ft. per sec.
Germany	223 gs.	0·311″	Covering of steel	2,034 ft. per sec.
Russia	215 gs.	0·300″	Covering of nickel	2,000 ft. per sec.

As yet, the new magazine bullet has only been used in the late Chilian campaign. The consideration of the wounds inflicted by it will be considered later, but meanwhile its main characteristics would seem to be—less stopping-power and greater penetration. Fig. 43 shows the shapes of the bullets above named, and includes also the Snider, Needle-gun, Chassepot, and Enfield bullets. Fig. 44 depicts the Lee-Metford bullet.

Number of wounds made by bullets.—There may be only *one wound* in cases where the bullet has lodged; or *two* when it has escaped; or the wounds may be *multiple*, the bullet splitting against a bone, and thus causing more than one aperture of exit, or one bullet may pass through more than one part of the body. More rarely two projectiles have caused a single wound. The aperture of exit is more often multiple than that of entrance. With the magazine bullet the number of wounds generally will be greater.

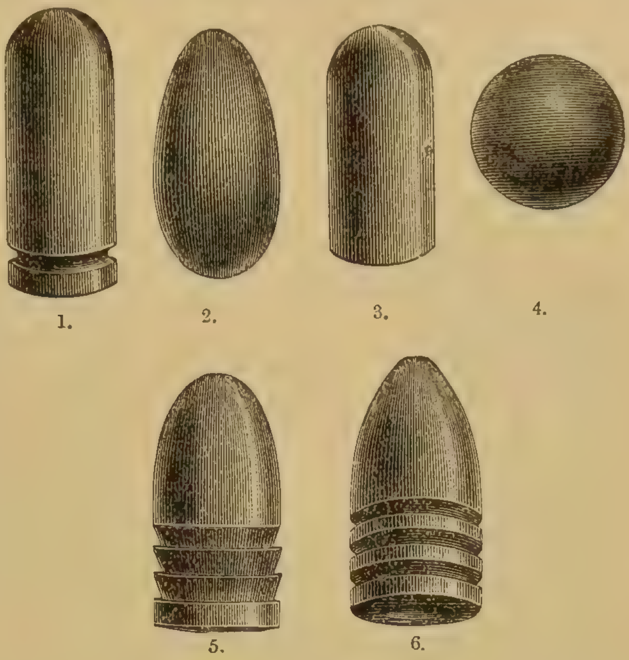

Fig. 43.—Bullets
1, Martini-Henry; 2, Needle gun; 3, Chassepot; 4, Round bullet; 5, Snider; 6, Enfield.

Primary general symptoms.—*Pain* may in some cases not be experienced at all. The wounded man may be first made aware of his wound by the hæmorrhage from the part. Again, no pain may be felt at the moment of impact, but shortly afterwards the most excruciating anguish comes on, especially if the bullet have lodged under the skin, and is stretching it. Again, if nerve trunks be injured, the pain is especially great. The gravity of the wound also has an influence; an extensive wound of the thigh is markedly painful. The smaller the missile and the greater its velocity, the less is the pain. The soft parts when wounded are less painful than the bones. In character the pain has been likened to that caused by a blow with a heavy stick, or to that of an electric shock. Race and nationality appear to influence its intensity. Mussulmans would seem to be comparatively insensitive. Fischer, in the Bohemian War, found the Italians and Poles most susceptible, the Hungarians least, and the Germans midway in this respect. Finally, pain may be entirely absent from the aperture of entrance, and only felt at that of exit, leading the wounded man to imagine he has only been struck at the latter spot.

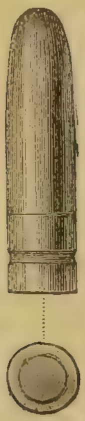

Fig. 44.—Lee-Metford Bullet.

Shock in some degree almost always accompanies gunshot wounds. It may be general or local. General shock is especially present in wounds from massive projectiles, in abdominal wounds, or where a limb of the body has been carried away. The severity of the shock and its long continuance indicate that some important organ has been wounded. It occurs less frequently where the soft parts only have been wounded. The usual symptoms are present. A pale cold skin, sunken eye, cold sweats, great flaccidity of the integument, frequent, small and scarcely perceptible pulse, with quickened superficial breathing. There is marked apathy, but consciousness is retained. Should death not supervene, the above state passes into that of reaction. The eyes lose their sunken state, the face flushes, the temperature rises, the pulse becomes stronger, and often delirium sets in.

In local shock the wounded part only is affected. The part in question is cold and pallid, without sensation or motion, or possibly abnormal sensations such as formication may be felt. This local shock may either become general or gradually disappear.

As regards prognosis, the loss of temperature is the most important feature. Rédard, with reference to this, states that every wounded man brought to hospital with a temperature of 96° F. will die, according to his experience. Again, if reaction does not come on by the end of the fourth hour, and in direct ratio to the previous fall, the condition is one of jeopardy.

Syncope, resulting immediately on receipt of a wound, occurs especially where the large nervous trunks are implicated. Psychical effects may also cause fainting. *Thirst* is nearly always intensely felt, being produced by the excitement undergone, by fatigue, dust and heat. Lastly, a condition sometimes observed after instantaneous death from gunshot wounds is that of *instantaneous rigor mortis.* The rationale of this occurrence is not yet determined. It is said to occur especially after wounds of the cerebro-spinal axis, and after death from hæmorrhage. In this condition the attitude of the soldier at the moment of death is preserved even against the force of gravity. It cannot be due to cold, as it has occurred when the temperature has been high.

Local symptoms.—*Primary hæmorrhage* arises from one of the large vessels being cut across, or it may be capillary. It has been comparatively rarely met with as a dangerous factor on the field of battle, the wounds hitherto inflicted having been seldom clean cut, but being generally contused or lacerated. Death, however, from primary hæmorrhage is likely to happen more frequently in the future from the magazine bullet, as a cleaner and more incised wound is made. Wounds of the main vessels may be fatal immediately, but are not necessarily so, for the hæmorrhage may be temporarily arrested by the retraction and curling-up of the inner coats of the artery. Thus a wound cutting completely across a vessel is less dangerous than a partial wound of the vessel wall. Where the hæmorrhage is capillary, it will ooze slowly, staining the surrounding tissues, and can usually be arrested by slight pressure.

Condition of the tissues.—With regard to the *soft parts*, the skin may be (1) *contused*. It is livid, relaxed, and often colder than the surrounding parts. The surgeon should in these cases always be on the look-out for extensive damage in the deeper parts. After the preliminary stage of pallor and coldness, redness, heat, and swelling supervene. The pain increases, and there is much subcutaneous effusion. Eventually the skin may slough.

The wound may be (2) of a *lacerated* character, superficial or deep. The sides and bottom of the wound are formed of the *débris* of the contused tissues. The pain is often severe and paroxysmal.

(3) *Canal-shaped wounds.*—With spent balls there may be only an aperture of entrance. Portions of the dress may be carried into the wound, or the wound may be of the "seton" character. With the old round bullet the aperture of exit was sharply distinguishable from that of entrance, but with the conoidal the differences have in a great measure disappeared, and with the magazine bullet are practically non-existent. With the round bullet in its entirety, striking the body at right angles, *the aperture of entrance* was rounded, inverted, ecchymosed, and of smaller diameter than that of the missile. Generally, also, there was a slight loss of substance in the integument. The *aperture of exit* was larger, more irregular, everted, and with greater loss of substance. The wound of exit, as a rule, heals first. With the magazine bullet both the apertures are much more cleanly cut. As regards the track of the bullets, the conoidal causes one with more cleanly-cut walls than the round, and is often so small as to be closed by the coming together of its sides, whilst that of the round bullet remains patent.

The wound in the *fasciæ* may be either a mere slit, or there may be loss of substance. Fasciæ resist the onward passage of the bullet; and where its velocity is not great, can turn it aside, or even arrest it. The sides of the wound may so completely fall together that its channel cannot be made out. The great importance of wounds of the fasciæ lies in the influence exerted by them in cases where infective inflammation has supervened. Conoidal bullets separate merely the crossing fibres of the strong fasciæ, so that the fascial openings have a smaller diameter than the general track, and thus any discharge of pus, should it form, will be interfered with.

Tendons are most frequently pushed aside during the passage of the ball, returning to their normal place afterwards, or they may be torn from their insertion. Large tendons, such as the tendo Achillis, have been perforated, or their edges may be notched. Dr. Chenu, in the war of 1870, described cases of arrest, and even of splitting of small bullets by tendons.

The *muscles* may be lacerated and contused, and the seat of much extravasated blood. The destruction caused by the missile is ordinarily limited to the neighbourhood of impact. Where a ball has channelled through several muscles, the track will often not be continuous. It is larger in diameter than the ball, irregular in outline, and contains a sanguineous lacerated pulp; or the canal may be extremely narrow, and difficult to follow. Very rarely is a muscle completely divided. If it be so, the ends retract for some distance.

As regards the *nerves*, the main trunks, frequently from their deep position and lax attachment, escape injury. Their branches are, however, more liable to be damaged. They may be contused and crushed, cut across or perforated (*e.g.* the sciatic). Various symptoms due to loss of motion and sensation are present, more especially the former, sensation being less influenced from the collateral communications that are naturally present. As sequelæ, trophic changes may occur, atrophy of muscle, lividity and gangrene of the skin, and even necrosis of bone. The neuritis and sclerosis of the nerve at the focus of injury may extend far beyond it, even to the great centres.

The *blood-vessels* may be either simply contused or their walls completely or partially divided. With the old round bullet arteries had more chance of evading the missile, on account of its less velocity and penetrating power. Owing to the smaller size, pointed end, and greater velocity of the magazine bullet, arteries in future wars will less frequently escape. Delorme holds that in the immense majority of cases the bullet contuses or cuts out a piece of the vessel on meeting it. Gahde states that arterial lesions are less common where only the soft parts are wounded than where fractures have occurred.

Where the vessels are simply contused, the projectile (having somewhat feeble velocity) causes an effusion of blood in the sheath of the vessel: this infiltration may be subsequently absorbed. If a main artery be affected, gangrene from thrombosis may occur, especially where the vein is also wounded, or secondary hæmorrhage from ulceration in the vessel-wall may ensue. Where the trunk of the vessel is wounded as well as contused it may be the seat of a lateral wound, or it may be completely cut across. The danger of fatal hæmorrhage is greater with the former. False aneurysm may occur at the seat of a wound in an artery, although shot wounds less frequently give rise to aneurysm than punctured wounds. Arterio-venous aneurysm may likewise result. In cases of wound of the large veins of the neck, the entrance of air into the veins has occurred: the liability to this accident must be borne in mind, when the surgeon has to extract a bullet in this neighbourhood. The diagnosis of a wound of a vessel is in some cases rendered easy by noting the site, the absence of pulsation in the vessel below, and the pulsatile character of the hæmorrhage; but if the wound be small and the tissues greatly infiltrated, the diagnosis may be difficult. Wahl notes as an important sign of only partial wound of the wall, namely, a buzzing noise at the seat of wound, synchronous with the pulse: this is absent if the vessel be completely cut across.

Injuries of bones and joints.—The injuries received as the result of the impact of a bullet on the *bones* are influenced chiefly by the form of the missile, by the structure of the bone, and by the angle of impact. The conoidal bullet is responsible for a large amount of fissuring caused by its wedge action: with reference to this fissuring, Kirker has pointed out that the periosteal investment of the fragments is not much disturbed. With the round bullet, on the contrary, there is little fissuring, but great comminution. With regard to the structure of the bone, the effects on the long, short, and flat bones must be considered respectively.

The *long bones* may be (1) *simply contused:* here the velocity of the bullet is slow; the soft parts may be traversed, or simply bruised without any open wound being present. If there be only a wound of entrance, the bullet may lodge or fall out. The periosteum may be torn or lacerated, with effused blood; blood may be infiltrated into the bony wall or medulla. Where there is no open wound the diagnosis is difficult; it rests chiefly on the continual presence of great local boring pain, the swelling due to the periosteal effusion and the immobility of the part. The sequelæ may consist in local necrosis, or in septic inflammatory processes, starting from the periosteum or medullary cavities; or in healing with thickening of the bone and some temporary hindrance to free movement.

(2) Partial or complete *fracture* may be caused. The partial fractures have been well described by Longmore: they comprise (*a*) the "*gutter*" fracture, where the bone is furrowed. This occurs chiefly near the ends of the long bones in the neighbourhood of the spongy tissue. Should no sepsis occur, healing takes place after exfoliation of the crushed parts, leaving a depression. (*b*) A portion of the bone may be detached, as a *splinter.* Crests of the bones and projecting surfaces are chiefly affected. Their relation to the periosteum is important as regards prognosis. After experimenting with the Martini-Henry bullet, Longmore found that a larger proportion of fragments at the part struck retained their periosteal connections when this bullet was used, than was the case with the Enfield bullet; long fragments detached by fissuring were held in close apposition by the periosteum. The importance of this connection is manifest. Chauvel points out that at a close range the bone is splintered in all directions, the splinters being quite free and deprived of all periosteum; where the distance is greater and the velocity less, the splintering is less and the fragments are held together by periosteum. (*c*) The bone may be completely *perforated*, especially in the neighbourhood of an epiphysis; from the perforation, fissures may extend in all directions. Fischer states the canal thus made may remain permanently. (*d*) *The external table may be driven into the cancellated structure.* Other injuries included amongst "partial fractures" are where the bone is (*e*) simply *perforated*, the bullet lodging or subsequently falling out. Such cases may cause great difficulty in diagnosis, as in the well-known example in Garibaldi, where the presence of the bullet was only ascertained by the porcelain probe of Nélaton. A small bullet may also, after penetration into the medullary cavity, be subsequently displaced upwards or downwards. The ball may, after penetration, become encysted, as

in a case occurring in the Afghan War, where the bullet remained without symptoms in the upper maxilla; or suppuration and necrosis may occur, or an acute osteomyelitis may carry off the patient. Lastly, the bone may be (*f*) *fissured.* Longmore here describes "the indirect fissured fracture," where the fissure occurs at some distance from the point struck, an interval of sound bone existing between the seat of known fracture and this indirect fissure.

Complete fractures are either transverse or oblique, and accompanied or not by splinters. In the latter case the bullet has struck the bone when spent, and may be found lying against the bone, or may have fallen out. Fractures with splinters are much more common. The main characteristics of these bullet fractures are shown to lie in the greater comminution of bone, the more numerous splinters, and the greater loss of bone substance.

With regard to the *short bones*, with which may be included the spongy ends of the long bones, we find the cases divided into two main classes, according as the neighbouring joint is opened or not. Where the joint is not opened, the bone may be (1) *simply contused*, the bullet striking it with feeble velocity. This is less likely to happen with the modern bullet. Accompanying it is effusion of blood either subperiosteal or into the cancellous tissue. Healing occurs after absorption of this blood, or after a subsequent osteitis. (2) *Furrowed wounds* may occur in all degrees, suppuration being especially likely to complicate the case. (3) *Penetrating wounds.* The round bullet, with its velocity expended, frequently caused this form of injury, the bullet being partly or wholly embedded, numerous fissures extending up to the articular cartilage.

The bone may be (4) *perforated.* This commonly occurs with the conoidal bullet. The aperture of entrance is depressed and surrounded by fissures, which also extend from the sides of the channel; the orifice may be embedded in splinters. (5) Lastly, osseous eminences may be *broken off*, as in the case of the great trochanter.

Lesions involving the opening of the joint are far more serious, from the danger of subsequent purulent arthritis. The ends of the bones entering into the formation of the joint may be shattered into fragments, the synovial cavity filled with blood, or in a less severe case fissures may extend into the joint from the point struck. If strict antiseptic healing can be ensured, a useful joint may result. Formerly the usual termination was in suppurative arthritis, the joint becoming disorganised, and death resulting from pyæmia. Between these favourable and unfavourable terminations, healing occurs with various degrees of ankylosis, or after long months associated with exfoliation of bone.

In contrasting the effects of bullet injury on the diaphysis and epiphysis, we find, as a rule, the nearer the latter the bone is struck the less is the shattering effect.

Lastly, as regards the *flat bones.* Here the more brittle the bone the greater is the injury, whilst the greater the velocity and the smaller the calibre of the bullet the smaller and cleaner is the wound. The injuries may be (1) *simple contusions,* with blood infiltrated into the diploë or under the periosteum ; the blood may then become absorbed, or where antisepsis is not attained, suppurative periostitis and osteitis follow with exfoliation. In the cranium abscess may form between the dura mater and bone. (2) With *fractures* only one table, the external, may be affected, where the ball has struck obliquely. Cases have been described where only the internal table has been fractured. Other forms of lesion are fissures complicating the fractures, separation of the sutures, furrowed wounds of the bones, lodgment after penetration, and perforation. Lastly, Bergmann has described cases of indirect fractures. At Plevna he met with six cases of fracture of the bone, which were held to be caused by the transmission of vibration from the part struck.

A word may be said with regard to gunshot injuries of the clavicle. These are especially dangerous from their liability to complicating lesions of the neighbouring large vessels, the subclavian and carotid, of the vagus nerve and brachial plexus, of the pleura and lung.

Among the *complications* of bone gunshot-injuries are to be noticed *fat embolism* and *thrombosis of the main veins.* In the first, the medullary fat is dislocated from its habitat and carried into the vessels, and thence it may reach the brain, lungs, or liver. If the emboli be large, death may ensue, with all the signs of apoplexy, or of pulmonary embolism.

With regard to the *sequelæ* resulting from inflammation of the affected bone, all gradations are met with from simple localised sequestra to acute infectious osteomyelitis. The latter is ushered in by repeated rigors and high temperature. The soft parts covering the affected bone are deeply swollen, and severe pain ensues. The discharge becomes foul; if the bone be visible the periosteum is seen swollen and detached. Pyæmia and septicæmia quickly ensue. As the affection is due either to a specific organism or to the decomposition of the wound discharges, this sequela should rarely occur at the present day.

With regard to the *joint* injuries, mention has already been made of some of them. Injuries of the joints may be either penetrating or the reverse. In the latter, the contusion of the soft parts overlying the joint causes a secondary synovitis, or a fracture in the bone may run up to but not into the joint. Contour wounds running round the joint, but without opening it, have been described. Perforations of the capsules of the knee and elbow joints, without simultaneous injury of the bones of these joints, have been described by Fischer, Langenbeck, and Bergmann. The diagnosis of a wound of the joint is tolerably easy with such joints as the elbow, but may be extremely difficult in the hip. A probe should never be used. Escape of synovia where present will, of course, indicate the lesion, but such escape is rare. The chief indications of the injury lie in the line of direction apparently taken by the missile, as shown by the apertures of entrance and exit, the presence of crepitus, and the abolition of the functions of the joint.

Effects of the magazine bullet.—Since the last great war in Europe all the important Powers have been re-armed with a new rifle, in which missiles of a greatly-reduced calibre are fired far more rapidly than formerly. The effects of this new bullet have been gathered partly from experiment and partly from experience in actual fighting. The results from experiment contributed by Marsh, Habart, Brun, Delorme, and others show the following. The new bullet is characterised by its smallness, hardness, lightness, pointedness, high velocity, and external cuirass (Fig. 44). As regards weight, one Martini-Henry weighs 46 grains more than two Lee-Metford bullets, and therefore the latter possess less stopping-power. As the human frame is not a solid resisting body, this decreased weight is not compensated for by the increased velocity. On penetrating the body the track is so narrow that practically no destruction of tissue remains in its wake, the path being cleaner and less lacerated. Such a bullet may pass between two contiguous bones without touching either. The number of wounded will be increased, as one missile can pass through a second or a third man, owing to its hard external cuirass. All splintering of the missile is abolished, such as formerly took place with the Snider, and to a less extent with the Martini-Henry. Thus, so long as the bullet meets with no bone in its course, the wounds are comparatively simple. Should a hard compact bone be struck, the latter will be shattered, whilst cancellous tissue will be simply perforated without much further injury. There will be more deaths from hæmorrhage, the vessels being more cleanly cut. From the lower trajectory of the missile, wounds of the skull may be more frequent. Finally, bullets will not ordinarily lodge, and thus a source of anxiety will be removed from the surgeon. The number of recoveries will be greater, the wounds being more cleanly cut and less septic.

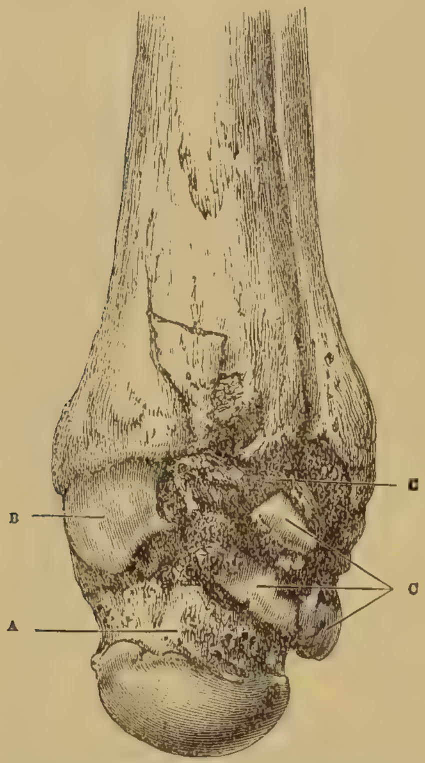

Fig. 45.—Injury caused by the Lee-Metford Bullet.

A, Head of astragalus: B, tibial surface of astragalus; C, fragments of astragalus. (*British Med. Journal*, June, 1894.)

At distances between 400 and 1,200 metres the injuries to the bones and soft parts have been found less severe than with the old leaden bullet, but after 1,200 metres fractures become more comminuted. In the epiphyses and short bones less extensively-fissured tracks are caused, and the bone fragments are less displaced. Splinters are smaller, and the joint lesions less comminuted. Simple perforations also are more numerous. On the flat bones of the cranium simple grooving of the bone is more frequent, and the edges of the fracture are cleaner. The cutaneous and muscular wounds and arterial perforations are narrower and cleaner cut. With regard to the relation between the bone and periosteum, it has been found that at a long range injuries of the compact bone were more extensive, compared with those at a shorter distance, but the splinters remain attached to the periosteum; whereas at the shorter range the periosteal attachment was destroyed, and the fragment displaced. Lastly, foreign bodies — as pieces of clothing — have been found more frequently to lodge than with the older bullets.

As to actual experience on human bodies we can learn from the results found in the Chilian War, the Bombay riots of 1893, and from certain accidental cases. In the Chilian War wounds healing without suppuration (in the majority of cases within eight to ten days) were produced. Where, however, the diaphysis of a bone was struck much splintering was caused, and the healing process was slower. In only one case did the missile cause a simple hole through the diaphysis. In one case explosive effects were observed. As a rule, there was no lodgment of the ball. The number of dead on the field was four times greater than that of the wounded, pointing to the large number of clean-cut wounds of vessels. Up to a distance of 400 yards the bones were shattered without exception (Fig. 45). At greater distances there were clean perforations and oblique fractures, whilst the fractures of the long bones were of larger size than usual. Again, in a case in the Bombay riots, the bones of the leg were found shattered from knee to ankle, necessitating amputation.

Complications of gunshot wounds.—These, such as gangrene, pyæmia, septicæmia, erysipelas, and tetanus, are elsewhere described.

Course of gunshot wounds.—Following impact by the old round bullet, and to a less extent by the Martini and Snider, the usual course of contused and lacerated wounds shows itself, but not invariably. Thus I have seen a subcutaneous wound of about eight inches long caused by a Snider bullet heal without a drop of pus. Wounds inflicted by the magazine bullet are more of the nature of subcutaneous lesions, and therefore more favourable to healing.

Treatment.—*Pain* and *shock* will first demand treatment (in the absence of more pressing symptoms such as hæmorrhage). For the *pain* a subcutaneous injection of morphia should be given. The *shock* should be combated by the recumbent position and hot bottles to the lower extremities, etc. If the patient can swallow, he should be given beef-tea, wine, or coffee; or, in severe cases, a hypodermic injection of ten minims of ether over the heart. In the local variety of shock rubbing of the limbs is of service. No operation should be undertaken, unless quite unavoidable, till the symptoms of shock have passed away. If, however, operation be inevitable, most surgeons advise that narcosis be avoided.

The treatment of the wound next claims our attention. As to the immediate duty of surgeons on the battle-field, everything that can ensure an aseptic course to the wound must be adopted. Nussbaum has laid down that the fate of the wounded man lies in the hands of the surgeon who first attends him. Wounds accordingly should never be examined at the front either with fingers or instruments, except in the case of wounds of the blood-vessels, or "in cases where the projectile has passed through one of the large cavities of the body, and, without wounding its contents, has remained in the wall of the cavity, then the removal of the ball is at once advisable, for fear that, if left, it might fall back into the cavity implicated. For surgeons at the front there is only one line of treatment—to occlude the wound provisionally, to lay the wounded part in a suitable position on the litter, and to render it practically immovable" (Reyher). Various forms of first field-dressing are therefore supplied to the men going into action. That used in the army in India consists of two pads of carbolised tow, enclosed in carbolised gauze with a few accessories. Sir Joseph Lister recommends dusting the wound with iodoform. The first field-dressing being applied, and the limb immobilised, the wounded man is then brought to the field hospital, where a thorough examination can be made. For an antiseptic solution wherewith to wash the wound Lister recommends one of corrosive sublimate. This substance being soluble in one-and-a-half times its weight of glycerine, one-fluid drachm of this solution will prepare a watery solution of corrosive sublimate (1 to 1,000) if added to four pints of water. Or a like solution may be made by adding ten grains of corrosive sublimate and fifty grains of tartaric acid to one pint of water.

The first point to be determined now is the lodgment or otherwise of the bullet. Where there are wounds of aperture or exit, this is, of course, readily settled. Should only one wound be present, the bullet need not necessarily have lodged, for it may have rebounded after striking, as in skull wounds, or have been drawn out of the wound by the portion of clothing it had propelled before it. To determine the route the patient should, if possible, be placed in the position he was in when wounded. There is, however, a certain amount of disadvantage attending this, for the bullet may become displaced by the necessary movement. In judging of the route it must be remembered that fasciæ, tendons, and bones can divert the missile. With regard to multiple wounds, instances are on record where one bullet has passed through both thighs, or where one projectile has divided on striking a bone.

In certain cases immediate digital examination is superfluous, as in the simple "seton" wound of the soft parts, the lesion of an epiphysis without fracture, or where the small bones of the hands or feet are injured. Here the wounds should be washed with an antiseptic solution. If no clothing can be seen to have been carried in, the parts should then be dusted with iodoform, and subsequently enveloped with any of the various wool-dressings in vogue—*e.g.* with salicylated cotton-wool or Tillmann's dressing—and finally, in the case of an extremity, be immobilised. But where we find comminuted fracture, free splinters, great damage to a joint, hæmorrhage from some large vessel, or wound of an abdominal viscus, digital exploration is demanded. The aperture of entrance is enlarged, if necessary, and the finger, previously disinfected, is slowly pushed into the deepest recesses of the wound. Its route is thus determined, the amount of injury estimated, and the presence of any pieces of cloth ascertained. Should the wound be too long for the finger, a vertebrated probe may be used, care being taken not to injure the vessels. To obviate the difficulty sometimes experienced in ascertaining the presence of the bullet, Nélaton's porcelain probe or Lecomte's stylet-pince can be employed. Longmore has suggested a simple method by means of the magnet of an ordinary pocket-compass, which, having some turns of copper wire covered with thread wound round it as an induction coil, forms the electric indicator. A copper coin, covered with flannel steeped in acid, is placed in a piece of zinc sheeting. One insulated wire with its exploring needle is attached to the copper coin, the other, with the compass held in its course, to the zinc sheeting. When the circuit is completed by contact with the bullet, the deflection of the magnet indicates its presence. Lastly, Deneux recommends chemical exploration. He fixes on the end of a flexible probe some charpie impregnated with dilute acid, which is then pressed against the suspected body. Iodide of potassium then applied to the charpie gives a yellow colour should lead be present, and ammonia a blue colour in the case of lead or bronze. Of all the above proceedings exploration by the finger gives the best results. Pieces of clothing can be distinguished, as well as the fixation or mobility of the bullet. In cases where we can obtain no positive evidence we have then only the local signs to trust to. The bullet being discovered, it must then be removed by forceps, such as Coxeter's, Luers', or Steavenson's.

The treatment of hæmorrhage.—The surgeon hitherto has had to treat more often secondary hæmorrhage, but in future wars primary hæmorrhage will be more frequently met with, as the modern bullet causes less contusion of the vessels, and a cleaner cut, whilst under antiseptic treatment secondary hæmorrhage will be less frequent, inasmuch as its chief cause was due to septic changes in the wound. The hæmorrhage may be arterial, venous, or parenchymatous.

(1) *Arterial.*—Primary or secondary. For provisional treatment on the actual field, a tourniquet may be improvised by a bayonet and bandage. The ordinary field tourniquet is apt to slip during the transit of the patient to the field hospital. Failing a tourniquet the limb may be bent on itself, or Esmarch's bandage may be used. A partial wound of the vessel is more dangerous than one in which

it is completely cut across. Arrived at the dressing station, the artery should be ligatured above and below the site of wound; the peripheral end must be found, notwithstanding the difficulty often experienced in so doing. Esmarch strongly recommends that the vessel should be tied above and below at the seat of the lesion, under rigid antisepsis. To effect this, the large size of the necessary incision need not now be a counter-indication. The surgeon should turn out the clots, and then tie the vessel. He should also isolate and separately ligature any branches given off from the impaired part of the trunk.

Secondary hæmorrhage is due either to septic processes breaking down the thrombus in the vessel, or to its puncture by splinters. In the latter case the surgeon should remove the splinters, and tie the vessel above and below the lesion. But when due to septic processes, it is necessary either to ligature at a distance or to amputate. Mosetig-Moorhof, however, relates a case of secondary hæmorrhage in the clavicular region, which was treated successfully by an iodoform gauze tampon, and other cases similarly treated, affecting the vertebral and posterior tibial arteries.

(2) *Venous.*—If a small vein be wounded, use an antiseptic compress. If a large, tie above and below. Küster recommends compression of the vein by small clamps for twelve to twenty-four hours. Where secondary hæmorrhage occurs from a large vein, the prognosis is bad.

(3) *Parenchymatous.*—When primary, raise the limb; apply ice; employ local pressure with an antiseptic tampon, or use styptics, the best of which is iodoform. Neudoïfer recommends a tampon impregnated with a 5 per cent. solution of peroxide of hydrogen. When secondary, the thermo-cautery or zinc chloride may be used. In cases of general sepsis, amputation has often been rendered necessary.

Lastly, how should those cases be treated where the hæmorrhage is suspended when the case comes before the surgeon? Fischer, Legouest, and Delorme advise that the vessel be sought for and tied; whereas Guthrie and Dupuytren and others hold that the parts be immobilised, a compress applied, and that the surgeon wait for events. The best practice would appear to be to cut down and tie above and below if the wounded artery be important and easily accessible, otherwise to follow out the second alternative.

The sequelæ following ligature of the principal vessels are (especially where the tissues are much infiltrated with blood) gangrene of the extremities, and extensive œdema of the distal parts.

The treatment of fracture.—The course of gunshot injuries of bone is chiefly influenced by the accompanying condition of the soft parts, the state of the splinters, and especially by the treatment first employed. Where the continuity of the bone is entire, as in "gutter" fractures, contusions, simple penetration, and fissured fractures, the surgeon should freely bathe the parts with an antiseptic lotion, and then immobilise them under an antiseptic dressing in plaster of Paris. As regards splinters — where they are not numerous, and are not associated with much deformity, and are retaining their connection with the periosteum—the surgeon should not attempt to extract them, but after replacing them carefully, should antiseptically occlude the parts and immobilise the limb; the splinters will then consolidate with the rest of the bone. Should they lie free and detached from the periosteum, they may be removed. Where there is much loss of substance in the bone, and great deformity and mobility of the parts, the wounds should be enlarged with the usual precautions, the completely free splinters removed, and then if conservatism seem possible, the wound should be disinfected and drained, and the part immobilised. But where the bone is completely shattered, amputation is the only resource.

As regards the treatment of gunshot fractures of the femur, James, of the United States Army, points out that where the knee-joint is not involved, the rule formerly laid down to amputate in fractures of the middle and lower thirds of the bone should be disregarded, provided the patient can be treated near the battlefield. He holds that if the knee joint be implicated, and generally if the femur be comminuted more than six inches, it is best to amputate primarily if possible; but an intermediate amputation is better than the risk of waiting for the secondary period. If conservatism be decided upon, let the surgeon remove all foreign bodies and loose or slightly-attached fragments of bone only, and all necrosed portions as soon as they are loosened from the periosteum. A simple retention apparatus with moderate extension by weight and pulley should then be provided, and the wound kept carefully antiseptic.

The treatment of wounds of joints on the field.—The joint should be syringed out with an antiseptic lotion, absorbent wool should then be applied, and the limb immobilised. At the hospital, if conservatism be decided on, counter-openings should be made, and the joint drained, and after trap-doors have been left, immobilised. Formerly a wound of a large joint necessitated amputation. This is not so now, although all joint-wounds are severe, and liable, especially where antisepsis cannot be carried out, to induce pyæmia. The surgeon should incise the joint and remove all foreign bodies, and endeavour to arrest any inflammation. If this be not possible, then excision or amputation is the only course. As regards the individual joints, in the case of the shoulder (unless the injuries be very slight), excision has so far given the best results. For the elbow, conservatism has given better results than excision or amputation. The same may be said of the wrist. In the hip both amputation and excision have been attended with very fatal results. In the knee and ankle conservatism should always be employed, if possible.

Rules for amputation.—These cannot be emphatically laid down at the present time. Many gunshot wounds of the large joints are now treated successfully with conservatism which would formerly have been submitted, without a doubt, to amputation.

But where the injury necessitates removal of the limb, primary amputation is certainly to be preferred to an intermediate or secondary operation. The following lesions entail primary amputation :—

1. Nearly complete avulsion of a limb.
2. All conditions of injury by which the further vitality of the limb is annulled.
3. Injury to the chief nerves and vessels, with comminution of the bone.
4. Great and irreparable destruction of the soft parts.

Secondary amputation will be indicated :—

1. Where suppuration, with extensive inflammation of the bone and soft parts, is wearing out the patient.
2. Where gangrene has supervened.
3. Where late hæmorrhage has come on, which cannot otherwise be stopped.
4. Where septicæmic affections or acute osteomyelitis are supervening.
5. It is recommended also by some on the onset of tetanus.

The discussion of wounds of the skull, spine, thorax, and abdomen will be found in the respective Articles on the surgery of these parts.

Injuries by massive projectiles.—These produce immense laceration of the parts struck, the wound being very irregular and much lacerated. Large portions, or even an entire member, of the body, may be torn away. The treatment to be carried out cannot be laid down in definite rules. Such treatment as is possible will depend on the individual case.

II. BAYONET AND SWORD WOUNDS.

Sword wounds are incised, and may be of any extent. As a rule, if, however extensive, they do not wound a fatal part, they do well. Where penetrating, as in the abdomen and chest, the usual rules of treatment are to be followed.

Bayonet wounds differ in their nature according to the form of weapon. The bayonet of the Martini-Henry, 17 inches long, inflicts a much more severe lesion than that of the Lee-Metford rifle. The Martini-Henry bayonet is of a triangular shape, and thus a punctured wound with three lines radiating from a centre is caused. The Lee-Metford has a "sword-bayonet" 12 inches long, hence the sides of the incised wound are parallel, and the lesion is more disposed to heal favourably. Bayonet wounds are very liable to be followed by septic inflammation, and the formation of deep-seated matter. The deep-seated vessels and nerves are also prone to be injured. The wound must be well washed out with an antiseptic lotion, be drained and then dressed with absorbent cotton-wool, and immobilised. Should inflammation arise, counter-openings for the discharge of pus must be made, and the freest drainage ensured.

III. ARROW WOUNDS.

In several of the British expeditions on the Eastern frontier of India soldiers have incurred arrow wounds, such as was the case in the expedition against the Akas and Abors. Hamilton points out the liability of these injuries to suppuration, profuse hæmorrhage and tetanus. In many cases the arrows have been furnished with poisoned tips. The chief object in the treatment of arrow wounds is the removal of the missile without breaking off its head. Where it has penetrated beneath the skin, all that should be done on the field is to cut off the projecting part of the shaft and apply the first field-dressing. At the hospital the subsequent extraction of the shaft and head must be carried out, with every care taken not to separate the head from the shaft in so doing.

If the point of the arrow can be felt on the opposite side of the limb into which it has penetrated, the projection should be cut down upon, and the missile extracted on this aspect. Should the head and shaft be found separated, the latter can be withdrawn on one side and the former on the other.

Should the arrow have penetrated a bone, Hamilton recommends it "to be rocked to and fro in a direction at right angles with the plane of the groove which is made on the outer or feathered end of the shaft." But this rocking is very likely to cause a separation of the head from the shaft. It would appear to be far preferable to guide a probe-pointed bistoury along the shaft to the point at which the arrow has lodged in the bone, and then to freely incise the track, and attempt to withdraw the head with forceps. If this be impossible, a wire should next be passed around the head of the arrow, and twisted round both the shaft and the forceps, and all three removed together. The arrow being removed, the track must be well syringed out and drained, and if a limb have been the point struck, it should be immobilised.

XII. BURNS AND SCALDS.

By C. B. LOCKWOOD, F.R.C.S.,

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Definition.—*Burns* are the effects of dry heat; *scalds* are caused by hot liquids, or by steam. The pathological and clinical results are, in any case, so much alike that it is unnecessary to consider them apart.

Mortality.—Excessive heat produces the most extensive and serious injuries, and causes a large proportion of the deaths in our great hospitals.

In ten years, from 1884 to 1893 inclusive, six hundred and ninety-six cases of burns and scalds were admitted into St. Bartholomew's Hospital. Of these one hundred and thirty-eight died. Burns are more fatal than scalds. Of four hundred and thirty-four who were burnt, one hundred and fourteen died (26·26 per cent.); and out of two hundred and sixty-two who were scalded, twenty-four died (9·16 per cent.).

During infancy both burns and scalds are exceedingly fatal. Of the hundred and fourteen who succumbed to burns, no less than sixty were under the age of five years. Of the twenty-four who died of scalds, no less than nineteen were under five years of age.

Within the period which has been taken, no one over sixty years of age died of scalds, although thirteen died of burns. Infants burn and scald themselves by playing with fire, or by upsetting or drinking from boiling kettles; adults are, perhaps, more often injured by the upsetting of lamps, or the explosion of kitchen boilers.

Classification.—Dupuytren's classification of burns and scalds into degrees, according to their depth, is convenient, and is usually adopted.

In the *first degree* the surface is scorched, with subsequent superficial redness and hyperæmia. This erythema is followed by desquamation. No trace of the injury remains beyond a slight discoloration.

In the *second degree* the epidermis is raised from the derma into vesicles or bullæ. The true skin inflames, but is otherwise uninjured, and no mark or scar results, except, perhaps, slight pigmentation (ephelis ignealis), unless from neglect or inappropriate treatment the denuded skin becomes more inflamed and ulcerates.

In the *third degree* part of the thickness of the true skin is destroyed by the heat. When this separates, a surface of true skin is left to heal by granulation. This, of course, leaves a permanent white scar, but without contraction or deformity, because the whole thickness of the skin has not been destroyed.

In the *fourth degree* the whole thickness of the skin is burnt, and the subcutaneous fat and connective tissue may share in the destruction. In the process of repair there is much scarring, with contraction and deformity. This is partially due to the actual loss of tissue, and partially, as Mr. T. Holmes remarks, to the formation of inelastic contractile scar in place of the elastic cellular tissue.

In the *fifth degree* the burn penetrates through the skin, subcutaneous tissue and deep fascia, and reaches the muscles.

In the *sixth degree* everything is charred to the bone.

Effects on the tissues.—Extreme heat affects the tissues in two ways. By its direct action it coagulates their albumen, dissipates their moisture, or consumes them entirely. By its indirect action it excites in them acute inflammation, which leads to desquamation, vesication, ulceration, sloughing, or gangrene. As a rule, both the direct and indirect actions of heat are seen in severe burns and scalds.

When the direct action of heat is sufficient to kill the tissues outright, the epidermis becomes white and opaque, and large areas of it separate from the true skin. The true skin becomes seared, yellow, parchment-like, and shrivelled; the deep tissues become brown, black, and charred, and their blood coagulated.

The acute inflammation which is caused by excessive heat leads, in the first degree, to desquamation of the superficial layers of the epidermis. When it is deeper, as in the second degree, the inflammatory effusion collects as a yellow fluid betwixt the epidermis and the dermis. When the inflammation is very acute, and when it attacks the dermis and deeper tissues, ulceration, sloughing, and gangrene are the results.

The causation of these changes by acute inflammation has been explained elsewhere (page 59). They occur when the inflammatory processes have passed through the phases of acceleration and retardation of the circulation and have ended in stasis of the blood-stream.

Thus, when the capillary circulation is stopped, ulceration ensues, owing to the death of the minute portions or molecules of tissue supplied by them. When the stasis is in the larger trunks and arterioles, sloughing occurs, because larger portions are deprived of nutriment and oxygen; and stasis in the larger arteries or veins causes large pieces of the body, or whole limbs, to become gangrenous.

Thus the local changes which follow burns and scalds depend partially upon their degree. The desquamation and occasional discoloration which follow the first degree hardly call for comment.

The vesicles and bullæ of the second degree usually break and dry up after their contents have become turbid. In the third, fourth, fifth, and sixth degrees suppuration and granulation follow, should the victim survive. In the third degree the suppurating surface is speedily covered with new epithelium by proliferation of that which remains undestroyed between the papillæ. In the fourth degree, the parts killed by the heat, or by the subsequent acute inflammation, are separated by suppuration and granulation.

The granulation tissue fills and repairs the gap left by the separation of dead tissues. Inasmuch as the whole depth of the skin is destroyed in the fourth, fifth, and sixth degrees, the epithelium which grows over the new material has to extend from that which surrounds the edge of the wound, usually a slow and uncertain process.

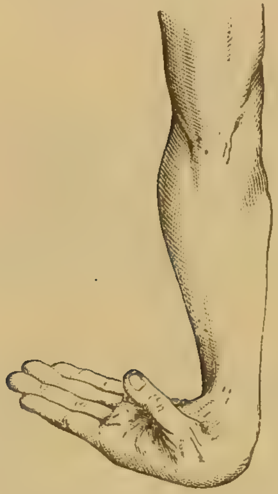
Fig. 46. — Contraction of Wrist after Burn of the Fourth Degree.

The destruction of tissue caused by burns and scalds is likewise complicated by contraction and cicatrisation of the reparative material, causing the most distressing deformity. Joints become immovably flexed, the chin drawn against the sternum, the eyelids dragged from the eye (ectropium), and the nose, mouth, or ear distorted. (Figs 41 and 46.)

Cheloid is prone to develop in the scars of burns (page 231).

Effects on the body generally.—The general effects of burns and scalds are grave and often fatal. This we should expect when we recall that the skin is not only an organ abundantly supplied with nerves and blood-vessels, but is also a most important excretory and respiratory apparatus. When excessive heat is applied to any extent of the body, it is followed by (1) shock and collapse, (2) reaction and inflammation, and (3) by various local and general diseases due to bacterial invasions.

(1) **Shock and collapse** cause about half the deaths which result from burns and scalds (A. E. Durham). The amount of shock depends upon the age and sex of the patient, the situation of the burn or scald, its extent and degree. In infants and little children the effects of shock and collapse are usually excessive. The face and surface of the body, especially the extremities, are pale and cold, and covered with a clammy sweat; the lips are white and tremulous; the pupils are dilated; the pulse is rapid and almost imperceptible; the respirations are quick and shallow, and the contents of the stomach, bladder, and rectum are evacuated spontaneously. Children often have convulsions; adults, shiverings and rigors. The temperature of the body is lowered; there is extreme mental depression and apprehension; delirium is not rare; the tongue and mouth are dry, with distressing thirst; and restlessness, coma, and stupor are usual towards the end.

Pain is more excessive in the slighter than in the graver degrees; its absence is a sign of serious import. Travers, in his classical work, gives many examples illustrating these fatal complications of burns and scalds. Their effects are more to be apprehended in infants, delicate females, and in burns and scalds of the chest and abdomen. The extent of the injury, or, in other words, the number of peripheral nerve-endings involved, is usually more important than the depth of the burn or scald. I have seen a fireman die collapsed a few hours after a burn of the first and second degree, involving the face and trunk. Mr. Durham says that recovery rarely occurs when more than half the surface of the body is burnt or scalded, and Von Nussbaum says it is rare after a third. Travers thinks the constitutional effects are less when the cuticle is charred, or killed, or raised in blisters, than when it is detached and ravelled in rolls like wet paper.

After death from shock or collapse no distinctive appearances are seen. The membranes of the brain, and the abdominal and thoracic viscera are engorged with blood, and, therefore, this has sometimes been called the stage of "congestion," the other stages being those of reaction and inflammation, and of suppuration and exhaustion.

In some instances death results soon after the burn or scald from cardiac thrombosis. This is said to be associated with disintegration of the blood-corpuscles. It is betokened clinically by dyspnœa, precordial pain, and great irregularity and tumult of the heart's action.

(2) Shock and collapse are followed by *reaction*, and reaction by **inflammation** and the various complications due to sepsis. The inflammation that follows the application of heat cannot be prevented, but its amount may be controlled by skilful treatment.

(3) **Suppuration and sepsis** are due to the multiplication and action of the bacteria which always inhabit the skin, especially that of uncleanly people. They are also caused by the introduction of bacteria from without. The prevention of suppuration after burns is in a very backward state. Suppuration is apt to be looked upon as inevitable, and its treatment often left to inexperienced persons. The number of deaths from sepsis after burns and scalds is a reproach to surgery. In addition to suppuration, the local infection may cause ulceration, sloughing, gangrene, lymphangitis, abscess, erysipelas, and cellulitis. When the bacteria enter the circulation they cause septicæmia and pyæmia. After deaths from burns and scalds pneumonia, pleuritis, pericarditis, and nephritis are commonly met with. (*See* page 257.) From histological examinations recently made I have come to the conclusion that these are due to bacterial invasion. In infants streptococci are particularly virulent, and are the unsuspected cause of many deaths.

Complications.—*Tetanus* is an occasional complication of burns. It may occur at any time before the wound is healed. The experiments of Vassale and Sacchi show that the juices of burnt tissues are highly toxic. It is probable that they contain a poisonous substance analogous to muscarin. This suggests the advisability of removing burnt parts as soon as possible.

In addition to the complications which have been mentioned, it occasionally happens that as the sloughs separate, *hæmorrhage* occurs from the larger arteries or veins.

Occasionally burns are deep enough to *penetrate the capsules of joints.* This is followed by suppurative arthritis, which may also be excited by the extension of septic processes, either directly from the wound, or through the circulation, as in pyæmia. The great serous cavities of the chest and abdomen may suffer in the same manner.

Should shock and acute sepsis be survived, a period of *suppuration* and *exhaustion* usually precedes recovery. This is often called the third period, and may be said to begin after the sloughs have separated, and about a fortnight after the injury. During this period the symptoms are those of hectic fever, and should the suppuration be very prolonged, it ends in amyloid disease of the various organs, especially of the kidneys, liver, and spleen. Also during this stage the wound contracts and cicatrises, and may produce extreme deformity.

Sometimes cheloid grows as cicatrisation is completed. Burns which are too extensive to heal leave chronic ulcers. I have occasionally seen these become epitheliomatous.

Visceral changes.—In a small proportion of cases of burns or scalds small hæmorrhages, acute inflammations, or ulcerations are found in the *intestinal tract.* In ten years, from 1884 to 1893 inclusive, 138 patients died of burns and scalds in St. Bartholomew's Hospital. During that period I can find but three post-mortem records of pathological changes in the alimentary tract, although Erichsen says that ulcers of the duodenum were found six times out of 22 cases which he examined. In one case—that of a man aged twenty-six years, who was burnt upon the face and arms—there were small submucous hæmorrhages the size of a pin's head at the pyloric end of the stomach, in the duodenum, and in the cæcum. In the case of a child small punctiform hæmorrhages were found in the duodenum. The specimen from the third case is in St. Bartholomew's Hospital Museum.* It demonstrates acute inflammation and ulceration of the mucous membrane of the duodenum after an extensive scald of the chest. The patient was a child a year old, who lived twenty days after the injury. The ulcers are usually found in the duodenum, and not far from the opening of the bile duct. They may perforate the bowel, and after that event become occluded with inflammatory lymph.† Cases have been recorded in which they have ulcerated into the duodenal vessels and caused fatal hæmorrhage. They may be multiple. Nothing is accurately known about the pathology of these ecchymoses, inflammations, or ulcerations of the alimentary tract. The interest which surrounds them has led authors to give them undue prominence. Their existence is, as a rule, unsuspected during life. It is quite fanciful to imagine that they are due to any increased action of Brunner's glands, rendered necessary by the destruction of sweat glands. Pain in the region of the duodenum, increased by pressure, with vomiting of blood, or diarrhœa with blood in the motions, would render their existence probable. They have caused death by hæmorrhage or by perforative peritonitis. They are said to occur about the tenth day.

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* Series XVIII. No. 1969a.

† *See* Series XVIII. No. 1969. St. Bartholomew's Hospital Museum.

During the course of burns and scalds *albuminuria*, or even *hœmaturia*, may occur. They are both due to acute nephritis. After prolonged suppuration, albuminuria may be caused by amyloid infiltration of the kidney.

Pneumonia and *pleuritis* are also frequent complications of burns and scalds. Their presence is often unsuspected, as the patient is too ill for a physical examination. Pain, cough, rusty sputum, and increased frequency of respiration would cause them to be suspected.

Engorgement of the vessels of the meninges of the brain and spinal cord, and sometimes *meningitis*, have been noted after deaths from burns and scalds.

Treatment.—The *immediate* treatment of burns and scalds is directed to the prevention of death from shock and collapse, and to the alleviation of pain. Brandy may be given by the mouth, but is often more efficacious if given as an enema. Two or three ounces of brandy should be administered, with the addition of a suitable dose of opium. If necessary, the rectum should be washed out before introducing the brandy enema. If the heart's failure be extreme, ether and liquor strychninæ may be injected deep into the subcutaneous tissue, or into the substance of the gluteus maximus. The excruciating pain should be relieved with a hypodermic injection of morphia

The dressing of a burn or scald may be a painful process, and therefore it is well to be prepared to give an anæsthetic. The garments should be gently and carefully removed, so as not to denude the injured surface.

The *local treatment* of burns or scalds depends upon their degree. In the first degree there is no breach of continuity, and sepsis is not to be apprehended. Dusting the surface with any soft, simple powder relieves the pain. If the area be small and shock absent, cool applications, such as cold water, lead lotion, cold starch, and so forth, may be prescribed.

In burns of the second degree the separated epidermis and the fluid in the bullæ protect the denuded papillæ until their highly sensitive surface is again covered with epithelium. It is therefore best to leave the bullæ alone until this has taken place. Should it be expedient to let the serum out, care should be taken not to injure or remove the epidermis.

In burns of the third degree the dangers of subsequent sepsis and suppuration have to be provided for. The whole area should be thoroughly washed and soaked with a strong disinfectant, such as carbolic lotion (1 in 20) or sublimate solution (1 in 1000), and thoroughly covered with an antiseptic dressing. Carbolic gauze (5 per cent.) may be used, or the whole area wrapped in sheets of salicylic wool (10 per cent.). Iodoform gauze, or iodoform wool, is also a suitable application. There is, however, a danger of iodoform poisoning. For similar reasons it is inadvisable to apply carbolic gauze or sublimate gauze or wool to large areas, although they are exceedingly good in ordinary cases. It is difficult to find an antiseptic application which fulfils the requirements—namely, to relieve pain, prevent sepsis, and exclude air. Most of those at our command achieve only one of these desiderata, others are irritant or poisonous. Vegetable applications, such as flour and starch, are objectionable, because they are apt to decompose; oxide of zinc, bismuth, or prepared chalk are to be preferred. A number of oleaginous preparations have been recommended. One of the most popular and anodyne applications is a mixture of linseed oil and lime water in equal parts. This is called Carron oil, because of its use at the Carron iron-works. Wertheimer recommends that thymol be added to increase its antiseptic action. Carbolic oil is an inefficient antiseptic and often causes carboluria. It should be used with great caution. Antiseptics mixed with oil or fat become inert. Iodoform with vaseline forms a good ointment (ʒj to ℥j).

The effects of sepsis are to be watched for as soon as reaction is established. They are usually severe about the time when the sloughs begin to separate. Immersion in warm baths is a particularly valuable expedient for preventing sepsis. Von Nussbaum says that it has done much to lessen the mortality of burns in Germany. Even children will bear immersion in hot water (100° F.) for many consecutive days and nights. The hot water excludes air, prevents sepsis, and promotes the separation of sloughs. When limbs are injured its application is easy.

After the separation of the sloughs, the cicatrisation and distortion have to be guarded against. This may be done by means of splints and weights. One of the most efficacious means of preventing it, and of at the same time promoting the healing of the injury, is to cover as much as possible of the surface by Thiersch's skin grafting, by transplantation of flaps or strips of skin, or by planned incisions. The other complications are to be dealt with as they arise.

Severe burns of the extremities may necessitate amputation.

Scalds of the pharynx.—Infants and children not infrequently scald the fauces and pharynx by drinking from the spouts of boiling kettles. This is commonly followed by spasm of the glottis and by œdema of the upper aperture of the larynx, of the epiglottis and glottis. The swollen aryteno-epiglottidean folds and the swollen epiglottis can be felt with the finger after a gag has been introduced. The slighter degrees of œdema may be relieved by scarification leeches to the throat, steam inhalations, small doses of tincture of aconite, or of wine of antimony. But if the entrance of air be dangerously impeded by the œdema, or by the attack of spasm, laryngo-tracheotomy, or the high operation of tracheotomy, should be done. This is a dangerous expedient, and the fatal result may be erroneously attributed to the operation by the relatives. As death from spasm of the glottis is imminent in burns or scalds of the larynx or pharynx, and as the air-passages may require opening, the surgeon should on no account leave the case unprovided with skilled assistance. The acute inflammation that follows the injuries often ends in tracheitis and broncho-pneumonia.

XIII. THE INFLUENCE OF CONSTITUTIONAL CONDITIONS UPON INJURIES.

By FREDERICK TREVES, F.R.C.S.,

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It is needless to state that the age, the state of health and the general condition of a patient, affect in varying degree the prognosis after injury and the result after surgical operation.

In the case of urgent operations, performed for the immediate purpose of saving life—as in the relief of strangulated hernia—few considerations weigh with the surgeon save the one great need. But in slighter operations it is of infinite importance that every possible consideration be given to all circumstances that may affect the patient's well-being.

Every surgeon must have met with instances where he has regretted the neglect of this fundamental precaution. I once snipped off with the scissors a small fibrous epulis growing from the gum of a little boy. It was discovered afterwards—what should have been known before—that the patient was the subject of hæmophilia. The small wound became the seat of almost uncontrollable hæmorrhage, and it was not until a fortnight had elapsed that the patient could be said to be out of danger. In another case—at the patient's earnest request—a small sebaceous cyst was removed from the scalp of a man of fifty. The wound soon broke down, suppurated freely, and became the starting-point of a low form of erysipelas of which the patient nearly died. It was discovered after the operation that the man was suffering from diabetes, a fact of which he himself was not aware.

In forming a proper estimate of the risks involved by operations—so far as the condition of the patient is concerned—and the prospect of sound recovery after injury, many factors have to be considered, and in the paragraphs which follow the more important are dealt with.

Age.—Age exercises a considerable effect upon the result of injuries. Taking amputation as a type of injury, it will be found that quite young children—those under the age of five—do not bear operation well, the mortality being as high as between the ages of thirty-five and fifty. The mortality is lowest between the ages of five and fifteen, and these years certainly give the best results from operations and injuries of almost every kind. After fifteen the death-rate begins steadily but slowly to increase. The variation between the whole period from twenty to forty is not considerable; but the risk of death after operation is twice as great in patients between those ages as it is in individuals under twenty. In patients over forty the mortality is nearly three times in excess of the rate observed in patients under twenty. The increase in the risk of death between fifty and seventy is very rapid.

In *children* wounds usually heal well; the patient's organs are probably healthy and vigorous, and the nutritive activity of the body is in its prime. Children show great recuperative power, and are free from the effects of that mental anxiety which often acts so injuriously upon adults. They are able, moreover, to endure a long confinement in bed and a tedious suppuration with comparatively little ill effect.

On the other hand, children suffer severely from shock and the effects of acute pain. Pain, if unrelieved, may in a few hours reduce a child to a state of collapse. Shock is certainly the chief danger in operations upon young and healthy children.

It has been said that children bear the loss of blood badly. Hæmorrhage must, however, be regarded relatively when comparing children with adults. If the weight of the body be taken in conjunction with the amount of blood lost, it will be found that children bear hæmorrhage well, and, in the case of repeated bleedings, they bear it remarkably well.

Operations should not be performed, if possible, during the first dentition. Children are then often restless and excitable, liable to digestive disturbances and to convulsions, and apt to develop a high temperature under little provocation.

The natural restlessness of children is often an obstacle to the perfect success of an operation, and injuries in the region of the pelvis are apt to be complicated by the difficulty of keeping the child clean.

Of the influence of *old age* upon injuries and operations, Sir James Paget writes:—"Among the old there are even greater differences than among the young in the ability to recover from operations; and age, if reckoned by years, is not the only thing we must estimate. . . . They that are fat and bloated, pale, with soft textures, flabby, torpid, wheezy, incapable of exercise, looking older than their years, are very bad. They that are fat, florid, and plethoric, firm-skinned and with good muscular power, clear-headed and willing to work like younger men, are not, indeed, good subjects for operation, yet they are scarcely bad. The old people that are thin and dry and tough, clear-voiced and bright-eyed, with good stomachs and strong wills, muscular and active, are not bad; they bear all but the largest operations very well. But very bad are they who, looking somewhat like these, are feeble and soft-skinned, with little pulses, bad appetites, and weak digestive power, so that they cannot in an emergency be well nourished.

"The old are, much more than others, liable to die of shock, or of mere exhaustion, within a few days after the operation. They bear badly large losses of blood, long exposure to cold, sudden lowering of temperature, loss of food. Large wounds heal in them lazily. Their stomachs, too, are apt to knock-up with what may seem to be no more than necessary food. Many old people are in less peril with a scanty diet than with a full one. Their convalescence is often prolonged. . . . . There are some to whom convalescence is more dangerous than disease."

In the aged especial risks attend injuries of the lower limbs, or of the lower part of the trunk, or the back. The necessary long confinement in the recumbent position is very apt to be attended by congestion of the bases of the lungs and a low form of pneumonia. This same position favours the production of bed-sores.

Sex.—Other things being equal, it would appear from statistics that women bear operations and injuries somewhat better than men. This fact may be explained by the circumstance that they are more tolerant of confinement to house and bed, lead less active lives, and adapt themselves more easily to the surroundings of a hospital ward. They are probably more temperate and regular in their lives, and more patient.

It is well not to operate, unless compelled, during menstruation. In perhaps the larger number of instances of operations performed during this period, no ill effects are noticeable; in the minority an unaccountable rise of temperature with often considerable nervous and digestive disturbances are met with.

Still more desirable is it that no operation be performed during pregnancy. The special risk incurred in such a case is that attending abortion. Apart from this risk there is little to anticipate, and wounds do well. Ovariotomy and other grave abdominal operations have been performed during the various stages of pregnancy without inducing abortion and without evil results. It appears to be impossible to estimate the risk of miscarriage after a surgical procedure or injury.

During lactation, also, operations should be avoided when possible; the patient is usually in comparatively feeble health, and certainly not in the best condition for a serious call upon the nutritive powers.

The robust and the feeble.—Experience shows that the best subject for an operation, or the best result after injury, is not always to be found in the strong lusty man in the prime of life.

Such a man has his mode of life suddenly interrupted when he comes under the surgeon's care. His blood-vessels are full; his viscera have adapted themselves to the exigencies of an active life; his tissue changes are rapid and extensive, oxygenation is quickly disposing of the great refuse matter which is continually accumulating at the very moment when the tide is abruptly checked. The man finds himself motionless in bed, every circumstance of his life is changed, he has no time to adapt himself to his altered position, and it is a matter of little wonder that the inflammatory process which may be induced runs riot and is not readily controlled. Circumstances are not improved by his altered mental condition, by the shock of the accident, the horror of mutilation, and the possible miseries of the future.

The feeble subject who has become wasted and worn by some such trouble as chronic joint disease is, on the other hand, acclimatised to bed-life; his diet, his muscular changes, his breathing powers, have all adjusted themselves to the molluscous condition. His viscera are healthy, there is no accumulation of débris to be rid of, and possibly even confinement is ceasing to be irksome. To such an individual amputation comes as a relief. The change that amputation brings in his life is even agreeable, and opens up the prospects of a new existence.

The great difference in the mortality of amputation for injury and for disease serves to emphasise this point. It must be distinctly understood, however, that these differences are only partly due to the patient's condition. They perhaps as largely depend upon the circumstances of the amputation, which must of necessity be uncertain in operations for injury where it is difficult to ascertain the limit of the sound tissues.

Obesity and plethora.—The very corpulent are certainly not good subjects for either injury or operation. In some of them wounds do quite well. These will probably be young persons in whom the disposition to corpulence is hereditary, who are in sound health, and take every reasonable means to prevent increase of weight.

All obese individuals about or beyond middle life are, as a rule, bad subjects in a surgical sense, and more especially the men. The excessive corpulence may have been induced by gluttony or drinking habits, or have been encouraged by indolence or disease. These patients often breathe with difficulty, and cannot assume the entirely recumbent position. They soon become helpless, their mere bulk renders it difficult for them to be moved in bed and for dressings to be applied; their skin is frequently unwholesome, and they are not readily kept clean.

The edges of the wound come ill together. The immense layer of subcutaneous fat is indifferently supplied with blood, and has probably been damaged. Portions of this tissue have been broken up and isolated from a blood supply. The thickness of the parts involves much strain upon the sutures. If after the operation the patient incline towards the affected side, the whole wound region becomes pendulous, drainage is difficult, and the application of pressure in the dressing of the part is almost impossible. In these circumstances, the wound is apt to become septic, sloughing is not uncommon, and deep-seated suppuration is comparatively frequent. A low type of inflammation often involves the surrounding skin, and the discharges become offensive and ill-conditioned. Such patients often die suddenly, others become exhausted or succumb to an intercurrent disease. The most favourable often make but a tardy recovery.

The treatment of fracture is often a difficult matter in the unwieldy bodies of the very corpulent.

Plethora as a simple condition does not compromise the success of an operation. Indeed, the "full-blooded" pass through a surgical experience well enough, provided that the plethora depend upon no diseased condition.

Alcoholism.—A scarcely worse subject for an operation or an injury can be found than is provided by the habitual drunkard.

The condition contra-indicates any but the most necessary and urgent procedures, such as amputation for severe crush, herniotomy, and the like. The mortality of these operations among alcoholics is, it is needless to say, very high.

Many individuals who state that they "do not drink," and who, although perhaps never drunk, are yet always taking a little stimulant in the form of "nips" and an "occasional glass," are often as bad subjects for surgical treatment as are the acknowledged drunkards.

Of the secret drinker the surgeon has indeed to beware.

Even abstinence from alcohol for a week or two before an operation does not seem greatly to modify the result.

A severe injury or an operation in an habitual or occasional drunkard is apt to be followed by an outburst of delirium tremens. It must not be assumed that an operation upon a subject of alcoholism must of necessity turn out badly. The evil result is, however, sufficiently frequent to justify a refusal to perform any but urgent operations, and the occasional fact that grave wounds in heavy drinkers may heal kindly and well is rather an illustration of good fortune than of surgical success. Such injuries as lacerated and contused wounds and compound fractures call for special anxiety when met with in the persons of those who drink to excess. The simple fact is that the tissues of the alcoholic appear to be capable of offering but little resistance to bacterial growth.

Tuberculosis.—On the whole, it may be said that tuberculous patients stand operations remarkably well, and this especially applies to children. In a large proportion of the cases the operation rids the patient of a long-abiding trouble, and a source of persistent irritation and weakness. It is sometimes surprising to note how a pale, wasted, cachectic-looking child will improve and gain in flesh and in looks almost directly after such an operation as amputation of the leg for the removal of a wholly carious foot. Some of the best examples—so far as a speedy recovery is concerned—of amputation at the hip-joint have been met with among tuberculous children.

It must be assumed that in these and in other cases there is a freedom from serious visceral disease such as lardaceous degeneration of the liver.

Wounds in the tuberculous are remarkably affected by their surroundings. The patient requires fresh air and the most favourable hygienic conditions. Results may be obtained at the seaside which can hardly be expected in the crowded wards of a city hospital.

The tuberculous patient has no great power of sound plastic repair. Healing may be rapid, but it is not always substantial. As Verneuil well says, operations upon the tuberculous abound in "half-successes, incomplete results, and unfinished cures."

Upon tuberculous patients of middle age operations must be undertaken warily. The wounds in these individuals, especially when they involve the diseased area, often do badly, heal but indifferently, and are apt to be associated with inflammatory processes of the lowest type.

On the question of operations upon the subjects of actual and active *phthisis* Sir James Paget writes as follows :—"The fever and other accidents that may follow an operation may do special harm to a tuberculous patient . . . . The fear of such a calamity should dissuade you from all operations of mere convenience, and from all measures of what may be called decorative surgery, in phthisical people; but it should not always dissuade you from operations that will cure diseases from which they suffer much, and by which their lives are wasted, as they are by fistula and diseases of the bones and joints. In these and the like cases the main question is whether the local disease—say, a diseased joint—is weighing on the patient so heavily or aggravating his phthisis and shortening his life so much as to justify an operation attended with more than the average risk of life and health.

"In all cases of acute or progressive phthisis great risk is incurred by almost every operation. . . . . The case is very different with chronic or suspended phthisis. In these it is often advisable to incur the somewhat increased risk of even a large operation, in order to free the patient from the distress and wasting of a considerable local disease, such as that of a joint; and I should be disposed to say that it is always advisable to cure, if you can, a small disease such as fistula. I say if you can, for you will often be disappointed."

Syphilis.—In the great majority of cases syphilis does not injuriously affect the prospects after injury or the course of an operation. If the patient be rendered cachectic, or be the subject of visceral disease, he is placed in the same unfavourable category with those who are similarly affected from other causes. Wounds during the progress of secondary syphilis more often heal well than show any evil tendency. Occasionally they become the seat of transient syphilitic manifestations, and heal indifferently, or break down after a speedy closure. Such an event may occur without the appearance of any distinct syphilitic change in the part. The same may be said of operations performed late in syphilis or many years after its occurrence. They usually do well. In the minority of the cases, however, primary healing is not secured; or the wound heals, and breaks down again; or remains open, and becomes the seat of a dull, persisting suppuration, or of an ulcer possessed of specific characters. This, perhaps, more often happens when the incision involves tissues which have previously been damaged by syphilitic disease. Thus it comes to pass that plastic operations not infrequently fail in syphilitic persons, especially when performed for the relief of deformities produced by some destructive manifestation of the disease itself. The healing of fractures has been noticed to be delayed occasionally in the subjects of syphilis, and repair to follow when the patient is put under specific treatment.

Rheumatism and gout have practically no effect upon the immediate future of an operation. The wound heals kindly and well. It is unnecessary to say that an operation should if possible not be performed during an outbreak of either of these conditions. It must be remembered, also, that any of the sequelæ of gout or rheumatism may complicate the issues of an operation. Such are the cardiac changes so often attendant upon the former disease, and the degenerations of the kidneys and other viscera which are apt in course of time to follow upon the latter.

An operation not infrequently determines an attack of gout, but such attack usually has no noteworthy effect upon the progress of the wound. Verneuil remarks that gout sometimes manifests itself at the site of injury by fluxions with acute pains, which simulate frank inflammation, and which, although of a temporary character, may suspend or retard the healing process.

Injuries involving the periosteum are apt to be followed by excessive action in that membrane when the patients are conspicuously rheumatic.

Cancer does not render a patient a bad subject for injury or operation. The result of the operation may be modified by other conditions, such as the age and temperament of the subject, and the presence of visceral disease. Cancer as such appears to exercise no effect upon the healing process. Indeed, operations for the removal of malignant growths in old and broken-down individuals often do remarkably well.

Anæmia, especially when due to loss of blood, has no special effect upon a surgical wound. The healing may be slow; the patient is perhaps rendered unduly liable to the more serious complications which follow upon wounds, and has little power to meet such misfortunes.

Leucocythæmia has a most disastrous influence upon operation wounds. Splenectomy, although performed many times in the subjects of leucocythæmia, has been followed by one uniform result—the patients have died.

Serious, if not fatal, results have followed in less grave procedures, and in the leucocythæmic person even a trivial operation is dangerous. They stand in great peril of hæmorrhage, and become the ready subjects of low forms of inflammation, of cellulitis, and allied conditions

Hæmophilia forbids a surgical operation of any but the most pressing kind, and forms a serious complication of any wound or severe contusion. The subjects of hæmophilia do not always bleed desperately after a wound; perhaps the most certain hæmorrhage will occur after wounds implicating the mouth. Still, a member of a "bleeder family," who has nearly bled to death from a slight accidental cut of the lip, may undergo an amputation of the foot with no more than the usual loss of blood.

Malaria.—The complex associations of malaria and injury are very clearly dealt with by Verneuil in the following passages:—

"Malaria may give rise, at the site of the injury, to various complications, such as hæmorrhage and neuralgia, complications which assume an intermittent type, and which yield to the employment of quinine.

"The influence of the poison, however, is not always shown by periodical disturbances. Certain wounds may assume a bad appearance, or at least remain stationary, until, the cause being suspected, quinine, which acts like a charm, is administered.

"The wound may occur under one of the following circumstances:—

"1. In a patient actually affected by intermittent fever. In this case the wound, especially if it be followed by hæmorrhage, rapidly and markedly aggravates the disease.

"2. In a patient who has previously been the subject of ague, but who appears to have entirely recovered. The wound, even when slight, may induce a fresh onset of ague; although the recovery from the last attack of fever was five, ten, fifteen, or even more years ago. On the other hand, the wound itself may become the seat of some local intermittent complications, the patient being free from the usual manifestations of the disease.

"3. In a patient who has never had intermittent fever, who is living in a healthy country, but who has formerly lived in a malarial district. The wound in such cases may apparently give rise to intermittent fever or to intermittent complications. It is clear that the injury, not being able of itself to produce a true intoxication, has merely provoked the explosion of a hitherto latent disease. These latter cases are not very rare, and are especially observed in large cities and in the healthiest regions."

Acute diseases, erysipelas, and inflammation.—It is needless to say that no operation, except such as is so urgent as to be necessary to save life, should be performed during the progress of any acute disease, such as pneumonia, an eruptive fever, and the like.

The same may be said of erysipelas. Incisions have to be made in the course of that disease to relieve the tension and to evacuate pus, but they cannot rank as operative measures. If an amputation be rendered necessary in a subject of erysipelas, the less danger would attend the postponement of the operation until the acute period of the fever had passed.

It is most important to avoid, when possible, any operation upon inflamed parts. This applies as well to so small an operation as the removal of a pile as to the excision of a large tumour. With operations in the present sense are not classed such surgical measures as are employed for the relief of inflammation.

Affections of the nervous system.—The mental state of a healthy patient as expressed by the terms "nervous," "neurotic," "excitable," "apathetic," has little definite effect upon the result of an operation or injury.

The least favourable frame of mind is that marked by gloom and utter apathy, and by a morbid, stoical indifference, difficult to dispose of.

Operations and injuries in *hysterical or epileptic* patients are apt to be complicated in their after-treatment by outbreaks of the nerve affection. While attacks of both hysteria and epilepsy are clearly often induced by an operation or injury, on the other hand a precisely opposite effect may follow the surgical measure.

The *insane* bear operation unusually well, provided that they are in sound health and amenable to treatment, and of cleanly habits. In many subjects of chronic mania, of melancholia, and dementia, the general health is quite broken down, and as a consequence they become unfit subjects for any operative treatment. In those of the insane, also, who are violent, restless, mischievous, or of very dirty habits, the success of any surgical measures may be frustrated by the patient.

In not a few instances insanity appears to have been induced by operation. The patients are mostly women, and the operation for the most part one concerning the breast or pelvic organs. The occurrence of this unfortunate circumstance is neither frequent enough nor sufficiently well defined to influence a surgeon in the performance of a necessary operation.

It is needless to point out that injuries affecting paralysed limbs or the lower extremities of the subjects of locomotor ataxia can scarcely be expected to turn out well. The gloomiest forebodings are, however, often not realised.

Diabetes offers a serious bar to any kind of operation, and injuries involving open wounds, hæmorrhage or damage to blood-vessels are exceedingly grave in the subjects of this disease. A wound in a diabetic patient will probably not heal, while the tissues appear to offer the most favourable soil for the development of putrefactive and pyogenic bacteria. The wound gapes, suppurates, and sloughs. Gangrene very readily follows an injury in diabetics, and such patients show a terrible proneness to a low form of erysipelas and of spreading cellulitis.

Diabetic gangrene of a limb is determined by many causes, among which especial attention must be given to inflammatory conditions, atheroma of vessels and peripheral neuritis. There was a time when amputation for diabetic gangrene was considered to be absolutely hopeless. Of recent years, however, this operation has been carried out with success. Three main factors have contributed to produce this result:—(1) The observation of antiseptic precautions of an exceptionally rigid kind; (2) the carrying out of a precise anti-diabetic treatment; and (3) attention to the condition of the arteries in determining the site of the amputation. Mr. Godlee, who has recorded some successful cases of amputation in diabetic gangrene, comes to the following conclusions upon this subject:—"A large proportion of the cases of gangrene of the lower extremities of diabetics will be found to depend either on arterial degeneration or on peripheral neuritis; and that in the former class it is probable that the changes in the arteries will extend at least as high as the knee. Whilst, therefore, it is right to amputate in those of the former class if the disease be progressing rapidly, not lower than the knee, those of the latter may either be left alone, or if amputation be undertaken it need not be at a great distance from the seat of disease."*

Visceral disease.—(1) *Heart disease and atheroma.*—In the matter of heart affections, it may be said that the patient whose heart is feeble or fatty, or embarrassed by valvular disease, is exposed to extraordinary risk from the shock of an operation; but apart from this, heart disease, if it have induced no widespreading tissue change, appears to add little to the danger of the undertaking. On the other hand, as Verneuil points out, valvular lesions and degenerations of the muscular tissue of the heart may, by changing the condition of the entire circulation, modify the composition of the blood, cause impairment of the viscera, alter the tissues, and bring about a condition very unfavourable to the healing process. Such patients show a disposition to passive hæmorrhages difficult to check, together with œdema of the wounded region, to patches of erythema, to erysipelas, and even to gangrene. There is a local atony which indefinitely delays healing and converts the wound into an ulcer.

Operations are often performed upon limbs the arteries of which are affected by atheroma. It is surprising how well ligatures maintain a hold upon such vessels, and how well they remain closed. The risk that would appear to be most pressing, that of secondary hæmorrhage, is in actual practice seldom encountered. That wounds in such patients are more liable to secondary bleedings than are wounds involving parts supplied by normal arteries is true, but the occurrence is not frequent. The real risks in these cases are from gangrene, from sloughing of the flaps of an amputation, or from the breaking down of the simplest wound, and from diffuse inflammations of a low type.

(2) *Lung disease.*—The relation of phthisis to injuries has been already considered. Any chronic lung affection, such as chronic bronchitis, usually indicates impaired health, and offers difficulties in the after-treatment on account of the embarrassed breathing, the

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* Medico-Chirurgical Transactions, 1893.

disturbance of parts produced by coughing, and the imperfect oxygenation of the blood. Operations and injuries in such individuals can hardly be expected to follow a quite even course.

(3) *Affections of the alimentary canal.*—In the matter of affections of the alimentary canal there is little to be said. The effect that any disease of the stomach or intestines may have upon an operation is to be measured by the effect it has on the general health. The subject of chronic dyspepsia can hardly be well nourished, and the subject of habitual constipation is burdened with a trouble which an operation serves to complicate. It is unnecessary to state that an operation should be avoided, if possible, during the course of diarrhœa or dysentery.

(4) *Diseases of the liver.*—Affections of the liver have a very injurious influence upon operations and injuries. Even the slighter forms of hepatic trouble serve to compromise the future of an operation, and the more defined diseases of the liver have a definite ill-effect upon surgical wounds. Such are cirrhosis of the liver and the conditions of fatty or amyloid degeneration. Advanced forms of these affections offer a serious bar to operation. Operations performed in the earlier stages of the former disease will certainly be injuriously affected. The subject of cirrhosis is probably a drunkard; the subject of amyloid degeneration the victim of long-continued suppuration.

The risks these patients run are numerous. Some succumb to shock, others die of exhaustion. In all there is a probability that the wound will not heal, but that it will slough and suppurate, and become the seat of spreading inflammation of a low type. Pyæmia was unduly common in these patients.

No question is more difficult to decide than that which concerns the period in the progress of lardaceous disease of the liver, beyond which it is practically unjustifiable to operate. In the quite advanced stages of the disease a serious operation is certainly not justifiable. In the earlier and middle periods an operation such as an amputation may be performed with ultimate admirable success, for it not only rids the patient of his trouble—probably a suppurating joint—but it removes the cause of the visceral complication.

(5) *Kidney disease.*—It may be safely said that the results of operations and injuries are more powerfully influenced by diseases of the kidneys than by a corresponding disease of any other organ. An operation upon the subject of advanced Bright's disease or of surgical kidney is a desperate matter. A patient may look fairly healthy, may appear well nourished, may be temperate and living a most regular life, and the operation may be but a trifling one; yet the complication of albuminuria renders the surgical procedure most serious and hazardous. Many an elderly man has died almost suddenly from the effects of rough catheterisation, and it has been found after death that he was the subject of an unsuspected pyelitis.

Quite slight operations of no urgency—such as that for the relief of Dupuytren's contraction of the palmar fascia—have placed the subjects of Bright's disease in great danger of death.

In no case should an operation on an adult be undertaken without a preliminary examination of the urine.

It is true that in some instances—as in a form of albuminuria met with in connection with large abdominal tumours—the existence of the albumen is no bar to an operation. It is true also that patients with Bright's disease have recovered admirably from large operations. The fact remains that organic disease of the kidney is one of the most serious complications with which the surgeon can be concerned.

The subjects of kidney disease exhibit nearly the same evil tendencies after operation as have been alluded to in dealing with hepatic troubles. They are exposed to the additional risk of death from suppression of urine and uræmia. Such patients often die of exhaustion many days, or even a week or more, after the operation. They are especially prone to all the evils incident to wounds. Primary healing can never be depended upon.

A plastic operation is unjustifiable in a subject of kidney disease. The operation wound is liable to break down, to suppurate, and be the seat of secondary hæmorrhage, of erysipelas, of cellulitis, and even of gangrene. When pyæmia was common in hospital wards, the subject of kidney disease became its readiest victim. Surgeons have learnt how to ward off pyæmia, but they have yet to learn how to meet the complications of Bright's disease.

XIV. ANÆSTHETICS.

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For the prevention of pain during surgical operations two recognised modes of procedure are open to the surgeon. He may either induce and maintain a state of general anæsthesia by the administration, for example, of nitrous oxide, ether, or chloroform; or he may bring about a condition of local anæsthesia by the use of such agents as cocaine, the ether-spray, or chloride of ethyl. Speaking generally, the former plan is applicable and reliable in every case in which a painful operation is contemplated; whilst the latter, in addition to its having but a limited range of applicability, is in many cases distinctly unreliable.

I. GENERAL ANÆSTHESIA.

1. Preparations and precautions.

These may be briefly summarised.

(1) Putting aside nitrous oxide, for which no very special preparation is needed, the bowels should be thoroughly evacuated by appropriate means. This is usually best effected by a purgative the day before, and an enema on the morning of the operation. The bladder, too, should be empty.

(2) Whenever practicable, in ordinary surgical practice, an interval of from four to five hours should have elapsed since the last meal. In the case of nitrous oxide, about three hours' fast is advisable. In exhausted subjects, some clear soup or beef-tea may be allowed three hours before the operation. A little brandy or whisky, with an equal quantity of water, is permissible fifteen minutes before the operation when signs of faintness are present.

(3) The early morning (8 a.m. to 9.30 a.m.) is the best time for ordinary surgical operations.

(4) The administrator should on all occasions observe the patient's colour, respiration, and pulse. Should any marked departure from the normal be detected, a stethoscopic examination should be made.

(5) That anæsthetic should be selected which is most suited to the particular case.

(6) The mouth should be inspected, and artificial teeth removed. Inadequacy or absence of nasal breathing should not escape attention.

(7) All constricting clothing should be unfastened, even for the most trifling operation.

(8) Except in the case of dental and similar operations, the patient should be recumbent, in a comfortable posture. A position half-way between the supine and lateral is often assumed for choice. The head should be turned to one side.

(9) A third person should always be present during the administration.

(10) No case should be regarded as trivial. Difficulties and accidents may arise when anæsthetising a perfectly healthy patient with the safest known anæsthetic for a minor operation.

(11) The anæsthetist should devote his undivided attention to his patient, and should take no part in the operation.

(12) He should have at hand:—(*a*) Appliances for opening the mouth, and maintaining it in that position: *e.g.* a wooden wedge, a Mason's gag, and a small dental mouth "prop"; (*b*) a pair of tongue forceps; (*c*) instruments for tracheotomy. Of the various restorative remedies, brandy, nitrite of amyl, tincture of digitalis, and liq. strychninæ hydrochlor. are the most reliable.

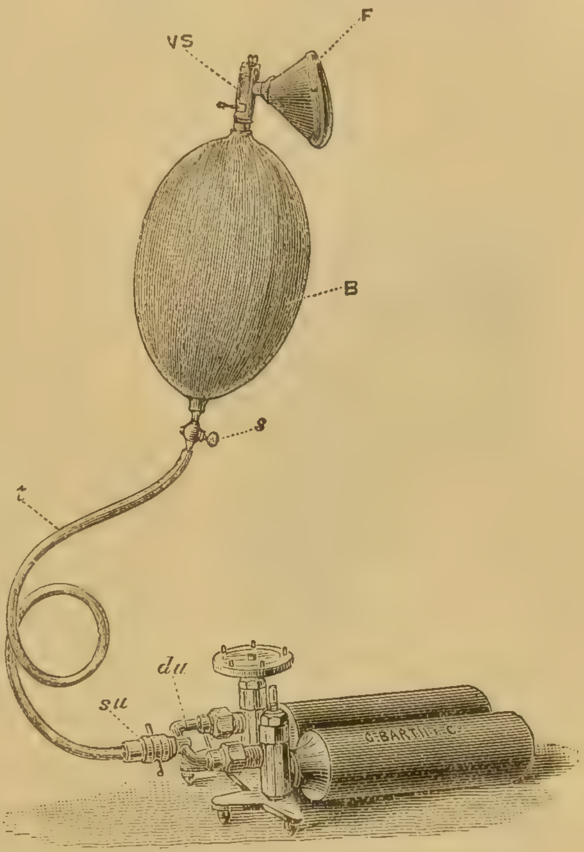

Fig. 47.—Apparatus for administering Nitrous Oxide.

F, Face piece; VS, valved stopcock; B, gas bag; *s*, stopcock; *t*, rubber tube; *s u*, single union; *d u*, double union. Two cylinders of nitrous oxide with foot key are also shown.

2. The chief methods of producing general anæsthesia.

1. **By nitrous oxide free from air or oxygen.**—The most convenient apparatus is shown in Fig. 47. To ensure success, an accurately-working apparatus possessing wide channels, efficient valves, and a face-piece which exactly fits the patient's face are

indispensable. The bag having been partly filled with gas, the face-piece is carefully adjusted. After a few breaths of air through the apparatus, nitrous oxide is allowed to enter the face-piece. The bag is kept fairly full by rotating the foot-key. Respiration grows deeper and quicker, the features become dusky or cyanotic, the pulse is accelerated, and the pupils usually dilate. In from thirty to seventy seconds respiration loses its rhythm and becomes "catchy," jerky, or tumultuous; stertor may be heard; and clonic muscular movements of the face and extremities appear. The administration of nitrous oxide for a dental operation is usually carried to this point; the face-piece is then removed and the operation begun. An anæsthesia of about thirty seconds remains after the face-piece is removed. Re-breathing is not, as a rule, advisable. But should the nitrous oxide unexpectedly fall short, a few moments of to-and-fro breathing towards the end will prevent the failure to anæsthetise which might otherwise occur.

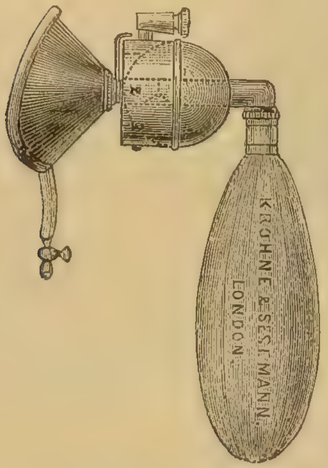

Fig. 48.—Clover's Portable Regulating Ether Inhaler.

2. **By nitrous oxide mixed with air.**—By giving an occasional breath of air through the stopcock during the administration of nitrous oxide, the inhalation may be so lengthened that operations lasting for several minutes may be performed. Care must be taken not to give too much air, as this would interfere with anæsthesia. On the other hand, too little would render the administration difficult, owing to the stertor and spasm which would result. In dental practice an occasional breath of air with nitrous oxide, by allowing a longer inhalation, leads to a longer after-anæsthesia.

3. **By nitrous oxide mixed with oxygen.**—A special apparatus is required. The anæsthesia produced is the safest and most perfect with which we are acquainted. The asphyxial phenomena of ordinary nitrous oxide anæsthesia (cyanosis, stertor, and "jactitation") are prevented, and a deeper and more sleep-like anæsthesia results. The mixture is known as "schlafgas" in Germany. The method is valuable (1) in patients with serious cardiac or pulmonary disease; (2) in very old persons; (3) in anæmic, cachectic, and feeble subjects; and (4) in children. In addition to its value in dental surgery, it is very useful for short operations within the nose and throat, and in orthopædic surgery.

4. **By ether, with Clover's regulating inhaler.**—This ingenious apparatus is shown in Fig. 48. It consists essentially of (*a*) a face-piece carrying a whistle-shaped metal tube fitting into (*b*) a spherical metal ether reservoir, and (*c*) a bag When the reservoir is rotated on the whistle-shaped tube attached to the face-piece, more and more of the current which passes between

the patient and the bag is made, by an ingenious arrangement of slots, to travel over the ether in the reservoir. An indicator pointing to "0," "1," "2," "3," and "F" (full) on the circumference of the reservoir shows the extent to which the air current is thus deflected. It must be remembered that fresh air can only be admitted by removing the whole inhaler, or its bag, from time to time. To use the apparatus: (1) Pour out any ether which may have remained from a previous administration. (2) In cold weather partially immerse the reservoir in lukewarm water for a minute. (3) Pour in 1½ oz. of pure ether. (4) Accurately adapt the face-piece to the face. (5) Fit on the bag. By pressing the face-piece rather more during expiration than inspiration the bag will become distended with expired air. (6) Keep the indicator at "0" for half a minute. (7) Very gradually rotate the ether reservoir, so that the "0" moves continuously, but almost imperceptibly, away from the indicator. (8) Continue to rotate the reservoir, giving no fresh air till stertor commences. The inhaler may then be removed for one inspiration of air, but care should be taken to give no more than this for the present, and rather to increase than to diminish the strength of ether vapour. (9) When the breathing has become regular and stertorous, and the cornea insensitive to touch, a few more inspirations of fresh air may be allowed. (10) Generally speaking the indicator may be kept between "1" and "2" after the patient has been placed well under. The inhaler should be removed for fresh air about every ten or twelve breaths. Feeble subjects should be allowed more air and less ether than more vigorous persons.

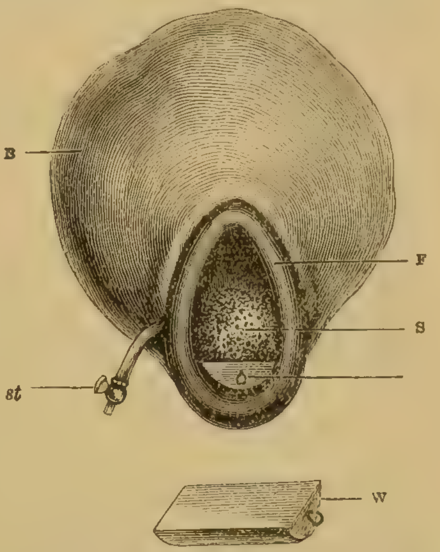

Fig. 49.—Modified Ormsby Ether Inhaler.

F, Face-piece; B, bag; S, sponge for ether; W, water chamber for preventing freezing of sponge; st, stopcock for inflating cushion of face-piece.

In deep ether anæsthesia the eyes are fixed, the cornea is insensitive to touch, the pupils are moderately dilated, mucus and saliva are freely secreted, especially in children and young persons, and the muscular system is relaxed. The most characteristic feature of the anæsthesia, however, is the remarkable circulatory and respiratory activity. The face is flushed, the heart's action increased in rate, and incised parts bleed very freely. Physiological experiments have shown that the blood pressure falls but slightly, whilst the heart's cavities do not dilate as in the case of chloroform (MacWilliam). Respiration is usually deep, quick, regular, and stertorous.

5. **By ether with Ormsby's inhaler.**—Fig. 49 shows the

most recent modification of this apparatus. From 2 to 4 drachms of the purest ether should be poured upon the previously moistened sponge, and the face-piece gradually applied. Swallowing, temporarily suspended breathing, and coughing are not uncommon, even with care. Excitement and struggling are more frequently met with than when using Clover's apparatus. Gradually, however, the breathing becomes more regular and stertorous, and the usual signs of ether anæsthesia appear. Fresh air should be allowed, by removing the inhaler, about as often as with Clover's inhaler. This method is inferior to Clover's for *inducing* anæsthesia; but when consciousness has been destroyed by nitrous oxide or the A. C. E. mixture, Ormsby's inhaler is particularly useful for *maintaining* anæsthesia with ether.

6. **By ether. "Open" method.**—A towel folded into the shape of a cone, at the apex of which a sponge wrung out of tepid water is placed, may be employed; but a Rendle's mask (Fig. 50) of leather or celluloid, partly covered with domett, is more convenient. The ether should be gradually given at first; but when excitement commences the anæsthetic should be freely pushed, and the usual signs of anæsthesia will be produced, though often rather tardily. The method is wasteful and inferior to others for general use. It may be advantageously chosen, however, in cases in which air limitation would be open to objection, and in those in which very little anæsthetic indeed is needed.

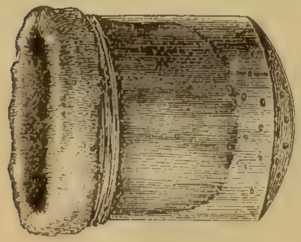
Fig. 50.—Rendle's Mask, made from Celluloid.

7. **By nitrous oxide, followed by ether.**—These anæsthetics may be thus given in succession. An ordinary nitrous oxide apparatus (Fig. 47) and an Ormsby's ether inhaler (Fig. 49) are employed. Nitrous oxide is administered in the usual way till jerky respiration and clonus occur. One inspiration of air is then allowed, and the ether inhaler, previously charged with ether, is applied during the following expiration. The next few inspirations are usually cut short or prevented by the strong ether vapour, and considerable cyanosis is occasioned for a few moments. Respiration soon becomes more regular, however, and at this juncture fresh air may be allowed in sufficient quantity to re-establish the colour. When anæsthetising children, feeble subjects, and old persons, somewhat more air should be allowed during the transition from "gas" to ether than is generally advisable. This particular method is well adapted for anæsthetising children, adolescents, and very nervous women. Nitrous oxide and ether may also be given together by employing the stopcock and gas-bag of Fig. 47 fitted to a Clover's inhaler (Fig. 48). The bag, filled with "gas," is attached to the charged ether chamber, and the face-piece is applied. Air is at first breathed; nitrous oxide is then turned on, and half the "gas" breathed out through valves; the remaining half is then breathed to

and fro by turning the valves off; and ether is admitted in increasing quantities by rotating the ether chamber. When clonic movements, deep stertor, or much cyanosis arise, one breath of air should be given. The administration is subsequently conducted as already described (page 274: paragraph 4). This particular method gives better results than the preceding in dental practice and in anæsthetising powerfully-built or alcoholic men for ordinary surgical operations.

8. **By chloroform. "Open" method.**—This is to be preferred to other methods for ordinary cases. A double thickness of lint pinched up into the shape of a small fan, the corner of a towel drawn through a safety-pin, a Skinner's mask (Fig. 51), or any similar arrangement permitting free access of air, may be employed. Closely-fitting inhalers should never be used. The best form of drop-bottle for sprinkling the chloroform is that known as Thomas's. A few drops of the anæsthetic should first be dropped upon the lint, towel, or mask, which should be held a trifle away from the face. The object of the anæsthetist should be to keep up the more or less continuous administration of a well-diluted vapour; not to give the anæsthetic intermittently and irregularly. He must steer between two extremes. On the one hand, too strong a vapour, with the absence of sufficient air, will cause struggling, rigidity, and suspended breathing; on the other hand, too dilute a vapour, or the intermittent administration of very small quantities at a time, will cause delay, imperfect anæsthesia, pallor, feebleness of pulse, and vomiting. These remarks apply more particularly to moderately healthy and vigorous subjects, not to exhausted or feeble persons. The few drops first sprinkled on the lint should be quickly followed by a few drops more, and the lint more closely applied. Any holding of the breath should be met by slightly removing the lint. As the administration proceeds, respiration grows audible and deeper. Incoherent speech, excitement, and muscular rigidity are not uncommon in certain subjects. Should respiration be temporarily suspended during the stage of rigidity, the chloroform should be removed during the deep and quick breaths which follow, but re-applied gradually as these breaths lessen in force and frequency. If this precaution be not adopted, an over-dose may be given; for the circulation, having already absorbed a considerable quantity of chloroform prior to and during the suspended breathing, will quickly absorb a considerable quantity more during the forcible respirations which follow. Sometimes there is no rigidity or excitement. But under either set of circumstances the breathing gradually becomes regular and softly snoring, the dilated pupils of partial anæsthesia grow smaller, the muscular system becomes completely relaxed, and the corneæ

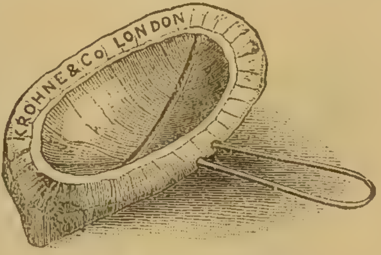

Fig. 51.—Skinner's Mask.

insensitive to touch. The patient is now ready for any surgical operation. The circulation under chloroform is not as vigorous as that under ether. The pulse is slower than normal (in most cases), and incised parts bleed less freely than under ether. All physiologists agree that a considerable fall in blood pressure takes place. According to the Hyderabad Chloroform Commission,* the fall of pressure which occurs with regular breathing is of vaso-motor origin, and is, "if not a safeguard, absolutely harmless." Whilst admitting that the fall is at first chiefly due to vaso-motor paralysis, MacWilliam has shown that actual dilatation of all cavities of the heart occurs under chloroform, and that the fall of pressure is, in the later stages of administration, largely due to this cause. Gaskell and Shore, in another important contribution to the subject, maintain that the effect of chloroform on the vaso-motor centre is rather one of stimulation than depression, and attribute the fall to the direct action of chloroform upon the heart. More recently, Thornton and Hare have gone over the ground again, and urge that the vaso-motor paralysis is the dominant factor in the fall of blood pressure, but admit that cardiac depression may contribute, especially when the heart is previously diseased. They disagree with the Hyderabad Commission that the low blood pressure is in any way a safeguard; and, in conclusion, corroborate MacWilliam's statement that dilatation of the heart's cavities is invariably produced in deep chloroform anæsthesia. Quite recently, Pickering has shown, by experiments on the heart of the chick embryo—a heart not yet possessing nervous connections with the central nervous system—that chloroform has a distinctly depressing effect upon the cardiac muscle itself.

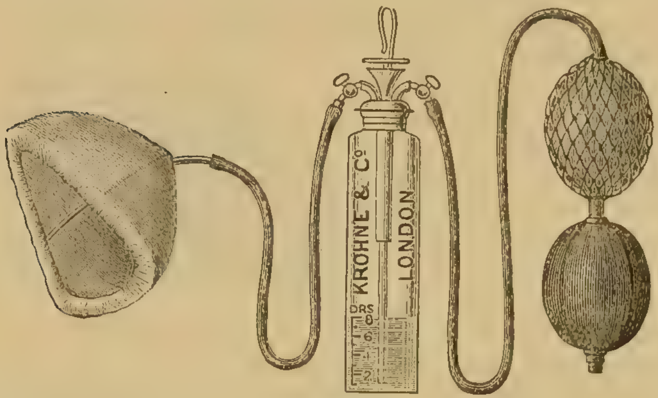

Fig. 52.—Junker's Inhaler, with Flannel Face piece, for administering Chloroform.

In administering chloroform, attention should be paid to the following points:—(1) The respiration. This should either be heard or felt throughout. A soft regular snoring is to be encouraged. (2) The pulse. This should be carefully watched, in addition to the breathing. (3) The colour. Simple cyanosis points to some interference with respiration, whilst circulation is but little deranged; it is, hence, common when the air-supply is restricted or when the respiratory movements are inadequate. Pallor, the indication of circulatory depression, may arise from surgical shock, impending vomiting, inadequate breathing, or the administration of an over-dose. (4) The corneal reflex. (5) The pupil. A minutely-contracted pupil usually, but by no means necessarily, means a rather light anæsthesia. A moderately-contracted pupil is common in proper chloroform anæsthesia. A moderately-dilated or dilated pupil may either mean a deep or a light anæsthesia. In some cases a widely-dilated pupil is present throughout perfectly established anæsthesia. The pupil is of little use as a guide till the patient has been anæsthetised for some little while, and even then the effects of more or less of the anæsthetic must be observed before inferences can be drawn. (6) The act of deglutition. This is absent in deep anæsthesia. (7) The state of the muscular system. (8) Phonated expiratory sounds. These point to moderately deep anæsthesia.

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* Report, p. 137.

9. **By chloroform with Junker's apparatus.**—The latest form of this inhaler is represented in Fig. 52. By working the hand bellows, air is made to bubble through the chloroform in the bottle, thus becoming charged with anæsthetic vapour. The face-piece into which this chloroform-laden air is pumped should fit the face loosely, so that the vapour may be freely diluted with fresh air. Having made certain that the inhaler is in working order, the anæsthetist should apply the face-piece with one hand, whilst he pumps gently, steadily, and rhythmically with the other. The great advantage of Junker's inhaler is that a more or less equable and well-diluted chloroform vapour may be continuously administered. Its disadvantages are that its management occupies a considerable share of the anæsthetist's attention, and that difficulty may be experienced in producing deep anæsthesia with it, especially in vigorous or alcoholic subjects. But Junker's inhaler is of great value for keeping up anæsthesia during operations upon the mouth or nose. For these cases a bent metal mouth tube, or a silk catheter for introduction through the nose, may be attached to the flexible exit tube of the inhaler, in place of the face-piece. The modified Mason's gag of Fig. 53 is useful in some cases; the flexible tube of Junker's apparatus being attached to the metal tube running along the arm of the gag.

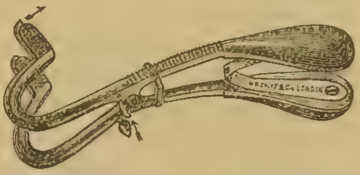

Fig. 53.—Mason's Gag, fitted with Tubes for the Transmission of Chloroform Vapour.

10. **By the A. C. E. mixture.**—This mixture, which consists of one part of alcohol, two of chloroform, and three of ether (George Harley), may be conveniently administered by means of a Skinner's or Rendle's mask (Figs. 50 and 51). For infants and young children the former may be used throughout; but for adults the latter should be substituted after partial anæsthesia has been secured. The anæsthesia produced resembles that of chloroform in most respects; but respiration is deeper and circulation more active. There are two important points in administering this mixture: viz.

that small quantities only (half to one drachm) should be added at a time; and that fresh air should be freely admitted throughout.

11. **By the A. C. E. mixture, followed by ether.**—This is an excellent method of producing anæsthesia in many cases. A Rendle's or Skinner's mask (Figs. 50 and 51), or both in succession, should be used for the mixture; and an Ormsby's inhaler (Fig. 49) for the ether. Partial anæsthesia having been induced by the mixture, the Ormsby's inhaler, charged with ether, should be gradually applied. Excitement and struggling are rare.

12. **By morphine with a general anæsthetic.**—The injection of from $\frac{1}{6}$ gr. to $\frac{1}{3}$ gr. of morphine, with or without $\frac{1}{120}$ gr. of atropine, is strongly recommended by some as a preliminary measure before general anæsthesia. The susceptibility of the patient to morphine should be previously ascertained, and his general state of health carefully considered before this mixed narcosis is employed. The injection should be made about twenty minutes before the hour fixed for operation. When patients are thus treated, or when an opiate has for other reasons been given, it will be found that much smaller quantities of the anæsthetic than usual are required; that excitement, struggling, rigidity, salivation, and cough are exceptional; and that it is often possible to keep up a passive and analgesic condition with extremely small doses of the anæsthetic. The combination of morphine and chloroform is said to prevent inconvenient vascularity in cerebral operations (Horsley); and the analgesic state which may be secured by the method has been found of service in the surgery of the mouth and nose (Thiersch). In giving an anæsthetic after morphine, care must always be taken to administer as little as possible, otherwise the respiratory functions may become depressed.

Other methods.—The so-called "bichloride of methylene" (a mechanical mixture of chloroform and methylic alcohol), ethidene dichloride, bromide of ethyl, and amylene or pental, are inferior in one or more respects to the anæsthetics above considered.

3. The choice of the anæsthetic and of the method of its administration.

Looking at the matter in its broadest aspect, we may arrange cases requiring a general anæsthetic in two main groups: (1) those presenting no special peculiarities—in other words, *ordinary or average cases*; and (2) those in which some departure from our usual method of procedure is necessary—these may be called, for purposes of description, *exceptional cases.*

1. **The choice of anæsthetics and methods for routine use in ordinary cases.**—Our endeavour should be to select that anæsthetic which has the least risk to life attached to it. It hence follows that, whenever practicable, nitrous oxide should be chosen. When this gas is administered as has been described, it will be found that many operations often regarded as beyond its range may be successfully performed, so far as the avoidance of all pain is concerned. In the event of nitrous oxide being inapplicable, as it is in most surgical cases, the next safest anæsthetic—viz. ether—should,

if possible, be chosen. Ether may often be satisfactorily given although apparently contra-indicated. For general application Clover's method (Method 4) is the best; but if the anæsthetist is desirous of sparing the patient the initial discomforts of ether, he may either give nitrous oxide (Method 7) or the A. C. E. mixture (Method 11) as a preliminary anæsthetic.

2. **The choice of anæsthetics and methods for special patients and cases, the operation being of such a nature that nitrous oxide alone is inadmissible.** *Age, temperament, general physique, etc.*—For infants the A. C. E. mixture on a Skinner's mask (not a cone) answers well; but ether may be safely given on a cone of lint. Chloroform should be administered on lint, on an open Skinner's mask, or by means of Junker's apparatus with flannel face-piece (Fig. 52). For children of from two to four years the A. C. E. mixture should be given to partial anæsthesia, and then ether from a Clover's or Ormsby's inhaler substituted. For senile subjects (over 70 years), the A. C. E. mixture (Method 10) answers best; should any respiratory difficulty appear, chloroform should be substituted: and only small quantities of the anæsthetic will be needed when once anæsthesia has been produced. Extremely neurotic and hysterical women are best anæsthetised by nitrous oxide and ether (Method 7). Stout, flabby, breathless subjects, with a feeble circulation and a congested appearance, are best anæsthetised by the A. C. E. mixture, followed by ether (Method 11), or by the A. C. E. mixture alone (Method 10). For anæsthetising very muscular and vigorous men or alcoholic subjects, ether is preferable to all other anæsthetics; it may be advantageously preceded by nitrous oxide or the mixture (Methods 7 and 11).

Affections of the respiratory system.—In advanced chronic bronchitis, emphysema, or phthisis, the A. C. E. mixture usually gives the best results. A very profound anæsthesia must be avoided. Should any difficulty arise, chloroform may be substituted. When much dyspnœa is present, the last-named anæsthetic should be used from the first. Chloroform is also indicated when cyanosis from laryngeal disease, pressure upon the trachea, abdominal distension, or similar causes, is present. In long-standing pleural disease ether may be successfully given if care be exercised. (*See* next page: Operations upon the pleura or lung.)

Affections of the circulatory system.—When anæsthetising patients with advanced affections of the heart, pericardium, or blood-vessels, all unnecessary strain upon the circulation should be avoided. In most cases the A. C. E. mixture cautiously given (Method 10) answers admirably. Should much respiratory difficulty pre-exist, or should it arise under this anæsthetic, chloroform must be used. In very exhausted or collapsed patients, ether given in small quantities, and with more air than is customarily allowed, produces the best results.

Affections of the central nervous system.—If the patient be lethargic or semi-comatose from cerebral disease, very little anæsthetic will be required. In such cases the use of morphine before ether or chloroform should be avoided. In anæsthetising epileptics a mouth-prop should be placed between the teeth, in case an attack occurs.

Renal disease.—Patients with advanced renal disease may be safely anæsthetised by ether, provided that other morbid conditions contra-indicating this anæsthetic are absent.

Menstruation, pregnancy.—It is as well to withhold anæsthetics during the menstrual period. Pregnant women should be anæsthetised by methods which are not likely to induce convulsive movements, struggling, coughing, or vomiting.

Operations within or about the nose or mouth.—The best plan in these cases is first to place the patient well under ether (preceded, if desired, by nitrous oxide or the A. C. E. mixture), and then to keep up unconsciousness with chloroform. Many operations (*e.g.* those for the removal of adenoids, tonsils, or nasal polypi, as well as those for the extraction of numerous teeth) may be very conveniently performed after a free administration of ether, no further application of the inhaler being necessary. But for long operations on the jaws, tongue, palate, etc., this plan hardly suffices, so that a change to chloroform should be effected, the conjunctiva being allowed to regain its reflex to a slight extent before the chloroform vapour is pumped into the mouth or nose by the Junker's apparatus already described (Method 9). The sitting posture is only admissible when ether is employed. When it is necessary to give chloroform, either from the commencement or after ether, the patient should, if convenient to the surgeon, either be placed upon his side, with his cheek upon the pillow—an excellent posture in many cases—or upon his back, with his head completely extended over the end of the table. Should any other posture be necessary, the anæsthetist must be careful, lest blood accumulate in undesirable quantities above or within the larynx. As a rule, only a moderately deep anæsthesia should be maintained. If the hæmorrhage is profuse, either repeated sponging will be needed or the head must be turned to the side occasionally for drainage. Unattached sponges, so large that they can be just introduced into the mouth, answer well for sweeping round the back of the throat, and several of them should be at hand.

Operations in the region of the neck.—Ether produces so much vascular engorgement in this region that many surgeons greatly prefer chloroform or the A. C. E. mixture. Chloroform is certainly the best anæsthetic for tracheotomy, laryngotomy, and operations upon the thyroid gland. Cardiac depression from interference with the vagus may arise in some cases of this class.

Operations upon the pleura or lung.—In most cases of empyema a small quantity of the A. C. E. mixture should be first administered on a Skinner's mask, and then ether should be slowly given from a Rendle's inhaler. Deep anæsthesia must be avoided. If ether be badly borne, the mixture or chloroform may be substituted. In recent cases of empyema, with fever and cyanosis, general anæsthesia even of moderate degree, is very hazardous.

Abdominal operations.—In most cases ether may be employed. When once the patient is fully anæsthetised, Ormsby's inhaler produces better results than Clover's. Jerky breathing and venous engorgement may be due to (1) too great a restriction of air, (2) too much ether, or (3) some easily remedied obstruction about the mouth or pharynx. Should any delay arise in securing tranquil respiration or complete relaxation, chloroform should be substituted, care being taken to administer this anæsthetic in small quantities so long as respiration remains quick and forcible. Generally speaking, a deep anæsthesia should be maintained throughout, in order to avoid rigidity, retching, and coughing. In most cases of intestinal obstruction, however, a deep anæsthesia is usually contra-indicated, owing to the patient's exhausted condition. In such cases it is important that the head should be kept on its side throughout, for vomiting may occur very quietly; and if the head be in the mid-line, asphyxial symptoms of a grave character may arise.

Operations upon the genito-urinary organs and rectum.—Very deep anæsthesia is needed in these cases. For lithotrity and suprapubic cystotomy in fat or elderly subjects, the A.C.E. mixture usually answers well.

Operations upon the brain and its membranes.—The A.C.E. mixture or chloroform should be chosen, owing to the turgid state of the vessels under ether. (*See* Method 12, page 280.)

4. The difficulties, accidents, and dangers of general anæsthesia.

1. Respiratory.

(*a*) **Difficulties in or failure of respiration, due to the presence of some mechanical obstruction to the free entry or exit of air.**—Mechanically-obstructed breathing during anæsthesia is more common than is generally supposed. As examples the following conditions may be cited. The lips of edentulous subjects may become approximated, thereby preventing inspiration. The tongue may obstruct respiration in three ways: it may become greatly enlarged from vascular engorgement; it may be drawn backwards by muscular spasm (as in deglutition) during the stage of rigidity preceding deep anæsthesia; or it may gravitate towards the pharyngeal wall when the head is in the mid-line and the muscular system relaxed. Pre-existing inadequacy of the nasal passages greatly favours the supervention of embarrassed breathing, especially in muscular subjects with good teeth. One of the commonest causes of temporarily suspended breathing is partially performed deglutition, the larynx remaining pushed up against the epiglottis. Laryngeal spasm, known by inspiratory stridor of high pitch, is common in moderately deep anæsthesia. It is often connected with the presence of a plug of mucus within the larynx or with a small quantity of blood, and subsides after a few coughs. The spasm may, however, be reflex—due to the operation. The condition is not dangerous under ether; but the impeded respiration to which it necessarily leads must be carefully watched when using chloroform. Patients with enlarged tonsils should be anæsthetised with caution, as the stertor induced sometimes passes into complete obstruction. Among the adventitious substances which may impede or arrest breathing may be mentioned vomited matters, blood, mucus, pus, portions of morbid growth, extracted teeth, previously loose teeth or tartar, and dental mouth "props."

The *treatment* which should be adopted for the relief of obstructed breathing is simple. An attempt should first be made to remove the cause. Putting aside the presence of foreign bodies, obstructed breathing may in nine cases out of ten be immediately relieved by pushing the lower jaw forward from behind, care being taken to see that the teeth are not "locked." This manœuvre not only brings the tongue away from the pharynx, but the epiglottis from the larynx. It may advantageously be combined with extension of the head and neck. Should it not succeed, the mouth must be opened by means of a wedge or gag, and the finger passed to the base of the tongue, which should be hooked forwards. Should this not answer, the tongue must be pulled forwards vigorously with the tongue forceps. If this produces no good effects, the chest should be forcibly compressed from the front and sides. Lastly, should breathing still remain obstructed, laryngotomy must be performed. Should any of the foreign bodies above enumerated be the cause of the obstruction, steps must, of course, be immediately taken for their removal, or for the admission of air by laryngotomy. If, after a free air-way to the lungs has been established, respiration still remains in abeyance, artificial respiration should be performed. (*Vide infra.*)

(*b*) **Threatened or complete failure of respiration occurring independently of any mechanical obstruction.**—Respiratory failure of this kind may arise (1) from paralysis of the respiratory centres, or (2) from spasm of the respiratory muscles.

(1) Paralytic cessation of respiration is most commonly due to an over-dose of the anæsthetic; but it may be connected with intercurrent asphyxia, syncope, or morbid states of the respiratory system. When chloroform is given in toxic quantities, the paralysis of breathing which occurs is almost invariably associated with an extremely feeble pulse and pallor; whereas with ether the circulation is not dangerously depressed at the moment when respiration ceases, save in the case of patients in a very feeble state of health before the administration.

The treatment which should be immediately adopted in paralytic respiratory failure is artificial respiration. In minor cases, and when the chest walls are sufficiently elastic, rhythmic compression of the thoracic parietes will suffice; but in ordinary cases Silvester's method should be systematically applied. The patient should be placed horizontally, with his head over the end of the table or side of the bed. His tongue should be drawn out, to avoid obstruction. The anæsthetist, standing behind his patient, should grasp the arms just above the elbows and press them forcibly and steadily against the sides of the chest (expiration). He should now slowly extend them over the patient's head (inspiration). This double procedure should be repeated regularly about twenty times in each minute. Care must be taken that air really enters the chest. Should sufficient help be at hand, expiration may be assisted by making abdominal pressure.

(2) Spasmodic respiratory failure is apparently most common during the stage immediately preceding complete narcosis, and is probably more liable to arise in muscular and in emphysematous subjects than in others. It has proved fatal on several occasions, owing to the impossibility of overcoming the rigid fixity of the chest. It may co-exist with obstruction to breathing. Minor cases may yield to Silvester's artificial respiration or to forcible compression of the chest from below the costal margin or from the sides. But in the graver forms of spasm, direct lung inflation by bellows through the mouth or through the opened larynx appears to hold out the best chances of success.

2. CIRCULATORY.

There is one fact which stands out prominently in connection with circulatory failure during anæsthesia: namely, that except when this failure occurs as the result of the surgical procedure, it is in the great majority of cases connected with or dependent upon embarrassed or suspended breathing, and must, therefore, be treated by measures directed towards the re-establishment of respiration. Medical literature abounds with instances of misdirected treatment. Whilst the exhibition of such cardiac stimulants as ether, brandy, digitalis, and ammonia may be strongly called for when the circulation has been depressed from hæmorrhage, shock, or prolonged exposure, they are, generally speaking, useless in other circumstances: as, for example, when the patient has become asphyxiated during the stage of excitement and struggling, or when the circulation has suffered from the toxic effects of chloroform.

The *predisposing causes* of circulatory failure during anæsthesia are:—(1) Extreme exhaustion or shock prior to the administration; (2) cardiac and respiratory affections, attended by impaired cardiac action; and possibly (3) apprehension and fear. Instances of (1) and (2) may be found in such cases as amputation at the hip joint in a hectic and feeble subject, primary amputations for railway injuries, and operations upon patients with advanced heart affections, or with pleuro-pneumonia attended by empyema and pyrexia. Whether apprehension and fear ever really lead to syncope during anæsthesia is very uncertain. The so-called "deaths from fright" at the beginning of inhalation have hitherto been met with under chloroform, not under other anæsthetics.

Of the *exciting causes* of circulatory failure may be mentioned:—(1) The surgical procedure itself; (2) any pronounced asphyxial conditions; (3) the administration of toxic doses of the anæsthetic; and (4) the act of vomiting.

(1) The *surgical procedure* must be regarded as one of the most frequent causes of circulatory depression during anæsthesia. When hæmorrhage is profuse or protracted, when the patient has been lying exposed for a long while upon the operating-table, or when operations are being performed in the neighbourhood of the vagus or solar plexus, all grades of surgical shock may be recognised, however carefully and skilfully the anæsthetic may be given. Symptoms attributable to these causes usually come on more or less gradually, though they may (as in the case of vagal irritation) be so sudden as to be indistinguishable from those attending the gravest forms of syncope due to an excessive dose of chloroform. Generally speaking, the pulse becomes weaker and quicker, the face pale, the eyelids more and more separated, the extremities cold, and the pupils dilated. As a rule, respiration is but slightly affected. In minor cases of surgical shock the anæsthetic should be sparingly given, the head kept low, the body warm, free respiration encouraged, and an enema of hot brandy and water administered. Should the patient be able to swallow, the mouth may be kept moist and the lips rubbed with the corner of a towel soaked in brandy and water. In cases presenting more or less sudden symptoms under chloroform, it is best immediately to lower the head, raise the legs, and, if necessary, perform artificial respiration. Cloths wrung out in very hot water applied to the cardiac area will sometimes act beneficially in promoting circulation. Strychnine and digitalis may be given with advantage in cases which are not attended by too urgent symptoms (♏ij of the liq. strychninæ hydrochlor. and ♏v of the tinct. digitalis, hypodermically). The hypodermic injection of ether is not likely to be of any service if that anæsthetic is already present in the circulation; but in surgical shock which has arisen under chloroform, ether will produce improvement in the pulse. The intravenous injection of saline solution (ʒj of common salt to Oj of water) has also proved successful in some cases.

(2) The term *asphyxial syncope* may be applied to circulatory failure following arrested breathing. In these cases the cyanosis produced by the impaired or suspended respiration gives place, with various degrees of rapidity, to pallor and cardiac failure. Errors have frequently been committed in regarding the circulatory depression as the primary factor, the observer having overlooked the initial respiratory symptoms. Other things being equal, feeble patients are more liable than others to succumb to asphyxial syncope; but the most healthy and vigorous subjects are by no means free from this liability, more particularly when chloroform is employed. This is owing to their being prone to muscular rigidity and suspended breathing during the so-called "struggling" stage of the administration. The circulation at the time when the muscular spasm occurs contains a considerable quantity of chloroform; absorption from the pulmonary passages continues to take place, although respiration is in abeyance; the right heart becomes more and more engorged from the asphyxial condition; all the cardiac cavities dilate; the blood

pressure rapidly falls; and the heart ceases to beat. It is an interesting and significant fact that vigorous patients do not die under ether during the struggling stage, although respiration is just as, or even more, liable to become arrested, the stimulant effect of this anæsthetic apparently sufficing to keep the circulation going till respiration becomes re-established. Extremely feeble and exhausted patients may, however, fall victims to asphyxial syncope even under ether. This agent protects, as far as any anæsthetic can protect, against cardiac failure; but the heart may be so feeble that a comparatively slight impediment to free respiration may arrest it. In cases of strangulated hernia, for example, the temporarily arrested breathing incidental to vomiting may be sufficient to lead to syncope; and a similar sequence of events sometimes occurs in cases of empyema, the determining cause of the symptoms being respiratory embarrassment, arising either before the patient is fully anæsthetised, or subsequently when the operation is proceeding. The all-important point in connection with cases of this group is the uselessness of devoting time and attention to the re-establishment of the circulation without first restoring respiration.

(3) Circulatory failure from an *over-dose of the anæsthetic* takes place after complete anæsthesia has become established. When nitrous oxide or ether is given in toxic quantities to moderately healthy patients, respiratory paralysis ensues, cyanosis becomes marked, and the pulse, after a considerable interval, ceases to beat. The administration of an over-dose of chloroform, however, has a somewhat different effect, for the reduction of blood pressure and cardiac action becomes so conspicuous a feature of the case that, when respiration ceases, an ashy pallor rather than cyanosis is present, and there is little or no interval between the cessation of breathing and the disappearance of the pulse. Moreover, in some cases the circulatory phenomena of chloroform poisoning may overshadow the respiratory, pallor and heart failure being evident whilst respiration is still proceeding. But whilst we have in nitrous oxide and ether agents which, when given in toxic doses, do not depress the circulation of healthy patients to the extent which is observed with chloroform, it must be borne in mind that in exhausted subjects the use of nitrous oxide or ether in toxic quantities may be followed by rapid circulatory failure, similar in its main features to that met with when an over-dose of chloroform is given to a patient in good health. The treatment of all cases in this class is the same: namely, to proceed at once to artificial respiration, with the object of restoring the pulmonary circulation and of ridding the blood as quickly as possible of the poison which it has absorbed. Partial or complete inversion (Nélaton) may advantageously be combined with artificial respiration in some cases. Direct compression of the heart from below the thoracic margin is said to have been successful in a few instances; but any procedure of this kind should only be undertaken as an adjunct to artificial respiration.

(4) Circulatory depression connected with *vomiting* is more

common under chloroform than under other anæsthetics. Directly the stomach has ejected its contents, the colour of the face returns, and no further trouble ensues. The cases of fatal vomiting syncope have in all probability arisen from the temporary asphyxia, incidental to vomiting, acting upon a feeble heart.

5. The after-condition of the patient.

It is very exceptional for the administration of nitrous oxide to be followed by any unpleasant after-effects. Headache, nausea, and vomiting may, however, occur, and are more prone to do so if the gas has been given very soon after a full meal, or if a double inhalation has been found necessary.

After the use of chloroform or ether for a surgical operation, the patient should, whenever practicable, be turned upon his side, and then left perfectly undisturbed. In this position stertor will subside, mucus and saliva will drain away, and irritative cough will be prevented. The anæsthetist should remain by his patient till the conjunctival reflex has become fully established, and till respiration, which is liable to be interfered temporarily with during vomiting, has become regular and quiet. The room should be kept dark, noise prevented, visitors excluded, and sleep encouraged. Ether is more frequently followed by nausea or vomiting than chloroform, although the worst forms of persistent vomiting seem more common after chloroform. Elderly persons suffer but little; whilst florid young men and young women are frequently greatly distressed. Alcoholic patients quickly recover consciousness, and may display surprisingly few symptoms, even after protracted anæsthesia. The mucus ejected after ether is frequently bile-stained; but it may be brownish (simulating beef-tea) from a minor degree of congestive hæmatemesis.

The patient's feelings are usually the best guide as to the interval which should elapse before nourishment is taken. As a rule, nothing should be allowed for about four hours, when some clear soup may be given. After short administrations, some tea or coffee with dry toast is usually permissible at the end of three hours.

As to the treatment of distressing nausea and vomiting after ether or chloroform, it is best, in the first instance, to give draughts of very hot water, two or three ounces at a time, at intervals of a quarter or half an hour. Should this fail, hot strong coffee, without milk or sugar, or some hot water with gr. 10 of bicarbonate of soda dissolved in it, may be tried. Small doses of oxalate of cerium have proved useful in obstinate cases. The disagreeable taste left by ether is best relieved by allowing the patient to suck a very thin slice of lemon from time to time.

Tracheitis, bronchitis, pulmonary œdema, hemiplegia, albuminuria, nephritis, uræmia, glycosuria, various forms of insanity, fatty degeneration of the heart and other viscera, and jaundice, have one and all followed the use of anæsthetics. These sequelæ, however, are very rare.

II. LOCAL ANÆSTHESIA.

For the great majority of surgical operations, general anæsthesia is undoubtedly preferable to all other means of preventing pain. But there are, nevertheless, numerous cases in which local anæsthesia offers such advantages that general anæsthetics are now rarely needed. Then there are cases in which the advantages of the one system are so equally balanced by those of the other, that one surgeon will prefer a general, another a local anæsthetic. And lastly, in exceptional circumstances, for example, when a patient displays an insuperable objection to losing consciousness, when a brief operation has to be performed upon a patient whose condition is such that the risks of a general anæsthetic are very considerable, when ether or chloroform cannot be obtained, or when no assistance is at hand, local anæsthesia may prove of great service.

Of the means which have been adopted for the production of local insensibility to pain, two only are sufficiently reliable to merit consideration. These are (1) the employment of cocaine, and (2) the application of some frigorific process by which the part involved becomes numb and more or less completely anæsthetised.

1. Cocaine.

(*a*) **Properties and local action.**—Aqueous solutions of the hydrochlorate, of various strengths, are generally employed. They should either be freshly prepared, or should contain boracic acid, saccharine, corrosive sublimate, or salicylic acid in small proportions, to prevent the growth of fungus to which unprotected solutions are otherwise liable. Experience has shown that whenever the drug can be so applied that it comes into contact with sensory nerve-endings, these become temporarily paralysed, and an analgesic area results. Solutions applied to the conjunctiva or to the mucous membrane of the nose, throat, or larynx, quickly render the part insusceptible to pain, and a similar though not so satisfactory a form of analgesia follows the application to the mucous membrane of the genito-urinary passages and rectum. The skin can only be thoroughly anæsthetised when the solution is subcutaneously injected. Solutions of cocaine produce vaso-motor constriction over the area to which they are applied, a point of some importance in those surgical operations in which an anæmic state of the part is desired.

(*b*) **Indications, contra-indications, and modes of application.**

Ophthalmic surgery.—In the large majority of cases cocaine answers remarkably well. It cannot be relied upon in operations upon young children and very nervous subjects; when any inflammatory condition of the eye is present; or when the operation to be performed involves sensitive parts to which the drug cannot obtain access. A 5 per cent. solution is usually employed for instillation, though weaker or stronger solutions may, in certain cases, be advisable. ♏ j of a 5 per cent. solution should be dropped into the eye every minute for ten or fifteen minutes, till the fixation forceps can be borne without pain. Wafers containing gr. $\frac{1}{50}$ of the hydrochlorate of cocaine are useful (Carter and Frost). For cataract operations both eyes should be cocainised, even though one eye only is to be operated upon. For squint operations it is usually necessary, after the preliminary instillation, to inject a minim or two of a 5 per cent. solution beneath the conjunctiva in the neighbourhood of the muscle requiring division. The eye should be kept closed after instillation, to prevent corneal desiccation.

Intra-nasal, pharyngeal, and laryngeal surgery.—Cocaine is equally valuable in most minor operations within the nose, throat, and larynx. It is not to be relied upon when the parts are inflamed; it is not a suitable anæsthetic for the removal of post-nasal adenoid growths; and for the removal of tonsils many surgeons prefer a general anæsthetic. Analgesia may be produced (1) by directly applying a solution of the drug by a camel-hair brush or with pieces of absorbent cotton-wool twisted round the end of a probe, or (2) by means of a spray, the atomised solution being projected into the nose or throat. In the former method, which is usually adopted for intra-nasal operations, cauterising turbinated bones, removing "spurs," etc., a 10 per cent. solution is commonly employed, though it may be necessary to use a 20 per cent. solution, or even to add a few crystals of the salt to the wet wool or brush. In the latter method, which is especially serviceable for operations within the pharynx and larynx, a 3 per cent. or 5 per cent. solution will suffice for children; whilst for adults a 10 per cent. solution should be used (Hovell). For examining a sensitive naso-pharynx with the finger, a 2 per cent. to a 5 per cent. solution sprayed through the nose, so as to fall on the posterior surface of the soft palate, answers well; or the naso-pharynx and soft palate may be painted by a brush bent at right angles. Tonsils may be removed either after they have been painted or sprayed by the solution. When the part to be operated upon is congested, some difficulty may be experienced in obtaining anæsthesia, and stronger solutions than usual may be required.

Urinary and bladder surgery.—In certain operations and manipulations within the urethra and bladder, cocaine is sometimes employed; but as it is difficult or impossible to inject a solution beyond the membranous portion of the urethra without employing a catheter, a general anæsthetic should be used if absolute freedom from pain is desired. For catheterisation and similar procedures, from half to two drachms of a 5 per cent. solution should be injected into the urethra. A similar solution is employed for injecting into the bladder when it is desired to render the mucous membrane of that organ insensitive.

Dental surgery.—Cocaine is of great value in scaling, separating, examining, or treating sensitive pulps, etc., solutions of from 5 per cent. to 20 per cent. being employed. The drug is also very useful in relieving the pain of dental periostitis, a small piece of blotting-paper soaked in a 10 per cent. solution being applied to the gums adjacent to the offending root. Cocaine is far inferior to nitrous oxide as an anæsthetic for tooth extraction. Should local be preferred to general anæsthesia in any particular case, two or three injections, representing in all not more than gr. ⅓ of cocaine, should be made adjacent to the roots of the tooth (Hern).

Other surgical operations.—The pain occasioned by the repeated introduction of the hypodermic syringe, the uncertainty of the process in many cases, the possibility of struggling, emotional disturbances, or toxic symptoms arising, and the repugnance which most patients feel to retaining consciousness during surgical operations, have combined to retard the advance of cocaine into the domain of general surgery. But there may be exceptional circumstances in which cocaine analgesia is called for. A solution of 2 per cent. (Reclus) will be found to suffice for most cases. The part to be operated upon, as well as the hypodermic syringe, should be scrupulously clean, and rendered aseptic by the customary methods. The patient should be recumbent, and his face covered with a light handkerchief. When evidences of feeble circulation or great nervousness are present, a small quantity of brandy or sal volatile may be given. Whenever practicable, the part to be operated upon should be rendered bloodless, as suggested by Corning, by bandaging and the application of an elastic cord or tape. This has the effect of increasing the local and lessening the general action of the cocaine, and when the cord or tape is removed any remaining cocaine is carried off by the hæmorrhage which occurs. The punctures of the hypodermic needle may be rendered painless, if desired, by the use of ice, ether spray, or chloride of ethyl. Care should be taken to avoid blood-vessels when injecting. Reclus places the maximum of cocaine for each operation at from gr. ⅓ to gr. ij. He uses a (Pravaz's) hypodermic syringe with a long fine needle, and advises that the solution should be injected whilst the syringe is being withdrawn, so as to leave long anæsthetic tracks subjacent to the proposed skin incisions. When the skin has been anæsthetised, the deeper tissues must be injected or subsequently cocainised during the operation.

Dr. Coppinger, of the Haslar Hospital, who has used cocaine injections extensively, considers the following conditions to be essential:—(1) The punctures should be made through healthy non-inflamed skin or mucous membrane; (2) The solution should be injected into the cellular tissue, the point of the needle being felt to move freely before the piston is pressed; (3) There should be an interval of ten minutes before the operation is commenced. Dr. Krozius, in an interesting paper, urges that in some cases it is possible, by injecting the cocaine solution into the neighbourhood of a nerve trunk along a line at right angles to the axis of the limb, to produce "peripheral analgesia," not only of the skin, but of other structures over the area supplied by the nerve. He agrees with Reclus that a 2 per cent. solution is quite sufficient.

(c) **Systemic effects of cocaine.**—The local application of cocaine is not infrequently attended or followed by symptoms pointing to its absorption into the general circulation. Such symptoms are most likely to arise when the drug has been injected directly into a blood-vessel or into a highly vascular part. There is good reason to believe that many of the alarming symptoms which have attended the use of cocaine have either been due to unnecessarily large doses, to impurities in the solution employed, to want of proper antiseptic precautions in introducing the cocaine, or to psychical causes unconnected with the action of the anæsthetic. Reclus, who collected and analysed seventeen fatal cases of cocaine poisoning, came to the conclusion that in all cases an excessive dose had been used. But it must not be forgotten that patients display very great differences in their behaviour with cocaine, toxic symptoms having undoubtedly arisen on many occasions from simply painting or spraying a mucous surface with comparatively dilute solutions. Amongst the phenomena which have been recorded are:—Headache; vertigo; pallor; a cold, moist skin; a feeble, slow, or rapid pulse, becoming imperceptible in grave cases; incoherence of speech; nausea; vomiting; unconsciousness; trismus and other muscular spasms; epileptiform attacks; dilated or unequal pupils; and disturbances of respirations culminating in dyspnœa and asphyxia.

In the *treatment of cocaine poisoning*, the first indication, as a rule, is to restore the circulation. The patient, if not already lying down, should at once be placed horizontally, and some rapidly acting stimulant, such as brandy, whisky, or sal volatile, administered. A hypodermic injection of ether is an excellent remedial measure. The patient should be kept warm, the respiration watched, and any tendency to failure of breathing met by artificial respiration. Nitrite of amyl, nitro-glycerine, and chloral have each been used with success.

2. FRIGORIFIC PROCESSES.

When heat is rapidly abstracted from the skin, mucous membranes, or other parts, by one of the following processes, partial or complete congelation of the tissues takes place, and, owing to the sensory nerve-endings becoming paralysed, analgesia occurs. Frigorific analgesia is of most value when simple cutaneous incisions or punctures have to be made. There are, however, several objections to the employment of cold as an analgesic agent. The natural appearances of the parts are altered; operative procedures upon the frozen tissues are difficult of execution; and the process of thawing is often attended by considerable pain.

The ether spray.—A special apparatus, consisting of indiarubber hand bellows, tubing, and an atomiser, is needed. Sir B. W. Richardson, the originator of the method, prefers equal parts of anhydrous ether and of rhigolene (crude hydride of amyl) to ether itself. It is said that when ether containing carbolic acid (five grains to five fluid ounces of ether) is used in the form of spray, analgesia occurs before the skin becomes hard, so that the manipulations of the surgeon are less impeded than in the case of the ordinary ether spray.

Ethylchloride.—This appears to be the most efficient of all the frigorific liquids which have been used for producing local anæsthesia, owing to its very low boiling-point (50° Fahr.). It is supplied in convenient cylindrical glass tubes with fine metal nozzles, and the heat of the hand suffices to project a jet of the chloride of ethyl on to the surface to be anæsthetised.

XV. SURGICAL DISEASES DUE TO MICROBIC INFECTION AND PARASITES.

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I. POST-MORTEM WOUNDS.

Ætiology.—The frequent occurrence of local or general septic infection amongst those employed in making autopsies has led to the use of the above name for a certain class of wounds followed by severe general and local symptoms. Similar infections, however, may be incurred by the surgeon, the symptoms of which differ in no way from those of the classical post-mortem wound. The special characters of these cases do not depend on any one specific organism, but probably on several; and this view is supported by the general resemblance of the local process to that observed in cellulitis from other causes. Individuals differ greatly in susceptibility to these infections, and beyond this, a certain immunity seems to be acquired by those habitually employed in the dead-house; such men are rarely attacked, the commoner victims being the new hand or the unaccustomed student. Common experience, however, while negativing the presence of a single specific virus, offers striking evidence of the special dangers dependent on the handling of the bodies of subjects who have died of pyæmia, septicæmia, or peritonitis; from infection from such sources the most serious forms of post-mortem wound have been seen to result.

Fresh corpses, again, are by far the most dangerous, the post-mortem room furnishing the cases in which both local and general symptoms reach their extreme degree; while infections from the dissecting-room are usually followed by moderate local reaction and slight general symptoms. A wound is the commonest mode of infection, and this is often of a trifling nature. Severe wounds usually bleed, and thus the poison may be removed; and again, the discomfort accruing from them leads to more thorough disinfection, and probably cessation of work. Pre-existent abrasions, or wounds of a slight nature, unaccompanied by pain or hæmorrhage, are a much more fertile source of trouble. The most striking characteristics of the acute cases are the early advent of general symptoms of septic poisoning, the rapidity with which the local process spreads, the tendency to early suppuration, often of an incomplete nature, and the sloughing of tissue.

Symptoms.—These may be either acute or chronic; while the acute cases must be again subdivided into those in which general or local manifestations take the first place.

(1) **Acute septic intoxication.**—This form—happily, the rarest—resembles closely the similar condition occasionally noted after a wound of the abdomen involving the peritoneal cavity.

The general symptoms consist in pain in the part, and the rapid supervention of collapse, as evidenced by pallor, failure of the general circulation, with rapid small pulse, restlessness, twitchings, delirium, rise of the general temperature, followed by unconsciousness, and death probably on the second day. The local signs are often slight: probably by reason of the abrogation of the general nutritive functions. The wounded spot may become slightly œdematous, and a small vesicle not infrequently marks the seat of primary inoculation; but otherwise, no marked local changes occur.

(2) **Acute cellulitis, accompanied by symptoms of general septic absorption.**—The local symptoms here take the foremost place. The upper extremity is almost without exception the part affected, punctures of the fingers or scratches on the backs of the hands from the ends of the ribs being common starting-points.

The first symptoms are malaise and local pain of a severe character at the point of inoculation. These are followed by rapid swelling and the formation of pus often during the first twelve hours; general rise of temperature to 103° or 105° F., often heralded by rigor and sickness, and acceleration of the pulse-rate.

The further progress of the case varies considerably, either as result of the nature of the poison inoculated, or of the peculiar susceptibility of the individual affected. In some cases gangrenous boils or pustules only develop, these being, however, often very chronic in their course. In other slighter cases a red line marks the spread of infection by the lymphatic vessels, and the glands in the axilla become swollen and tender. The process may then rapidly subside, with or without the development of a glandular abscess. The lymphatic glands here act as an effectual safeguard against general infection, probably seconded by a normal resistance on the part of the individual constitution. In the course of a week such an infection may leave little behind it beyond a certain amount of weakness, consequent on the general febrile disturbance. Such is the variety usually observed in the dissecting-room.

In the more serious cases, which are far from uncommon, both the local process and general symptoms take on a graver aspect.

A severe cellulitis rapidly spreads from the seat of infection, involving the fore-arm, the arm, and in the most grave cases the trunk also.

The local signs, however, again differ; the cellulitis may be of the hard brawny type, with much pain, tension, and redness; or the limb may be simply swollen, pale, and œdematous. This is probably in part dependent on the nature of the poison and the condition of the individual, and in part on the depth and situation of the wound. With regard to the latter points, it is, no doubt, of importance as to whether the wound has passed the limits of the subcutaneous areolar tissue, and whether the wound be on the dorsal or palmar aspect of the hand. The process, as it spreads, passes most rapidly along the perivascular planes of areolar tissue and the synovial sheaths of the tendons. Deep wounds affect the subfascial structures of the limb; and the comparative depth of the tendon sheaths, and more composite arrangement of the structures of the front of the fore-arm, render wounds of the flexor aspect more dangerous and more difficult to heal.

The advent and nature of suppuration vary. In some of the worst cases incisions allow the escape of serum, or, at the most, of very ill-formed pus, and the wounds have a gelatinous appearance. In others acute suppuration is noted on the first or second day, and is abundant. On the whole, the latter is by far the most favourable condition, as it much more commonly tends to limit itself by the eventual formation of an efficient barrier of inflammatory exudation. The local signs are accompanied by general symptoms of varying intensity. In all cases the temperature rises, reaching 102° or 103° F. The patient is often constipated; the tongue is foul, rapidly becoming dry and brown; and in the fatal cases sordes may appear upon the lips. There is complete loss of appetite, and sickness may be troublesome. The patient complains of headache, general malaise, and sleeplessness, often much aggravated by the pain in the limb, and delirium is common. The urine is scanty, high-coloured, and often contains albumen. The further course may be towards resolution, or the local and general symptoms may progress, and the patient will die of septicæmia, exhaustion, or of pyæmia. With regard to the latter termination, it must be borne in mind that definite signs of pyæmia may appear late in the case, often after the opening of abscesses in connection with the local process. Thus, in a case recorded by Billroth signs only appeared two months after an infection from a case of uterine carcinoma; and in the case of one of the post-mortem clerks at St. Thomas's Hospital Medical School, acute pyæmia developed after the opening of an abscess subsequent to a lymphangitis fourteen days after the primary inoculation.

The *prognosis* in these cases mainly depends on the constitution of the individual, and on the condition of the patient on the third or fourth day. If general symptoms continue grave after this date, a serious issue is only too often to be apprehended. A severe attack usually leaves a lasting mark on the constitution of the patient.

Treatment.—Prophylaxis is naturally of the first moment; and it cannot be too strongly urged that in no case should a post-mortem examination be performed by anyone with a wound on his hand, particularly in that most dangerous region, the margin of the nail bed. Secondly, it must be borne in mind that a wound even is unnecessary for infection, but that the sebaceous or sweat follicles may serve as points for inoculation ; and this emphasises the need for properly smearing the hands with a suitable ointment before performing an autopsy of any kind.

If a wound, of however slight a nature, be suffered during the performance of such work, the part should be bound round above, to encourage bleeding, thoroughly washed in an antiseptic solution—the best being 1 in 20 carbolic acid—and the wound sucked, further performance of the work being discontinued. It is only to be wished that the latter part of the above advice could be given to the surgeon also; but naturally, an operation must be finished. One can only say in such a case that the utmost care must be observed.

General treatment.—The subjects of these wounds need support by frequently-administered fluid nourishment and the free exhibition of stimulants, of which, perhaps, brandy is the best. In cases of threatening septic intoxication, stimulants are the only hope—and that a slight one. Quinine may be given in large doses (grs. v or more) in the early stages, when the temperature is high. Later, iron, in the form of Tr. ferri perch., is useful. The bowels must be kept acting regularly; and if diarrhœa is present, it must be checked—most usually by the use of opium. An efficient method of administration in such cases is often a starch enema, with Tr. opii ♏xx ; or better, a $\frac{1}{3}$ or $\frac{1}{2}$ grain morphia suppository. Subcutaneous injections of morphia are usually needed to relieve pain and procure sleep.

Local treatment.—The limb should at once be enveloped in a warm dressing with lead lotion, and if signs of serious cellulitis appear, the use of a hot mercurial bath (1 to 5,000) should be at once commenced. This is better employed for periods of one or two hours at intervals, alternating with a warm dressing,than continuously. If there be any objection to the use of mercury, boracic acid may be substituted. Great care must be taken to sling the limb with strips of bandage, so as to avoid any constriction of the upper part by allowing it to rest against the edge of the bath. Early incisions are needed to lower tension, relieve pain, and to allow the exit of pus. These should be sufficient in size and number to effect their object, but especial care should be taken not to carry them beyond the affected area, and not to incise the deep fascia when the subcutaneous tissue only is affected, or the surgeon may become responsible for considerable, even fatal, spread of the process. Ample nourishment and change of air and surroundings are most necessary during convalescence from an illness of this nature.

II. ANATOMICAL OR BUTCHERS' TUBERCLE.

Manifestations.—The hands of persons habitually employed in the manipulation of corpses of either men or animals not infrequently become the seat of a chronic thickening known under this name.

Its resemblance to lupus early gained for it the name of lupus anatomicus (Roser); and later observations have shown the condition in some cases to be an actual local inoculation of the tubercle bacillus. The absence, however, of instances of generalisation goes far to negative the view that all cases are of a specific nature, the majority, no doubt, being a result only of irritation from frequent contact with putrid animal matter, in some degree resembling the chronic form of eczema often seen on the backs of the hands of plasterers.

The commonest seat of the disease is the thin skin of the backs of the hands, especially over the metacarpal phalanges; similar thickenings are often also met with at the margins of the nails; and it may be noted that a corresponding thickening is often observed in old cicatrices of trivial wounds incurred in post-mortem work.

The cutaneous papillæ become enlarged in circumscribed limits, forming small areas, the surface of which is uneven, covered with a thick slightly opaque layer of epidermis, of a bluish-red colour.

These areas are often tender on pressure, readily become excoriated when serum exudes and forms a scab, and they may sometimes bleed from slight injury. They are usually on the back of the hand; much more rarely the fore-arm may be affected.

When the borders of the nails are affected, an isolated nodule usually exists, bluish-red in colour, tender on pressure. It evidently affects the skin only, and resembles somewhat a small epithelioma.

The course of all such cases is very chronic and they tend to get well if the exciting work is discontinued.

Treatment.—In some instances protection of the hand is all that is needed to effect a cure. In more obstinate cases isolated nodules are best treated by the application of acid nitrate of mercury or strong nitric acid. When the backs of the hands are extensively affected, scraping with the sharp spoon is the most effective form of treatment.

III. INSECT STINGS.

Varieties.—Many familiar insects attack man, but except in special circumstances the symptoms are of slight surgical importance. As a rule, the bites of *flies* cause merely slight swelling and redness: even the tsetse fly of Africa, which renders the keeping of animals impossible, in man gives rise only to a wheal, which has been likened to a mosquito bite. In situations such as the eyelid, where loose subcutaneous tissue exists, œdema may be considerable, and this may be developed even in the limbs if the bites are multiple. It must be borne in mind, however, that flies especially may be the carriers of specific virus, and that when they have come direct from putrid animal matter, cellulitis or severe local inflammation may result from their bites. Flies are probably sometimes the bearers of the virus of anthrax, and the important *rôle* played by the mosquito in the development of filariæ will be again referred to.

Some varieties of *spider* are decidedly venomous: thus Aranea nedecius in Italy and Aranea mactans in South America give rise in persons bitten by them to severe headache, pain in the back, a feeling of weakness, cramps, dyspnœa, and a sense of constriction around the chest. These symptoms last twelve to fourteen hours, and have been known to be followed by death.

The stings of *bees*, *wasps*, and *hornets* give rise to more severe symptoms than the bites of flies, especially when the insect is in an excited state. Burning pain is the immediate result, and this may be followed by more or less œdematous swelling. The pain is the more severe when the sting and poison sac of the insect remain in the wound; but pain and swelling usually subside rapidly when these are removed. Multiple stings, if numerous, may be more serious, producing more or less collapse; or even death from cardiac paralysis. The locality of the sting is also of importance: thus stings of the eyelids cause much swelling, and those of the fauces, mouth, and tongue, sometimes resulting from the introduction of the insect with fruit, may give rise to troublesome œdema of the tongue or larynx.

Treatment.—The treatment of fly bites seldom needs to consist of more than the application of lead lotion; a weak carbolic lotion gives rapid relief in cases of mosquito bite. For stings, the application of an alkaline lotion, preferably weak liquor ammoniæ or sal volatile is the best treatment. Swelling may be treated with lead lotion. Symptoms of collapse such as follow some spider bites and stings, indicate the exhibition of stimulants. Alcoholic patients frequently suffer more severely from insect stings than healthy persons.

IV. SNAKE BITES.

Poisonous snakes.—The occurrence of venomous snakes in the United Kingdom is practically limited to the common adder (Pelias berus); in Australia the brown snake (Diemenia superciliosa), the tiger snake (Hoplocephalus curtus), the black snake (Pseudechis porphyriacus), and the death adder (Acantophis antarctica); in India the cobra (Naja tripudians) and various species of vipers; and in America the rattlesnakes (Crotalus adamanteus, durissus, horridus, and miliarius), and the mocassin (Ancistrodon piscivorus), are more or less common.

Venomous snakes are provided with a pair of glands situated at either side of the lower jaw, much resembling the parotid in position. Ducts extend from these glands to specialised teeth, either grooved or canalised to their apices, by means of which the poison is introduced into the wound. The teeth or fangs—usually two in number—are long and curved backwards; but in snakes a special mobility of the upper jaw allows the fangs to be pressed vertically in the wound, while the poison is expressed by the special crotaphite muscle, which partly crosses the gland, aided by the musculature of the jaws. The wounds produced are double, pass vertically inwards; and in the case of large snakes the muscles are often penetrated, the depth of the wound depending not only on the muscles of the jaw, but also on the weight of the reptile and consequent force of impact with the victim.

The *poison* is a transparent, yellowish, somewhat sticky fluid, of faintly acid reaction when fresh, and containing water, albumen, fat, salts, and a peculiar poison, possibly of the nature of the albumoses. It acts more powerfully on small than on large animals, and children are more dangerously affected than adults. Bites implicating large veins are followed by the most rapid and severe symptoms.

The morbid lesions found in fatal cases are—congestion of the lungs and brain probably due to the difficulty in respiration, and in some snake bites local blood extravasations and congestion of the internal organs evidenced during life by vomiting of blood and passage of blood with the urine or fæces are special features. The blood is frequently incoagulable.

Symptoms.—Either at once, or after a few hours, the patient becomes anxious, faint, breathes with difficulty, experiences nausea, or vomits. In the slighter cases these symptoms may pass off. In severe cases, a cold clammy sweat breaks out, the pupils dilate, motor functions are more or less completely abrogated, delirium supervenes, followed by coma, and the patient dies of syncope, from cessation of the respiratory movements, or from collapse. The local effects of the poison are mainly the production of intense hyperæmia of the subcutaneous connective tissue, accompanied by pinkish œdema often following the course of the efferent blood-vessels, and more or less extensive blood extravasation (Wall).

Locally, the two openings with ecchymosed margins are found; there is severe pain and swelling, at first limited, but rapidly extending. When the bite is severe, and a large dose of the poison has been introduced, the part falls in temperature, and spots of ecchymosis, or more or less extensive gangrene, appear. In 94 per cent. of all cases the bites are found on the extremities; in some snakes, notably the cobra, the fang punctures are so minute as to need careful searching for.

Certain variations in the effects produced by the bites of different snakes have been noted; thus, in Australia those of the brown and tiger snakes are especially followed by disturbance of the motor functions, and that of the tiger snake by vomiting of blood. Black snake bites are characterised by anæmia, due to the collection of blood in the vessels of the abdominal viscera, and those of the death adder by local blood extravasation. In India, cobra bites are followed especially by respiratory symptoms, glosso-labio-laryngeal paralysis, salivation, and other indications of hyper-secretion; those of Bungarus fasciatus, another variety of colubrine snake, by slow development of symptoms, an interval of two to five days elapsing before their appearance, and those of the vipers by great pain, extensive blood extravasations, and sanious excretions.

Treatment.—The *local treatment* consists first in the application of an elastic ligature above the wounded point, to prevent as far as possible diffusion of the poison into the general circulation. In all severe cases it should be borne in mind that suction is of no avail for the extraction of the poison, but the wounds should be at once laid open and the whole of the hyperæmic and œdematous area excised. The raw surface should then be thoroughly treated with a solution of permanganate of potassium, which, when directly mingled with snake poison, destroys it by oxidation (Wall). Calmette's experiments show that snake poison is also rapidly rendered inert by the local application of a solution of hypochloride of lime. The *general treatment* consists in combating the collapse with stimulants. Specific forms of treatment have been sought in the subcutaneous injection of ammonia and permanganate of potassium, but have proved of no avail. In Australia the administration, hypodermically, of strychnia in large doses appears to have been followed by successful results. It is given on the theory that it acts in direct antagonism to the snake poison; and hence it is claimed that large doses are necessary, and that poisonous doses are innocuous in persons suffering from the effects of snake venom. In patients over fifteen, 10 to 25 minims of the liquor strychninæ, gr. $\frac{1}{75}$ to $\frac{1}{6}$ of the British Pharmacopœia, are injected, and, as a rule, not more than half a grain of strychnia is needed. The rule laid down is that sufficient must be given to ensure the symptoms of snake poison being overshadowed by those of the drug; and repetition of the injections is indicated by any relapse in the symptoms (Mueller). The treatment is now under trial in India under the auspices of the Government.

In support of this method of treatment, putting aside the question of specific antagonism, may be mentioned the well-known action of strychnine in improving the respiratory movements in cases of failing respiration during anæsthesia produced for surgical purposes.

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V. HYDROPHOBIA (RABIES).

Ætiology.—This is a disease the primary occurrence of which is almost entirely limited to the dog, but is communicable by inoculation to other animals and to man.

The disease, no doubt, depends on a specific virus, which, however, has not up to the present been isolated. The experiments of Pasteur show that the virus and its products tend to accumulate chiefly in the nervous system, whence it can be readily obtained, and other animals inoculated. Further, material obtained from this source by a treatment of desiccation can be so modified as to be capable of employment as a preventive agent. Prolonged desiccation renders the virus inert.

Although occurring in other animals than the dog, the majority of these suffer from the result of a dog's bite; and the tendency of

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the rabid dog to attack his own species accounts for the large number of cases observed in the canine race. Cats, wolves, foxes, and large animals, such as horses, cows, deer, etc., not uncommonly suffer. Fleming has shown that the normal proportion of dogs affected to other animals in Great Britain is from 90 to 96 per cent.

Mode of infection.—A breach of surface is necessary for the introduction of the virus; a bite is the ordinary source, and this need not exceed a superficial cutaneous abrasion in severity. The infecting fluid is, almost without exception, the secretion of the mouth, fauces and salivary glands, but the blood may be the carrier of infection; and a case has been placed on record in which a veterinary surgeon acquired the disease in making a post-mortem examination.

A large number of persons bitten by rabid dogs escape the disease; the proportion, however, has been very variously given as from 5 to 75 per cent. In this particular the locality of the bite, and its depth and severity, are of much importance. Dogs commonly snap at parts of the body covered by clothing, and it is possible that the teeth are thus more or less cleansed of the infecting medium. This may explain the immensely increased probability of infection when the face, hands, or uncovered parts are bitten, and the danger of bites from pet animals that are being fondled. Multiple bites are more dangerous than single ones, from the increased surface inoculated; but severe bleeding, especially from a single wound, may be favourable, as tending to wash out the virus introduced.

Statistics seem to prove conclusively that the bites of wolves and cats are more dangerous than those of dogs, and it seems probable that this depends not on the severity of the wounds alone, but also on a special virulency of the poison.

Period of incubation.—In human beings bitten by rabid dogs this is very variable; in a large majority of cases it has been shown to be roughly six weeks, but undoubted cases have been put on record of children bitten on the face in which it has not exceeded six days; and again, symptoms have undoubtedly developed as late as the end of two years (Horsley). This variability may depend in part on the amount of virus introduced, and in part on individual susceptibility. As a rule, however, symptoms very rarely develop after a period of four months from the date of inoculation.

In dogs bitten by rabid animals the incubation period is commonly from three to six weeks, less often from seven to ten; but it has been observed in some cases to extend over some days only, and in others months. During the incubation period the animals are not dangerous.

It has been discovered by Pasteur that the passage of the virus through successive series of rabbits much increases its intensity; and from this source he obtains his *virus fixé* for protective or preventive inoculations.

When the virus has been passed through twenty to twenty-five rabbits the incubation period is reduced to eight days; passage through a second series of twenty-five shortens the period one day more; and after the number reaches ninety, the incubation period is reduced to little less than seven days.

Rabbits inoculated subdurally with virus thus intensified exhibit symptoms with the utmost regularity on from the sixth to the eighth day, the condition induced being one of paralytic rabies, the preliminary stages of depression and excitement not being observed. Dogs inoculated in the same manner develop rabies on the eighth to the ninth day; but in these animals, sometimes the furious, sometimes the paralytic, form is observed. This appears to depend, at any rate in part, on the amount of virus introduced, the larger doses producing the paralytic form. Subdural inoculation of rabbits with virus from an ordinary rabid dog from the streets, on the other hand, does not produce symptoms before the fifteenth day.

Morbid anatomy.—The lesions observed, although constant, unless the individual has died as early as the second day from syncope, consist of little more than conditions of hyperæmia or congestion. In the central nervous system early congestion is followed by the migration of leucocytes into the perivascular lymphatic spaces and the interstitial neuroglia, and small parenchymatous extravasations of blood may also occur (Allbutt, Coats, Gowers, Greenfield). The changes in the central nervous system, however, are frequently concentrated in localised areas, so that the whole must be carefully examined (Horsley). Congestion is also found of the larynx, trachea, lungs, fauces, pharynx, œsophagus, and stomach, and often hæmorrhages into the mucous membrane of the latter organ. Congestion of the peripheral nerves, especially of the vagus, and of the sympathetic system, has been noted, and accumulation of leucocytes in the salivary glands, in the mucous glands of the larynx, and in the kidneys, has been described by Coats.

Symptoms. *Local.*—The local symptoms are usually insignificant. The wounds heal readily, as a rule, without local thickening or glandular enlargement; both the latter conditions have, however, been observed, but it is an open question as to whether the changes may not have been due to a contemporaneous infection of another nature, or to the local treatment adopted.

Prior to the development of symptoms, subjective sensations of heat, tingling, or pain have been noted; occasionally, severe lancinating pain in the course of the sensory nerves. In other cases the wound has been observed to break down, and produce a small amount of thin purulent fluid. Occasionally a crop of vesicles has been observed to develop around the site of the wound (Piorry).

Beyond these local vesicles, an eruption of vesicles in the oral cavity, on and beneath the tongue, on the cheeks, around Stenson's duct, and about the tonsils, have been described (Marichotti). These are said to occur only between the third and twelfth days after inoculation, so that, even if constant, their presence would often be overlooked, since they precede the development of general symptoms.

General.—These vary in degree, but, as a rule, may be divided into two stages—that of invasion and that of excitement. The typical paralytic stage occurring in dogs is replaced in man, as a rule, by exhaustion.

The stage of invasion is insidious, and may be absent or pass unnoticed. It is characterised by mental depression, irritability, with an ill-defined feeling of apprehension, and malaise. The patient looks pale and anxious and is restless. He may talk freely, the sentences being sometimes interrupted by sighing inspiration, but usually shows great unwillingness to speak about the possible cause of his illness. Sleep is broken often by dreams; the mouth is dry; the patient complains of thirst, but is disinclined to swallow. Loss of appetite, a feeling of nausea, with epigastric uneasiness, follow; the pulse is quickened, sometimes becoming very rapid; and the respirations are proportionately hurried and shallow. There is general hyperæsthesia and often increase in sexual desire.

The stage of excitement.—In two to three days these symptoms undergo further development. Mental agitation increases; ill-defined terror takes hold of the patient. He speaks excitedly in abrupt sentences and may suffer from hallucinations. The facial expression corresponds with the mental state, the brows are contracted and the face is pale, while the eyes are bright and wander constantly over the patient's surroundings. The mouth and fauces become congested and the patient ejects thick tenacious mucus with difficulty and much noise, and without any regard for those attending upon him. Solids can often still be swallowed; but while thirst is commonly severe, the dread of all liquids increases rapidly. When asked to drink, the patient takes the vessel with much precaution, often closing his eyes, and, feeling that he is making a crucial experiment, raises it with slow precision to his lips. The whole attention is devoted to the effort, which results usually in the violent ejection of the small quantity of fluid introduced. Intense agitation follows, the vessel being frequently thrown away, and a convulsive attack is often induced. The patient shudders, spasmodic movements of deglutition and respiration follow, the nares dilate, the commissures of the lips tighten, the scalenes, sterno-mastoids, respiratory and abdominal muscles contract successively, and then the entire muscular system, respiration being entirely suspended for some moments. These convulsive attacks may occur consecutively, like those of sobbing or weeping, continuing even for thirty to forty minutes; and during one of them the patient occasionally dies from syncope even as early as the second day. The general hyperæsthesia heightens, and increasingly severe convulsive attacks may be induced by the weight of the bed-clothes, draughts of air, bright light, and loud or unfamiliar sounds, the sound of running water being especially irritative. The mental condition is highly characteristic. The patient looks around with suspicion, often complains that his medical attendant does nothing for him, shows a great aversion to onlookers, and is surly and morose. This state may alternate with one of maniacal excitement, the patient throwing himself wildly about and often damaging his surroundings. At the same time, he retains in some measure his reason, may often be quieted by persuasion, and often gives those surrounding him advice to protect themselves against his possible violence. Sexual excitement, priapism, and seminal emissions are sometimes extremely troubling, and may give rise to difficulty in micturition, which is otherwise often frequent. The urine may contain albumen and sometimes blood. The temperature may rise considerably, but is oftener little elevated, or sometimes normal.

With the progress of the disease the strength of the patient rapidly decreases and emaciation may commence. The pulse becomes quick, irregular, and small in volume, and respiration quick and shallow, a deep inspiration often inducing a convulsive attack. Tenacious mucus accumulates in the mouth and fauces, articulation is thick and low, convulsive attacks increase in frequency, and death from asphyxia may occur during one of them. In other cases the progress is slower, the eyes sink, the brow sweats, the lips become blue, and the patient dies of slow asphyxia; or occasionally an almost complete remission of convulsive attacks may precede death from exhaustion, the mind in either case remaining clear to the end.

In some cases a more or less complete paraplegic condition has been noted before death. In rare instances the disease mainly assumes a paralytic form, and then strongly resembles that known as Landry's, or acute ascending paralysis, commencing in the lower limbs, gradually spreading to the trunk, upper limbs, and face, these phenomena being unattended by any of the classical symptoms of hydrophobia.

Diagnosis.—The early symptoms may be simulated by *hysteria.* Close observation of the patient's symptoms, which are often exaggerated or ridiculous, together with careful attention to the history and inquiries as to the condition of the source of the bite, will suffice to clear up any doubts. It should be borne in mind in these cases that many persons bitten by dogs not rabid often complain of subjective sensations in the wounds some time after the original injury. *Tetanus* occasionally follows the bite of a dog, and many cases have occurred in which this has been mistaken for hydrophobia. Attention to the following points will obviate any chance of confusion :—The length of the incubation period is usually very much shorter in tetanus; the mental state differs, the terrible anxiety being absent; the spasms are tonic rather than convulsive in character; the facial aspect (risus sardonicus) differs; and lastly, the marked rise of temperature seen in tetanus does not occur in hydrophobia (page 192). In the pure paralytic form the diagnosis from acute ascending paralysis can only be made by close attention to the history, and possibly confirmed later by inoculation experiments on animals.

Prognosis.—Prior to the introduction of Pasteur's treatment the disease has been considered a fatal one. Evidence seems clear, however, that patients subjected to the preventive inoculation treatment within six days of infection recover, with few, if any, exceptions. The especial danger of bites of the head—the mortality after which has been placed at eighty per cent.—and the peculiar virulence of the poison observed in wolves and cats, must be borne in mind even in cases subjected to the preventive treatment. The duration of the disease is usually from two to four days, but patients occasionally survive as long as seven.

Treatment. *Prophylaxis.*—Before proceeding to consider the question of treatment of cases, a word as to prophylaxis must be said. Proper precautionary measures have already succeeded in many countries (Sweden, Norway, Switzerland, Russia, Baden, Bavaria, Würtemberg) in suppressing the disease. The regulations necessary are simple, consisting in : (1) The destruction of all rabid or suspected dogs ; (2) the destruction of all wandering or homeless dogs ; (3) the wearing of muzzles by all dogs while rabies prevails, and for a period equal to the longest interval of latency after the malady has been suppressed ; (4) the forbiddal of the introduction of dogs from countries where rabies is prevalent, or the imposition of a sufficient period of quarantine (Fleming).

Local treatment.—Bleeding, if any, from the wound should be encouraged by placing a ligature around the limb above, and possibly the application of cupping-glasses. The wound should be thoroughly treated with strong carbolic acid, or in certain situations may be excised if the patient be seen early.

General treatment.—Failing the possibility of protective inoculation, the general treatment is palliative only. It consists in ensuring as complete quietness as possible, and in giving nourishment, resorting to rectal alimentation when swallowing becomes impracticable. Hydrate of chloral or morphia is useful ; and in the fully-developed disease the sufferings of the patient may be alleviated by the administration of chloroform, a small quantity often sufficing to maintain quietude.

Pasteur, convinced of the microbic nature of the virus of hydrophobia, conceived the idea that the development of the organism in the animal system might be inhibited by the employment of the waste products of the metabolic processes, dependent on its own growth and multiplication. He discovered that by subjecting portions of the nervous system of hydrophobic animals to a process of desiccation for fixed periods, the virus could be progressively weakened, and eventually rendered inert, without destroying these waste products. Dogs were first treated with portions of nervous tissue thus prepared, injections of gradually-increasing virulency being made ; and it was found that the animals were thus rendered immune to rabies.

This result obtained, it occurred to Pasteur that, considering the length of the average period of incubation of hydrophobia in man, the full development of the disease might be prevented by the inoculation of a similar material, provided the treatment was commenced sufficiently early. The first trial, made upon Meister on July 5th, 1886, confirmed this anticipation, and since then, in the years 1886-1891 inclusive, 9,461 persons bitten by ostensibly mad dogs have been treated at the Pasteur Institute in Paris on the same lines. Of this number, only ninety died; and thus a death rate of 0·95 per cent. is substituted for that of at least 14 per cent. occurring in persons bitten by rabid dogs not subjected to preventive treatment. During the years 1886 to 1893 inclusive, 14,430 persons underwent the treatment, of whom 72 died, a death-rate of 0·50 per cent. This is strong evidence in favour of the method, which, moreover, has been reported upon favourably by a Government Committee, after repetition of the experiments by Horsley.

Material for injection is prepared at the Pasteur Institute, as follows:—The spinal cord is obtained from rabbits rendered rabid by inoculation with virus, previously passed through a succession of animals of the same race to heighten its intensity. The nervous tissue is cut into small blocks, and desiccated for various periods by suspension over a small fragment of caustic potash in a sterilised flask at a temperature of 72° F. Portions of the required degree of modification are mashed up in a mortar, with slightly alkaline sterilised broth, to a state of fine emulsion, and this is employed in doses of from 1 to 3 cc., the larger quantity being used in the case of the more highly modified material.

The quantity of dry nervous tissue in each injection amounts to about 1 centigramme, and inequality in the strength of the material thus prepared is made up for by repetition of injections of the same reputed strength during the course of treatment.

Two formulæ are employed:—(A) "The simple," for ordinary bites of the limbs not numerous; (B) "The intensive," for wounds of the head or face, severe multiple wounds, and the bites of cats or wolves. Variations are made between the two formulæ to meet the severity of individual cases.

A. SIMPLE.

Day of Inoculation ...	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Number of days of desiccation of material...	14 13	12 11	10 9	8 7	6 6	5	5	4	3	5	5	4	4	3	3
Dose employed.. ...	3cc	3	3	3	2cc	2	2	2	1cc	2cc	2	2	2	2	2

B. INTENSIVE.

Day of Inoculation	1	1	2	2	3	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Number of days of desiccation of material ...	Mrn. 14 13	Eve. 12 11	Mrn. 10 9	Eve. 8 7	Mrn. 6	Eve. 6	5	5	4	3	4	3	5	5	4	4	3	3	5	4	3	5	4	3
Dose employed	3cc	3	3	3	2cc	2	2	2	2	1cc	2	1½	2	2	2	2	2	2	2	2	2	2	2	2

It will be noted that (B) is more prolonged than (A), and also that the strongest injection—*i.e.* of material dried only three days—is reached on the seventh day, while in (A) it is not administered until the ninth.

The inoculations are made alternately in the right and left flanks, and for the most part, patients complain of nothing during the treatment. In some cases a feeling of fatigue, enervation, and somnolence have been observed. Local inflammation does not occur at the seat of injection, but great care is taken by control cultures to make sure that each tube of material is pure.

The treatment is indicated in the case of any person bitten by a rabid animal; the main element of success being prompt resort to the method, the most frequent source of failure having been delay in its adoption. In young patients, especially those bitten about the head and face, or in cases of wolf or cat bite, early commencement of treatment is especially indicated. Bearing in mind the occasional very early appearance of symptoms, it is necessary for a favourable prognosis that inoculations should be commenced before the sixth day after the bite.

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VI. GLANDERS.

Ætiology.—This is a disease dependent on a specific organism (*Bacillus mallei*), occurring primarily in the horse, but transmissible to other animals and to man. It is also known as *Equinia*, or, when chronic, as *Farcy*.

Glanders is met with both in hot and cold climates, but is fortunately not readily acquired by man. Asses, mules, and goats contract the disease, and it is inoculable in guineapigs and rabbits. In some animals local sores only result from inoculation; this is usually the case in dogs. Oxen and swine are insusceptible to the disease. Both the acute and chronic (farcy) forms are common in the horse, but acute glanders is more frequent in man. In horses the chronic form is not always easy of recognition; hence, the disease may be overlooked either accidentally or with intent, and the animal worked for years. The bacillus of glanders much resembles that of tuberculosis in general appearance; it is, however, somewhat shorter and thicker, the ends more or less rounded, and indications of spore formation have been noted in it (page 35). It is not particularly resistant to heat, desiccation, or antiseptics, but experience has shown that the virus may remain active for months in infected stables; hence, special care is needed in disinfection. It is cultivable artificially on blood serum, agar-agar, or potato, and in the latter case produces highly characteristic brown masses. It is not coloured by the stains used for bacillus tuberculosis, and is readily decolorised by nitric acid. The organism is most readily shown in sections by staining with methylene blue, and decolorising with weak acetic acid (Schutz and Löffler); it is usually most abundantly found in small nodules of recent formation. Recently mallein, a substance similar to the tuberculin of Koch, has been employed hypodermically as a diagnostic and curative agent. It produces a characteristic reaction in glandered animals, but up to the present the results have not proved altogether satisfactory, as reaction sometimes occurs in animals in whom no trace of glanders is discoverable after death. It has been used in human glanders (Bonome).

Mode of infection.—Glanders may result from the bite of an infected animal, or a pre-existing wound or abrasion may become inoculated. It appears also that the contagion may be inhaled or arrested on the mucous membrane of the eye, nose, or mouth of persons standing near when a glandered animal sneezes. The disease may pass from man to man; but recorded instances of this are rare. Fatal cases have been observed from the accidental inoculation of workers with experimental cultures of the bacillus.

Period of incubation.—This may vary from one to fifteen days, but is occasionally much prolonged (Bristowe).

Morbid anatomy.—Glanders is characterised by the formation of nodules in the nasal mucous membrane, the respiratory passages, and internal organs; also by the development of nodules and abscesses in and beneath the skin, and in the muscles. In the chronic form, indolent swellings of the lymphatic glands are common; but in the acute, the glands often remain unaffected.

When affected, the nasal mucous membrane is dotted with numerous miliary papules, grey or yellowish-grey in colour, many of which may have broken down, leaving deep ragged ulcers, with bare or necrosed bone at their bases, the condition sometimes extending to the various accessory sinuses. Similar nodules are found in the larynx, trachea, and lungs. In the latter organs they are scattered throughout, and vary in size from that of a miliary tubercle to masses measuring an inch in diameter. The pulmonary deposits usually lie near the pleural surface, are whitish, and more or less arborescent in appearance, and may be unsurrounded by any marked inflammatory area; hence, pleuritic changes may be absent. Nodules may be found on the conjunctiva and in the skin generally; the latter corresponding to the cutaneous eruption. The abscesses in the limbs are deep and irregular, containing pus and abundant tissue *débris.* In acute glanders the secondary abscesses are indistinguishable from ordinary pyæmic deposits; in the chronic form there is a tendency to caseation. In the horse, the deposits of acute glanders when internal are usually limited to the respiratory passages; in farcy, numerous subcutaneous nodules connected by long indurated cords, corresponding to the lymphatic trunks, are met with.

Symptoms.—These may be either acute or chronic, while, as in tuberculosis, a chronic attack may at any time take on the characters of the acute form and rapidly prove fatal.

Acute form.—In man this form is the more frequent. The disease commences somewhat insidiously with malaise, headache, and pains in the limbs, and the patient often shows the same reluctance to speak of the possible origin of his disease as is noted in persons who have been bitten by a rabid dog. If a wound exist, it becomes painful and swollen, and surrounded by a bright red area, sometimes extending along the line of the lymphatic vessels. Occasionally signs of pulmonary affection first appear, but more often the patient complains of one or more hard painful swellings in the limbs, which soften rapidly, and when punctured may furnish at first thin blood-stained fluid, but always eventually pus.

Before the end of the first week deposits in the skin give rise to the characteristic eruption. The date of onset is somewhat variable; the whole body may be affected, and as the eruption is successive, nodules in varying stages undergoing progressive changes are found:—(1) Small, shotty, hard, red, papules; (2) somewhat rounded pustules often on a raised yellowish areola twice as wide as the pustule itself; (3) similar pustules on an inflamed red base; (4) large vesicles containing turbid serum, some of the size of bullæ; (5) the latter varieties dried up, leaving a scab upon an indurated base. At a later date suppurating ulcers may occupy the site of previous pustules, and the confluence of these may lead to the formation of somewhat extensive suppurating areas. In its various stages the eruption simulates closely those of varicella and variola. During the development of the eruption numerous abscesses of a pyæmic character form about the body, often deep and very extensive; or suppuration may occur in the joints. Deposits in the nasal mucous membrane, when they occur, are indicated by a thin watery discharge, becoming purulent and sanious as they break down; the nose at the same time becoming swollen, and reddened externally. Occasionally attacks of bleeding may be the only representative of this sign, which is inconstant. Deposits in the frontal sinuses give rise to severe headache, those in the conjunctiva are accompanied by much swelling of the eyelids, and those in the mouth or fauces to ulceration. Pulmonary signs may be those of pneumonia, bronchitis, or pleurisy.

General symptoms develop *pari passu* with the local manifestations. The bodily temperature, at first little raised, reaches as high as 105° to 106° F., fluctuating much as a pyæmic temperature, but without the frequent falls to normal. Profuse sweating is common; the pulse loses strength, and gains in rapidity; emaciation is rapid, giving a pinched appearance; the tongue becomes dry and brown; the urine contains albumen; sleeplessness is followed by delirium, often of a busy character, with picking at the bed-clothes; and the patient dies of exhaustion. Death may occur in less than one week, but is commoner in the second or third.

Chronic form.—In this the same complexity of symptoms is observed, but they are evolved slowly, the average duration of an attack being four months (Bollinger). The eruption and abscesses leave intractable ulcers and fistulæ. Nasal signs are less frequent, but chronic discharge may occur, with tendency to the formation of dark offensive crusts. Affection of the air-passages may give rise to hoarseness, sanguineous expectoration, pyrexia, and steady emaciation, and if the patient recover, convalescence is very prolonged.

Diagnosis.—In the initial stage the disease is to be recognised by the history alone, the early symptoms often simulating those of acute rheumatism or enteric fever. When fully developed, confusion with pyæmia is the main danger. The resemblance is the more striking since the secondary deposits closely simulate those of pyæmia, and both diseases occur in the same acute, sub-acute, and chronic forms. Attention to the history, the absence of initial rigors, the characteristic eruption, and the nasal discharge, if present, will aid in making a correct diagnosis. Virchow has pointed out the occasional simulation of a hard chancre by a nodule upon the penis.

Prognosis.—Acute glanders may almost be said to be a fatal disease, yet Bollinger reports one recovery in thirty cases; and the same observer has collected thirty-four cases of chronic glanders with a percentage recovery of fifty.

Treatment.—In the case of a bite or undoubted local inoculation seen early, excision and energetic disinfection or cauterisation are indicated. Later, the administration of quinine and abundance of good nourishment should be persevered in. Abscesses should be treated on ordinary lines, and the nasal symptoms by the spraying of carbolic acid solution (1 in 40) or perchloride of mercury (1 in 1,000). The internal administration of strychnine and arsenic has been recommended (Gamgee); and Bollinger suggests the use of carbolic acid. Lastly, cases of cure by the free inunction of mercury commenced early have been reported from Russia (Gold Gralewsky, Kondorsky).

VII. MALIGNANT PUSTULE. ANTHRAX.

Ætiology.—This is a gangrenous inflammation of the skin, due to the inoculation of a specific organism (*Bacillus anthracis*). The disease is one primarily met with in animals and from them is readily transmissible to man.

The disease in animals is widely distributed in both the Old and New Worlds. In England outbreaks are comparatively slight and uncommon, and a very large number of the cases observed in man are the result of infection from imported hides. It occurs in horned cattle, sheep, swine, horses, and wild animals, such as reindeer and buffaloes, and is readily produced by inoculation in rabbits and guineapigs. The carnivora are much less susceptible, though cats are more so than dogs.

The anthrax bacillus develops without the body in the form of bundles of long threads bearing numerous spores. The latter are extremely resistant to ordinary atmospheric conditions, heat, and desiccation, and remain capable of development for years if a suitable soil be offered.

The bacillus of anthrax is rod-shaped, with rectangular extremities, about $\frac{1}{3000}$ of an inch in length. It is at times somewhat enlarged at the ends, and shows signs of transverse fission—the method by which it multiplies within the body. In pleural exudation long threads similar to those developed in artificial media are seen. It is readily stained with gentian violet, Spiller's purple, or many aniline colours. In malignant pustule the organisms are found in the serum of the vesicles, and in great numbers in the deeper layers of the cutis clothing the papillæ, and obscuring their structure (Wagner, Charlewood Turner, R. Koch). They gradually extend on all sides, entering the blood-vessels and lymphatics. The central portion of the affected area rapidly becomes necrotic, and more or less extensive blood extravasations occur in and beneath it. Local reaction around is well marked, the tissues becoming hyperæmic, with small-celled infiltration and marked œdema. Bacilli are not demonstrable in the general circulation until some days after primary infection. (*See* page 31.)

The disease occurs in animals in two forms: an external, evidenced by the development of brawny swellings of the skin and subcutaneous structures, and an internal, involving the respiratory and alimentary tracts.

In man the same varieties are met with, the external being known as *malignant pustule.* This commences as an infection of the skin, and continues for a short time a local process. From the primary focus, however, the bacilli and the waste products of their metabolism enter the general circulation, and in the majority of cases cause death from septicæmia if surgical assistance be not forthcoming. Some individuals, however, offer a special resistance to the disease, and rare cases have been reported of spontaneous recovery. A previous attack offers no immunity from subsequent ones.

Pasteur, by cultivating the organism at an abnormally high temperature, has succeeded in lowering its virulence, and has thus produced a protective "vaccine," which is said to render animals immune to the disease. Wooldridge succeeded in endowing rabbits with immunity by injecting a chemical substance isolated from the blood of other rabbits which had suffered from the disease. The production of immunity by the injection of albumoses has been further worked out by Hankin.

Mode of infection.—Malignant pustule in man is met with almost exclusively on uncovered parts of the body. Infection may occur of a pre-existing pustule, wound or abrasion, or possibly by the bite of an insect which has rested or fed upon infected matter. The handling of infected hides is a common source of infection; and internal anthrax may result from inhaling or swallowing dust from them. Those employed in tending infected animals, veterinary surgeons, and slaughterers may be directly inoculated. Anthrax has been conveyed to a surgical wound by the use of catgut prepared from the intestine of an infected sheep.

Symptoms.—The first sign of malignant pustule, when no appreciable wound exists, is a dark spot resembling the sting of an insect, often surrounding a hair follicle. The papule enlarges, and a vesicle containing yellowish fluid forms, often changing its colour to red or a bluish-red colour. A similar papule bearing a vesicle may develop at the site of any small abrasion which has served as a point for infection. The appearance of the vesicle is heralded by burning pain, often leading the patient to scratch and rub the vesicle, thereby laying bare a raw red surface, from which serous fluid is exuded. A dry scab now forms on the surface, the edges of the papule enlarge and become much indurated, a hyperæmic and œdematous areola developing around it. A second ring of vesicles may develop around the centre of the papule, the centre meanwhile becomes dark from the extravasation of blood, and gradually dries and sinks below the level of the surrounding areola (Fig. 54). The process may continue in this manner till the carbuncle reaches a considerable size, the same swelling and œdema preceding its enlargement. At the same time, the poison is taken up by the lymphatics, the glands enlarge; and if a limb be affected, the whole extent may become swollen and œdematous. The appearance of the œdematous part varies in different regions: thus in the eyelid a semi-transparent bladder-like swelling is not uncommon, while the lips become thickened and rigid, the normal folds of the face being obliterated. Malignant pustules of the neck offer a special danger in the spread of the œdema to the larynx, tongue, and floor of the mouth. Of 1,077 cases collected by W. Koch, 490 affected the head and face, 45 the neck, and 370 the upper extremity. In some instances the process may closely resemble an ordinary cellulitis, highly œdematous, but without the development of vesicles.

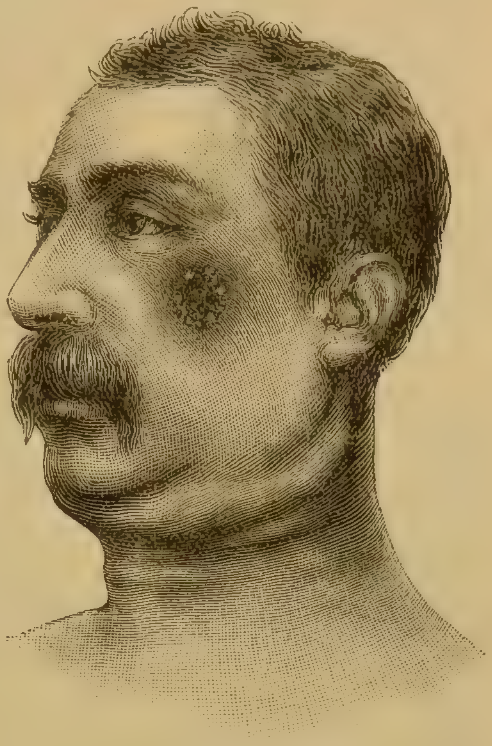

Fig. 54.—Malignant Pustule of Face. The enlargement of the submaxillary glands and the surrounding œdema are well shown.

For a period of about two days—in fact, prior to the entrance of the bacilli and their products in any large amount into the general circulation—the patient suffers little, except from a feeling of general malaise. The temperature then rises to 102° to 104° F., gradually falling intermittently; and if the case be treated by early excision, reaches normal in a few days. The malignant pustule itself is the seat of remarkably little pain, but acute pains may be experienced in the limbs. In fatal cases, sweating and diarrhœa set in, accompanied by delirium, fall of temperature, often below normal, great prostration, and eventual collapse.

Malignant pustule may serve as a local centre from which embolic secondary deposits are furnished, or it may occur secondarily to anthrax of internal organs.

Diagnosis.—The recognition of malignant pustule is usually easy, by observing the special appearances and carefully inquiring into the history. Davies Colley has pointed out that it is to be distinguished from facial carbuncle by its comparative painlessness, the dark centre, and the absence of the multiple yellow spots of commencing suppuration seen in the latter affection. Diagnosis can be made certain in doubtful cases by microscopic examination of the fluid in the vesicles or by the inoculation of a rabbit. It is well to bear in mind that the bacilli are only present in the blood after general symptoms have commenced to develop.

Prognosis.—Malignant pustule rarely undergoes spontaneous cure. If treated energetically, the majority of the cases recover rapidly. Lengyel and Koranyi lost only 13 out of 142 cases treated surgically. If secondary deposits occur, or signs of internal anthrax appear, the patient usually dies rapidly. In uncomplicated cases not treated, death occurs in from five to eight days.

Treatment.—The only treatment to be recommended is free excision, and this should be followed by cauterisation. Davies Colley has published several cases in which chloride of zinc paste or liquid carbolic acid were successfully used for the subsequent cauterisation. The local œdema and general symptoms usually rapidly subside after removal of the infecting centre, and it is difficult to say when it becomes too late to be worth trying this mode of treatment. In cases where excision is impossible, or such treatment refused, parenchymatous injections of 2 per cent. carbolic acid solution may be tried, as good results have been obtained by this method. They should be radial and frequently repeated, 20 to 30 minims of the solution being employed.

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VIII. ACTINOMYCOSIS.

Ætiology.—This is a disease dependent on the entrance and growth of the ray fungus, or actinomyces (a species of Cladothrix, Boström), into the system of man.

The disease corresponds with that observed in oxen, calves, swine, etc., with some modifications, the most important of which are the less marked tendency to localisation by the abundant formation of scar tissue, and to calcification. The typical course of actinomycosis in man is chronic, patients dying as the result of implication of important organs, of exhaustion from prolonged suppuration, or from amyloid disease. It must be borne in mind, however, that the tissues re-act in correspondence with individual idiosyncracy and constitutional conditions, and in some persons a defective resistance on the part of the tissues allows a rapid invasion and course in this disease. The morbid process spreads by direct continuity, invading all tissues indifferently; secondary embolic deposits may, however, occur, the most common examples of which are those observed in the liver in primary actinomycosis of the intestinal canal. Cases of so-called acute generalisation have been described, but these are probably partly pyæmic in nature.

The actinomyces itself is not pyogenic in the same sense as the various suppuration organisms, since it may exist for long periods in organs such as the brain (Bollinger) without inducing suppuration. When developing actively, however, it produces local necrosis of the tissue in which it is embedded and of the small-celled inflammatory infiltration surrounding it, and in this way a fluid more or less resembling true pus is furnished. Deposits in the alimentary and respiratory tracts are necessarily exposed also to external contamination, and in these cases abundant suppuration, induced by the ordinary pyogenic organisms, often occurs. The disease does not extend by the lymphatic system; and although the glands have been occasionally noted to be enlarged, no growth of actinomyces has ever been observed in them.

Mode of infection.—No evidence exists of the direct transference of this disease from animals to man, or from man to man. Both men and animals appear to be infected from the same source and in the same manner. The most common seat of infection is the mouth, the alimentary tract, some of its dependences, or the respiratory passages. Cases of external infection are rare, but a small number has been placed on record. The discovery of a grain of barley in an abscess of the back secondary to a primary deposit in the œsophagus (Soltmann) led to the inference that the carrier of infection might exist in grains of corn or barley, or in particles of straw of a like nature. In five carefully examined cases in man, bodies of this nature were discovered by Boström in primary actinomycotic deposits, and the extension of the process was shown to follow the line in which the grain had travelled. Boström strengthens the theory of this mode of infection by the following arguments:—In thirty-two cases of bovine actinomycosis, particles of straw and grain were found embedded in the mucous membrane between the teeth and gums and in the tongue; and further examination of the records of eighty-four published cases of actinomycosis in man showed that the disease commenced in 77 per cent. during the months August to January inclusive: in fact, during the months in which the freest opportunities for the chewing of corn and barley grains are afforded.

The common localisation of the disease to the jaws and neighbouring tissues gave rise to the theory that the existence of carious teeth led to the development of the process by affording a suitable nidus for the growth of the organism and its extension by the tissues of the alveolus. The fact that the disease is especially common in calves at the time of the second dentition appeared to support this; but the exclusion of carious teeth as the point of origin in Boström's cases of maxillary actinomycosis in man is against the theory; and it may be at the same time pointed out that disease of the jaws in man has been almost without exception peripheral in arrangement, while central disease would more naturally be expected if the process started from within the alveolus. In calves, however, disease of the jaws is often central, great expansion of the bone taking place. In other observed cases the origin of the disease has been clearly traced to chewing barley; and in two cases of external actinomycosis of the hand, in one the abscess developed in the palm, as the result of pressure of the sickle while reaping; and in the other a nodule developed on the back of the hand of a person engaged in thrashing grain (Bertha).

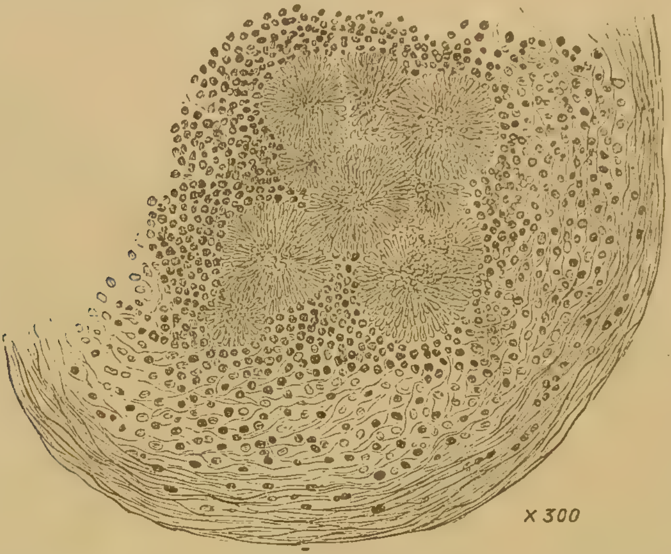

Fig. 55.—Section of an Actinomycotic Centre in a Cow's Tongue. Marked development of connective tissue and clubs. The actinomyces are treated diagrammatically, to illustrate the appearance usually observed in those obtained from urine, or pus.

The actinomyces.—The organism itself, obtained from the discharge from an abscess or fistula, or from the urine or fæces when the alimentary or urinary tracts are the seat of the disease, presents the following characters:—It is found in small nodules, varying in size from a grain of sand to a hemp-seed, rounded or mulberry-like in form, grey, greyish-green, sulphur yellow, or, when obtained from the fæces, dark brown in colour, and of the consistence of soft mortar. The variation in colour from grey to yellow appears to depend on the age of the organism, the older ones gaining the characteristic yellow colour; and it is these in which club formation is most marked. When first obtained, they are still clothed in a layer of round cells; but treatment with a few drops of weak caustic potash allows the following structure to be made out microscopically. Pressure of the cover-glass causes the primary nodule to break up into smaller ones, each apparently consisting of a series of club-like processes, radiating like palisades from a centre (Fig. 55). These processes are the so-called clubs, regarded by Boström and many observers as a degeneration of the terminal filaments of the fungus. The ultimate structure is

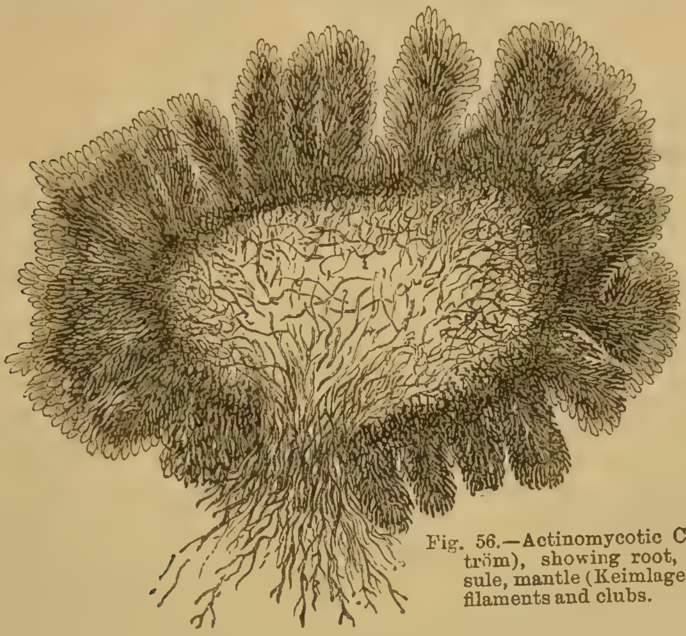

Fig. 56.—Actinomycotic Centre (Boström), showing root, hollow capsule, mantle (Keimlager), radiating filaments and clubs.

only to be made out in prepared sections of tissue. When so examined, each centre consists of an intricate mass of filaments, developing spores, and arranged in the form of a hollow capsule deficient at one spot, from which filaments pass from the interior into the surrounding tissue, forming a kind of root. The centre of the sphere consists of irregularly-arranged filaments branching dichotomously, the branches equalling the parent filaments in size. When they reach the periphery the dichotomous division becomes extremely free, and a highly intricate network is formed, in the meshes of which abundant round or oval spores are entangled. Beyond this bounding network, called by Boström the mantle or germ stratum (Keimlager), a series of slender radiating filaments project, at first branching little, and then abundantly, and beyond these the clubs are arranged, often more or less cast off from the central portion of the organism. Each club consists of a central filament enveloped in a laminated gelatinous sheath, pyriform in outline, or sometimes ending in finger-like processes. Between the clubs, slender wavy or spiral filaments, sparse or in groups, project into the surrounding tissue (Fig. 56). In the smallest young grey nodules, clubs may be sparse or absent; with increased age the clubs increase, while the filaments decrease in amount. The whole organism so arranged may be infiltrated with small-celled growth, especially the centre, and the whole may calcify, still retaining, however, the general outline of the original structure. The youngest actinomycetes are found in the discharge and breaking-down tissue only; they may be too small for naked-eye recognition, and sometimes the discharge may contain more mycelial threads than completely developed organisms.

Branched and single threads have been observed by Boström within cells, which later perish, and the contents are set free—a possible mode of extension of the local process. The actinomyces are often very sparsely distributed, especially in cases in which cicatrisation is a marked feature. (*See* page 49.)

Morbid anatomy.—The entrance of the actinomyces is followed by local necrosis of the tissue in which it is embedded, and the development of a surrounding area of small-celled infiltration, the amount of which varies with the power of resistance of the individual affected and the activity of growth of the organism. If resistance be strong, considerable inflammation is set up, the tissues around undergo cell multiplication, granulation tissue is developed, and well-marked connective tissue at the periphery. In these cases the disease spreads slowly, and the tendency is to localisation. If, on the other hand, resistance be defective, the small-celled infiltration remains the main feature, and the process spreads rapidly by destruction of the surrounding tissues. Attention has been drawn by Boström to the strong resemblance exhibited by these two varieties of the process with those observed in chronic and acute tuberculosis. The process observed at a single centre is repeated indefinitely by the development of fresh ones in direct continuity; and thus in the chronic cases large areas of doughy granulation tissue, bounded by dense scar tissue in the older parts, are formed.

Deposits invade tissues of the most varied character, infiltrating muscles, fasciæ, and organs in continuity, eroding and infiltrating bones, more or less obliterating serous cavities, such as the pleura, peritoneum, or joints. Blood-vessels, nerves, and tubes, such as the gullet, become infiltrated, and are rigidly walled in. The secreting tissue in the affected area of organs is destroyed without taking active part in the process, which in its extension sometimes encloses small islands of the original organ more or less unchanged. The bones are eroded, become rough and discoloured, and the periosteum is thickened, the condition being one of superficial actinomycotic caries. In chronic cases abundant osteophytic formation takes place, while in acute cases this may be absent, the process being merely rarefying and destructive. The joints may become invaded and extensive disorganisation follows.

Softening sooner or later occurs from the fatty degeneration, liquefaction, and disintegration of the small-celled infiltration and granulation tissue, the result varying from a thin serous or slimy fluid, containing disintegrating tissue, free oil globules, and a few leucocytes, to apparently well-marked creamy pus, sometimes darkly coloured from admixture with blood. In addition, actinomyces and small round bodies, regarded by Boström as spores of the organism, form the specific elements of the fluid. In acute cases large abscesses may form; in the more chronic the collections of fluid are more or less localised to the individual areas surrounding the organisms, with the result of the formation of a highly characteristic sponge-like mass. Boström considers this liquefactive process an index to the life and activity of the organism, and says that when a centre is exclusively formed by granulation and cicatricial tissue, the fungus is either inactive or dead.

Symptoms.—The typical sign of actinomycosis is the development of a chronic tumour. This enlarges steadily, with a tendency to reach the surface, when portions become more prominent, soften, and offer pseudo-fluctuation. If extension be in the direction of the skin, this becomes reddened or bluish-red, and later a yellow point may form on the most projecting spot, and spontaneous opening may occur. The marginal portion of the swelling is characterised throughout by great induration in the majority of instances. When softened spots are incised, serous fluid and a little blood only may escape; and a mass of soft granulation tissue, yellowish from fatty degeneration, or brownish from hæmorrhage, is exposed. In other cases pus may be found, thin, slimy, and sweet, or dirty, brown-coloured, and offensive smelling. Whether forming spontaneously or as the result of incision, chronic fistulæ persist, the mouths of these being irregular in outline, surrounded by an area of livid skin, often more or less undermined, and lined with soft readily-bleeding granulations, often speckled with deposits of the organism. The discharge contains more or less numerous actinomyces or actinomycotic elements. The general tendency is to spread constantly, the older portions of the infiltration inclining to cicatrisation. This tendency is so marked that when the case comes under notice the original site of infection may have so completely healed as to be unrecognisable, and to be traced only by the following up of cicatricial bands at the time of operation (Boström). The whole process is commonly attended by little pain, fever, or constitutional disturbance. In some cases, however, the process may, as already explained, be much more acute; and here fever is a marked symptom, sometimes pyæmic in character, and exhaustion may rapidly cause a fatal termination.

Primary infections of the mouth are common, and in these cases the disease has a special tendency to extend along the jaws. A chronic swelling, often giving rise to little or no pain, slowly creeps along the jaw, a feeling of tension in opening the mouth is followed by actual difficulty in moving the jaw and subsequent fixation, the tumour spreading and involving the neck or parotid region; or in the case of the upper jaw, creeping back and involving the base of the skull. In other cases the infiltration may extend directly downwards to the neck, often involving the vertebræ. In most instances the swellings follow the chronic course above set out, but acute cases have been observed attended with severe pain, fever, and œdema of the floor of the mouth, tongue, and glottis, disappearing only with spontaneous opening or incision of the affected area, the symptoms resembling those of angina Ludovici (Partsch).

When the pharynx is the seat of primary affection, difficulty of deglutition may precede the formation of a cervical tumour, and dragging pain in swallowing has been noted in cases of primary infection of the œsophagus. Infection of the gullet is of especial importance, as thence the process extends readily to the posterior mediastinum, spine, and lungs, and may come to the surface on the back. When the lungs are affected, primarily or otherwise, hacking cough, expectoration (sometimes blood-stained), fever, sweating, and emaciation may precede the formation of localised empyemata, or the development of large masses of granulation tissue filling the pleural space and replacing lung tissue. The abdominal organs may be involved secondarily to primary infection of the intestine, or by invasion from the thoracic space. Affection of the intestines may be heralded by pain and digestive derangement, followed by characteristic swellings and abscess formation. Pus from the fistulæ often resembles that from a fæcal abscess, and actinomycetes may be present in the stools. The liver or spleen may be much enlarged and characteristic abscesses form in them; and the kidneys or urinary passages may also be involved secondarily. In the latter case actinomycetes may be discharged in the urine. Any abdominal or pelvic organ may, in fact, be invaded. Pressure upon the veins by the indurated masses may occasion widespread œdema in any situation.

Diagnosis.—The chronicity of actinomycotic disease is its main characteristic, and the diagnosis may be rendered certain by discovery of the organism in the discharges or in the affected tissue. It should be borne in mind that the organism may be very sparsely distributed and is most likely to be found in the discharges and soft granulation tissue. Tumours have been mistaken for sarcomata, and the question of actinomycosis may arise in any chronic alveolar swelling, or in some tubercular deposits. Actinomycotic disease of the vermiform appendix may simulate ordinary appendicitis. The common absence of enlargement of the lymphatic glands is a valuable diagnostic point to be borne in mind.

Prognosis.—Spontaneous recovery has not been observed, though it appeared possible in a case of pelvic actinomycosis implicating the urinary and intestinal tracts, reported by W. B. Ransome. The prognosis depends entirely on the possibility of radically removing the disease. When this is possible, the prognosis is good; when vital organs are implicated, or from the position of the deposits removal is impossible, death occurs sooner or later. Patients may, however, live for years.

Treatment.—Extirpation of the deposits with the knife is by far the best treatment. Where complete excision is impossible the sharp spoon gives good results and the two methods may often be combined. The affected tissue readily scrapes away, but care must be taken with the firm peripheral portions. Scraping operations may be followed by slight local relapses, which must be treated in the same manner; the wound may be painted with a strong carbolic acid solution. The course of the disease is said to be checked by the administration of iodide of potassium (Ransome, Thomassen, Nocard, Meunier, Netter).

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IX. TÆNIA ECHINOCOCCUS.

Ætiology.—A cestode worm normally inhabiting the intestine of the dog, the ova of which when ingested by man, who serves as an intermediate host, develop into the so-called bladder stage, producing *hydatid cysts.*

Hydatid disease is met with in all countries in which the dog has his habitat, but it is especially frequent in Iceland, Silesia, and Australia, and is by no means rare in Great Britain. The parasite has been discovered in wolves, jackals, and a cat found in Brazil, and may possibly infest other animals.

The *worm* itself is about a quarter of an inch in length, and made up of four segments; of these, the first is the head, about $\frac{1}{100}$ of an inch in width, furnished with four pairs of suckers, and a central rostellum crowned with a double circlet of hooklets, thirty to forty in number. The second joint is insignificant; the third about twice the length of the second; while the fourth (proglottis) contains the double sexual organs, a marginal reproductive papilla, and a large number of eggs, amounting to from 500 to 4,000.

Thousands of these worms may exist in the intestine of the dog, more or less completely hidden by the villi. Oxen, sheep, and swine are far oftener the intermediate hosts of the parasite than man, and these animals, probably in great measure, keep up the supply of fresh scolices for the further propagation of the disease in the dog. Sheep dogs and those kept for sporting purposes are specially liable to be affected; and persons whose occupation, as is the case with shepherds, bring them into constant and close association with these animals, are the most frequent sufferers from the disease.

Hydatid cysts may be single or multiple; their increase is usually slow, but the rate may be variable; in some cases they may exist for a whole life-time. They are most common between the ages of twenty and forty, are commoner in women than men, and comparatively rare in children.

Mode of infection.—The ripe proglottides and globular thick-walled eggs expelled with the fæces of the dog are distributed over pastures and edible vegetables, and reach supplies of drinking water, and are therewith ingested by man.

The egg loses its chitinous capsule in the alimentary canal, and the small embryo, provided with six hooks, migrates into the tissues, afterwards undergoing cystic transformation. A period of five months elapses before the cyst reaches the size of a pigeon's egg, the stage at which scolices and brood capsules begin to develop.

Morbid anatomy.—As with other parasites, the entrance of the embryo into the tissues gives rise to irritation and the formation of a surrounding zone of small-celled infiltration, which gradually develops into a well-formed fibrous capsule — the *ectocyst* of Huxley. Loosely attached within this is the true cyst wall, or *endocyst*, of Huxley. This appears yellowish or dull white, and consists of two elements; the first, a highly elastic chitinous membrane arranged in concentric laminæ, the inner layers being those last formed. When set free, this membrane curls up in a highly characteristic manner, the outer layers of the wall being firmer than the inner, and microscopic examination reveals the striation due to its lamination. Within this layer is found the germinal membrane, composed of cells, granular matter, particles of carbonate of lime, muscular tissue, and a water vascular system.

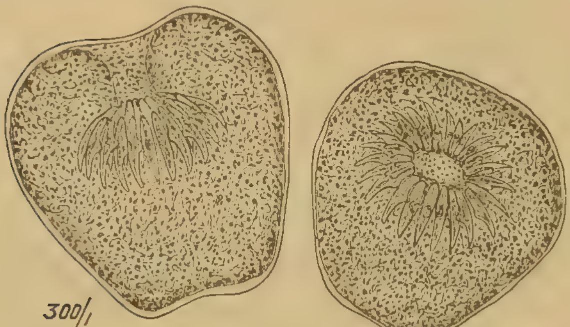

Fig. 57.—Scolices of Tænia Echinococcus.

A cyst so composed may remain sterile (acephalocyst), and yet attain a considerable size; more commonly the germinal or parenchymatous layer gives origin to numerous brood capsules, scolices, and daughter cysts.

The *brood capsules* originate, when the parent cyst has reached the size of a walnut, as small hollow elevations of the parenchymatous layer, and from these numerous scolices—as many as thirty or forty—may be developed. The *scolex* represents the head of the entire tapeworm; it varies in length from $\frac{1}{100}$ to $\frac{1}{60}$ of an inch. It is solid, cylindrical in form, somewhat egg-shaped posteriorly, whence a stalk containing nutrient vessels and muscular tissue connect it to the parent cyst wall; when detached the scolex dies (Fig. 57). The anterior part, separated from the remainder by a slight constriction, the neck, bears four suction-discs, and a central rostellum with a double crown of hooklets. The latter may be projected when dead, but earlier lies retracted in a canal formed by the invaginated neck. The body of the scolex contains numerous calcareous particles.

Besides brood capsules and scolices, the parent cyst produces *daughter cysts*, from which, again, cysts may develop for several generations, even to reach the number of thousands (echinococcus colonies). The mode of formation of daughter cysts may be endogenous or exogenous. Endogenous cysts, the commoner form, are the result of transformation of scolices or brood capsules; exogenous cysts are formed by the hernia-like protrusion of pouches from the parenchymatous through the cuticular layer of the cyst wall. Such cysts may lose all continuity with the parent cyst lying within the adventitious capsule. Such is the common condition of multiple hydatid cysts in bones; and the same mode of development is common in the mesentery.

Besides solid elements, the cysts contain fluid, with somewhat special characters. *Hydatid fluid* is clear, limpid, colourless, or slightly opalescent, of low specific gravity (1,004 to 1,015) normally contains chloride of sodium and no albumen, and usually scolices or hooklets. If the hydatid has been inflamed or is dead, albumen is present in greater or less amount (Heller). Succinic acid, in combination with sodium or calcium (Heintz), has been frequently found a constituent; also sugar, leucin, and tyrosin in hepatic cysts, and uric and oxalic acids in renal. In old hydatids the fluid may be turbid and blood-stained, or actually purulent and fœtid.

Another form of hydatid—the *multilocular*—has been described by Virchow; here no mother cyst is demonstrable, but numerous vesicles, from the size of a pea downwards, are embedded in gelatinous substance and enclosed in fibrous trabeculæ. The vesicles are often shrunken and mostly sterile, although some hooklets are always to be found. This form is most frequently met with in the liver and is rare.

The **size** attained by individual cysts depends in great measure on their surroundings; when these latter are readily compressible, the cysts may attain an enormous size. When the cysts are so situated as not to occasion dangerous pressure effects, they may exist for an indefinite time. The general tendency, however, is towards death of the parasite: when this occurs, the fluid may become absorbed, the cyst wall falls in, the adventitious capsule contracts, and spontaneous cure is effected. After this process an aggregation of soft shrunken cysts, or a solid caseous mass, liable to more or less calcareous degeneration, may represent the original colony. In other cases—especially in the abdominal cavity—suppuration may occur. The most dangerous termination, due usually to the pressure exerted by the accumulation of fluid and daughter cysts, is rupture; in the abdomen this may lead to acute inflammation or signs of collapse and death, or, in the lung, the sudden accumulation of fluid in the air-tubes may suffocate the patient.

The commonest **seat** of this disease is the liver, then the lungs and kidneys; cysts may, however, occur in any organ of the body, or in the connective tissue throughout. The common selection of the liver, no doubt, depends on the ease with which the parasite reaches the portal circulation. Cysts may be single or multiple ; the latter condition is far less common, and is most often seen in the peritoneal cavity. In cases of multiple hydatids the liver rarely escapes.

Symptoms.—The symptoms of hydatid tumours depend entirely on the region or organ affected, the early ones being the result of pressure on the surroundings of the cyst, the later ones on the supervention of inflammation, or possibly on rupture of the cyst. Spontaneous cure of the hydatid, with consequent contraction of its bulk, may lead to the cessation of all symptoms, or this may occasionally result from rupture of the cyst, with discharge of its contents, either into a hollow viscus or externally.

The signs of a hydatid cyst are those common to collections of fluid, the special characteristics being high tension and the occasional existence of a peculiar vibratory thrill, obtained by placing the fingers of the left hand on the tumour, and tapping the middle finger with one of the right hand. Rupture or puncture of a cyst is sometimes followed by the development of an urticaria-like or diffuse erythematous rash, accompanied by much itching. It must be borne in mind that shrunken cysts often persist as firm solid tumours, occasionally pedunculated and very movable.

Diagnosis.—The diagnosis is usually made by exclusion ; in some cases it is self-evident if daughter cysts be discharged. In cases of doubt, puncture may clear up all difficulty, but in cases of visceral hydatids this should never be undertaken willingly without consent having been obtained for the surgeon to proceed to a radical operation, and in some cases is strictly contra-indicated as a dangerous proceeding.

Prognosis.—The disease may give rise to little trouble, and all symptoms may subside spontaneously; it is remarkable how often old hydatids are met with in the autopsies of persons dying from other causes. Beyond this, the prognosis depends on the organ affected, and if this is one to be readily reached and operated on, the prognosis is decidedly a good one. Spontaneous opening of the cyst may, either in the case of the lung or of the intestinal canal, be followed by recovery.

Treatment.—The surgical treatment of hydatid disease consists in either incision and drainage, or complete removal of the cyst at one operation.

In the subcutaneous tissue the cysts are readily extirpated entire. When within a serous cavity, the mode of procedure differs as to whether inflammation has occurred or not. If suppuration has occurred, a free incision may be carried into the sac, if adhesions have shut off the general serous cavity. The sac should then be irrigated, and the membrane as freely evacuated as possible by gentle manipulation ; drainage will, in these cases, be necessary for a considerable period.

When no inflammatory changes have occurred, Volkmann's operation in two stages is the safest. In the first stage, the cyst is laid bare by an incision. the wound is packed to ensure gaping, and an interval of three to five days is allowed for the development of adhesions, shutting off the general serous cavity. On the fourth to the sixth day the wound should be uncovered and the cyst freely incised, after the presence of sufficient adhesion has been determined. The fluid and daughter cysts having been evacuated, the loose adhesion of the true cyst wall to the adventitious capsule generally allows the former to be completely separated, either by irrigation or gentle manipulation. A drainage-tube is inserted, and kept in for from forty-eight hours to a few days. Recent progress in aseptic methods allows the whole operation to be done in one stage, the cyst being sutured to the parietal serous layer and skin, if necessary, after slacking of the tense cyst by puncture with a very fine trocar. This offers some advantage with regard to time, but is less safe and in ordinary cases not to be recommended.

In cases of sub-phrenic hydatids, or hydatid cyst of the upper surface of the liver, it may be necessary to reach the tumour through the thorax. When suppuration has occurred, it is not rare to find the lower part of the pleural cavity obliterated, and in this case, after partial resection of a rib to get sufficient room, the cyst may be at once evacuated. If this be not the case, the parietal pleura of the lateral wall and that of the diaphragm must be united with sutures and the diaphragm incised, and the operation completed in a subsequent second stage, as already described. In the case of large visceral or subperitoneal hydatids, it is well to bear in mind that it is not always possible to extirpate the cyst wall completely, and that in such a long and very chronic fistula may persist for some time, in fact in such cases prolonged suppuration may give rise to lardaceous disease of the viscera. In some cases of hepatic, and in renal hydatids, the sac may be reached from the lumbar region without injury to the peritoneum. Mention should also be made of Landau's method of so suturing the liver to an abdominal wound as to bring the cyst into view, in cases of affection of the upper or posterior surfaces.

Pedunculated hydatids should be removed entire. When multiple, the cases are often beyond surgical treatment; two or three are best treated by successive operation; when more numerous the dangerous alternative of puncture is all that remains. The surgeon may occasionally be called to a case in which rupture of a cyst into the peritoneal cavity has occurred; such information as exists on this subject is decidedly in favour of incision, irrigation with sterilised water, and drainage.

Mention must be made of the time-honoured treatment by puncture in order to emphasise its dangers. It should never be resorted to except as a preliminary to incision, and here only when in doubt. Although many cases of apparent cure are on record, yet the occurrence in favourable cases of hydatid urticaria, in others of death from acute poisoning, or from flooding of an organ such as the lung, and the possibility of causing spread of the disease by infection of a serous cavity (Volkmann), absolutely contra-indicate it as a normal method of treatment.

X. DELHI BOIL.

Ætiology.—This disease is known also as *Oriental sore* (Aleppo boil, Biskra button, Gafsa button, Kandahar, Penjeh, or Natal sore).

It is a local sore, probably the result of infection by organisms, the nature of which has not been discovered. Considerable doubt exists as to the specific nature of the disease, which is met with in tropical and sub-tropical regions under various names.

In India it has been especially commonly seen in Delhi, though less frequently of late. It has also been common in other parts of India, Asia Minor, Arabia, Persia, Syria, northern Africa, the Mediterranean islands, etc.

Its occurrence is somewhat influenced by season, being most frequently met with at the end of the wet season in India; in Biskra particularly in September, October, and November. Sex and age affect its occurrence little, and one attack gives no immunity from recurrence. The boil develops on the uncovered parts of the body, especially the face, and successive inoculations may occur in the same subject. The period of incubation is very variable.

Mode of infection.—It has been suggested that infection by water used for ablutionary purposes is a source of infection; also that winged insects may transport the virus.

Symptoms.—Local itching, which may last some time, is followed by the development of a small red area, not unlike an inflamed mosquito bite, from which radiating vessels sometimes pass. The red spot becomes papular, gradually increasing in size, until it reaches the size of a pea or more, and desquamates. It is indolent, pricks on pressure, and is entirely limited to the skin and subcutaneous tissue. After some weeks, or even months, the dull red surface is seen to be dotted with deep yellowish-white spots, possibly inflamed hair follicles, and later serum exudes from the central part, gradually drying and accumulating in a scab. The scab increases in thickness to as much as a quarter of an inch, is greyish in colour, or, if hæmorrhage occurs, of a brownish hue. It is very adherent, and if left alone, may last for months. Beneath this scab ulceration begins, progressing the more rapidly with the less care given to the part. The ulcers are indolent, oval or irregular in outline, the margins sharp or bevelled in character, the floor uneven, covered with yellowish or sanious pus, and in parts, especially in the centre, covered with frambæsia-like prominent granulations sometimes dotted with minute blood extravasations. The ulcers may reach three-quarters of an inch in diameter; they may be single, grouped, or multiple on different parts, to the number of forty or fifty. There is little surrounding inflammation and no lymphatic glandular enlargement.

The *duration* of the process is very variable. After from two to six months the scabs may separate spontaneously, leaving an incomplete thin red cicatrix, with rapidly-healing granulations in the centre

In other cases the ulcer heals very slowly from the margins, often giving rise to considerable deformity, such as ectropion or deformity of the alæ nasi ; the hair follicles over the affected area are destroyed. When on the limbs, cicatricial contractions of the joints may result.

The local process is accompanied by no constitutional symptoms, but may be complicated by more or less serious attacks of acute lymphangitis or erysipelas.

Diagnosis.—The diagnosis must be made by attention to the history and the district in which the disease originated.

Prognosis.—This is good as to recovery, but cicatricial contraction may give rise to permanent deformity. Complications such as those mentioned above may give rise to a fatal issue. The progress of the disease may extend over months, or even a year or more.

Treatment.—The papule in the early stage may be destroyed with the actual cautery (Murray), or acid nitrate of mercury may be used for the same purpose. Painting the papules with a solution of iodoform in collodion is recommended by Hickman. When ulceration has occurred, the sharp spoon may be used to remove granulations and the base of the sore treated in a similar manner (Woolbert).

In any case, care should be taken that the discharge from the sore is not allowed to produce a fresh spot by inoculation ; and bearing in mind the possible origin of the disease in the water used for washing, this should be sterilised by boiling before use.

XI. CHIGOE OR JIGGER.

Ætiology.—This is an insect (*Pulex penetrans*) resembling the common flea, but about one-half to one-third of its length, and provided with a proboscis equalling in length the remainder of the body.

The insect is met with in the tropics of Africa, America, and the West Indies, attacking natives, and not infrequently Europeans, especially children who are apt to run about with bare feet. The neighbourhood of the toe-nails is the favourite seat of invasion, but the scrotum and other parts of the body are occasionally affected. The impregnated female buries her head and proboscis in the skin and swells up, remaining *in situ*, if not interfered with, until her eggs are matured and extruded, and causes local irritation, inflammation, and suppuration.

Treatment.—In the case of Europeans, care should be taken in affected districts not to place the bare feet on the ground. Natives may anoint the feet with turpentine or carbolised oil as a protective. When the insect has obtained entrance, it should be removed with a blunt needle, care being taken not to injure the body, or troublesome ulceration may ensue. If the insect be injured in extraction the wound should be treated with a strong solution of nitrate of silver

XII. TRICHINA SPIRALIS.

Ætiology.—This is a nematode worm, which, when introduced into the alimentary canal, develops numerous embryos, which migrate from the alimentary canal into the voluntary muscles of their host.

The worm infects many animals—swine, rats, cats, dogs, hedgehogs, moles, horses, calves, rabbits, guineapigs, etc.

When an animal is fed on flesh containing the embryos, the worm develops rapidly, and fully-developed examples may be obtained from the intestine at the end of two days. The female is the larger ($\frac{1}{12}$ to $\frac{1}{9}$ of an inch in length), and when six days old is found to contain numerous embryos, averaging at least 150 to each parent. The male worm is shorter ($\frac{1}{18}$ to $\frac{1}{14}$ of an inch in length), and provided with two conical projections at its caudal extremity.

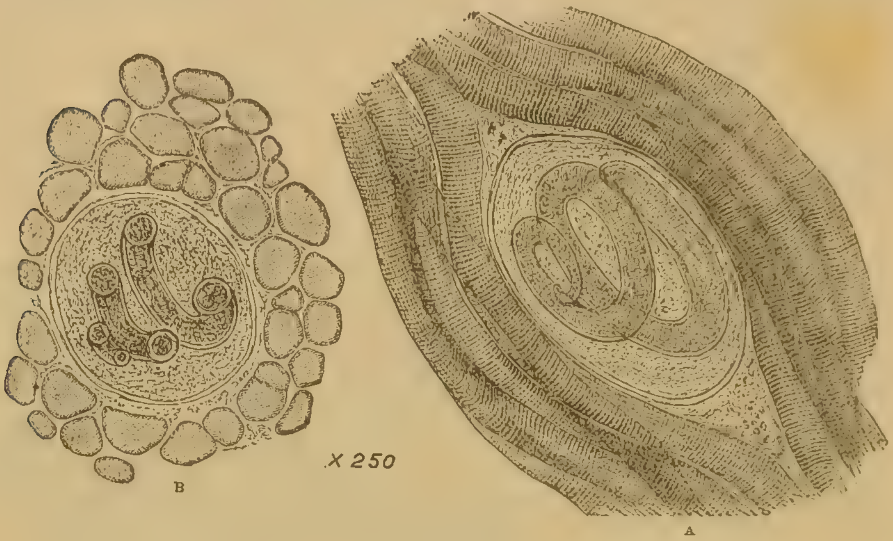

Fig. 58.—Sections of Muscle containing Trichina Spiralis.
A, Longitudinal; B, transverse.

The embryos set free in the intestinal canal migrate into the tissues, and are carried by the lymph stream into the general circulation (Askanazy), thence to be distributed in the voluntary muscles—their special habitat. Here they acquire an alimentary canal and rudimentary sexual organs, and at the end of the fourth week from leaving the intestine they assume a rolled-up form, and an adventitious capsule is developed around them (Fig. 58). This capsule is provided by the enclosing tissue, and more or less rapidly undergoes calcification, this process reaching a high degree at the end of twelve months. The parasite now remains quiescent, and may in time degenerate and perish; or by the death of its host, when an animal, it may be transferred to a new one, and again proceed to full development. Human muscle containing the parasite is pale or reddish-grey in colour, and the fibres tend to lose their normal striation and become fissured.

Mode of infection.—The disease is acquired by eating trichinous flesh—commonly pork, either raw or imperfectly cured or cooked.

Symptoms.—The special symptoms of trichinosis depend on the affection of the voluntary muscles; these appear in a week or a little later after infection. Stiffness and flexion of the elbows and knees are usually first noted, and this is followed by rigid extension of both limbs and trunk. Rigidity of special sets of muscles, such as those of mastication, of the eyeball, and of respiration, give rise to characteristic signs. The muscles feel swollen and tense, and are tender. Œdema of the eyelids, and later of the face and limbs, is sometimes observed when the disease is fully developed. The general symptoms before the appearance of the muscular signs are a feeling of lassitude, loss of appetite, perhaps nausea, sleeplessness, and slight fever. Later, the temperature may rise to 102°; the pulse is accelerated, and there is often profuse sweating. The tongue becomes dry, red, and furred, and the bowels are constipated; or sometimes diarrhœa is met with.

Diagnosis.—The rigidity of the muscles is the only sign likely to give trouble; if a wound existed, the question of tetanus might arise. Attention to the history of the case and date of the advent of symptoms, examination of the fæces for worms, possibly the removal of a portion of the patient's muscle for examination, would be means of arriving at a correct conclusion.

Prognosis.—Severe cases may prove fatal in four or five weeks, the patient dying of exhaustion, ulceration of the colon, or pneumonia. If the patient recovers, three or four months at least must elapse before the patient is convalescent. The most important point in the prognosis is the number of embryos which have reached the muscles, and this can only be judged of by the amount of stiffness, pain, and swelling which exists.

Treatment.—This consists in clearing the patient's bowels of the parent worms as fully as possible; to this intent, calomel or castor oil in large doses may be given. Glycerine is stated to have been successfully tried as a curative medicine in the United States.

As a preventive to the disease, thorough cooking of the meat is the only requisite; hence, probably, the rarity of the disease in Great Britain.

XIII. GUINEA WORM.

Ætiology.—This is a nematode worm (*Dracunculus medinensis*), which, introduced into the alimentary canal of man in the larval condition, migrates thence into the tissues, and is eventually discharged as a fully-developed worm from the surface of the body.

The parasite is met with in tropical climates (West Coast of Africa, Upper Egypt, Soudan, Nubia, Abyssinia, banks of Ganges, Persia, Arabia Petræa, Mauritius, Curaçoa, Antilles, Bahia).

The female worm, when met with in the tissues, lies coiled in the long axis of the limb, the head towards the distal extremity. The length varies considerably: thus, from one foot to forty inches (Ewart), but extremes from six to twelve feet have been recorded. The largest have been observed in Africa. The body is milk-white in colour, cylindrical, of an uniform diameter of $\frac{1}{10}$ of an inch, ending posteriorly by a pointed recurved tail, anteriorly by a rounded head, with a central opening surrounded by six small eminences. An enormous number of embryos are contained in the body, which, when set free, live and move actively for several days in water.

Having attained full development in the human body, the female worm discharges numerous embryos, and is expelled. The embryos set free in water enter an intermediate host in the form of a small crustacean of the genus Cyclops. After a stay of 35 to 36 days in the intermediate host, the embryos, having attained full larval development, may be conveyed in drinking water with the host into the stomach of man. Sexual maturity is accomplished here; the male worm probably perishes and is discharged with the fæces, and the female worm migrates into the tissues (Fédschenko). In the vast majority of cases the worm escapes from some point in the lower extremities. Thus, in 1,000 cases collected by Tilbury Fox, this occurred in 98 per cent.; and among 300 cases observed by Horton, in 206 instances the feet and ankles formed the seat of emergence. It has been, however, observed to come to the surface at the chin, in the floor of the mouth, at the orbit, nose, head, hand, scrotum, and over the chest wall. The worm has not been met with in internal organs. It is usually single, but has been met with in large numbers.

The period of *incubation* varies perhaps from twelve to fifteen months.

Symptoms.—When the adult worm reaches the subcutaneous tissue, an elongated swelling, somewhat resembling a thrombosed varicose vein, is developed. The worm may still continue to travel, but eventually considerable local pruritus precedes the development of a small vesicle or boil. When this ruptures, a serous fluid—clear if the worm is entire, turbid if embryos are already free—escapes, and the head of the worm appears at the bottom of a small cavity. The worm may now be expelled or removed, or sometimes the opening may heal, and a fresh swelling forms in the neighbourhood. In other cases considerable inflammation and the formation of an abscess have been observed, the inflammation and suppuration being sometimes severe enough to cause serious general symptoms.

Diagnosis.—The character of the swelling and vesicle, together with the locality in which they are met with, are usually sufficient for the formation of a correct opinion. Difficulty may occur in special regions; thus in a case reported by Duncan, in which the scrotum formed the seat of the tumour, orchitis was simulated.

Prognosis.—The disease affords no danger to life except in the cases where severe inflammation gives rise to complications. Severe subsequent contractions have been observed (Horton).

Treatment.—If undisturbed the worm is eventually expelled from the opening by which the embryos have been discharged in an average period of from fifteen to twenty days (Forbes). Manson recommends that this process should be allowed to complete itself spontaneously, unless the conditions are such as to allow the radical extirpation of the entire parasite at one sitting by excision. In regions where the disease is endemic, the operation of removal is often performed by the local barber, the worm being gradually extracted by winding it around a stick for several days. The main precaution necessary is care against rupturing the worm, and setting free the embryos in the subcutaneous tissue: an accident readily occurring, and usually followed by the development of severe inflammation and suppuration. Horton recommends the internal administration of tincture of asafœtida in ʒj to ʒij doses, which, he says, kills the worm and contained embryos, and allows of their safe extraction.

XIV. FILARIA SANGUINIS HOMINIS.

Ætiology.—This is a nematode worm (*Filaria Bancrofti*), which, gaining entrance to the body, migrates into the lymphatic vessels, and produces numerous embryos. The latter pass into the lymph stream and thence into the general circulation. In certain circumstances the embryos give rise to lymphatic obstruction, and the consequent development of lymph scrotum, elephantiasis, chyluria, or chylous serous effusions.

The parasite is met with in China, Japan, India, Mauritius, Bermuda, West Indies, Guiana, Brazil, Queensland, Malay Peninsula, Egypt, Zanzibar, West Coast of Africa, United States, Tahiti, New Caledonia, Madagascar, and South of England (Manson).

The *young worm* is probably ingested with drinking water, bores its way from the stomach, and eventually selects a spot—most commonly in the distal lymphatics of the extremities—as a permanent resting-place. Thence the embryos are discharged, the parent worm living an almost indefinite time; thus, cases are on record in which symptoms persisted twenty-eight, thirty-two, and even fifty years (Roberts; Manson). One pair of worms appears sufficient to produce all the symptoms of the presence of the parasite, but a number may be present; thus, in a case reported by Maitland, three male and five female worms were discovered coiled up in a mass, apparently within a lymphatic channel. Circumstances have not up to the present allowed an exact description of the male worm, which long remained undiscovered. The female is about three and a half inches in length, $\frac{1}{100}$ of an inch in breadth, uniform in calibre. The head is provided with a circular mouth and is destitute of papillæ. A narrow alimentary canal runs from the simple club-like head to within a short distance of the blunt-pointed tail, the remainder of the body being entirely occupied by the reproductive organs packed with numerous embryonic filariæ. The reproductive outlet is close to the head. (Cobbold; Manson).

The *embryo* when born is enclosed in a shell or sheath, within which it may be either coiled up or extended, and which it retains permanently while within the human body. This variation in form at birth is of extreme importance, the coiled embryo of oval shape measuring $\frac{1}{500} \times \frac{1}{760}$ of an inch, while when uncoiled its measurements are $\frac{1}{75} \times \frac{1}{3500}$ of an inch. When uncoiled, it is rounded at the anterior extremity and tapers posteriorly, the retained sheath projecting as an empty space behind the minute worm, which does not completely fill it longitudinally. According to Manson, the coiled embryo, which he regards as an abortion, is responsible from its large size, for the lymphatic obstruction. Lodging in the efferent vessels of the glands, it interferes more or less with the passage of lymph, causing dilatation and varicosity of the vessels, or complete obstruction. The uncoiled embryo, on the other hand, which nearly corresponds in width with the red blood discs, readily traverses the blood-vessels, and it is this which is found in the general circulation (Fig. 59).

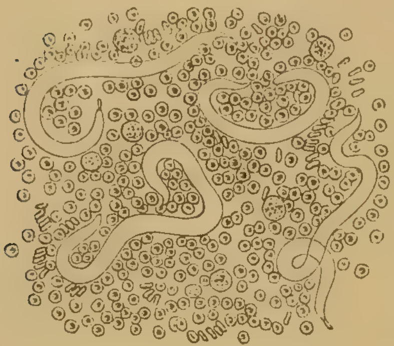

Fig 59.—Embryonic Filariæ in Blood. The red discs are somewhat shrunken, also the sheath of the embryo.

The presence of the embryonic filariæ in the cutaneous blood-vessels undergoes diurnal variations. While the patient is moving and active during the day, none are found, but they appear between six and eight p.m., increase in number till about midnight, and then they gradually decrease, to disappear entirely about eight or nine a.m. S. Mackenzie observed that the retention of a patient in bed during the day resulted in a reversal of the order of appearance of the filariæ.

Manson describes two further distinct forms of filaria embryo, one of which is found during the day only; the other is present both by day and night.

The embryonic filariæ undergo no further development in the human body; but according to Manson's observations, they find an intermediate host in the mosquito. This insect, biting at night, abstracts blood containing embryos, and during the four to six days which it lives prior to depositing its own eggs, the embryos acquire a mouth with three or four nipple-like papillæ, an alimentary canal, rudimentary generative organs, and a peculiar three-lobed caudal appendage. After depositing its eggs upon water the mosquito dies, and its body with the contained filariæ falls into the water also, ready to be ingested by man.

The presence of the adult worms does not appear to cause any of the more serious symptoms of the disease beyond lymphangitis, although its death may occasion the formation of an abscess. Obstruction of the lymph stream by the embryos is followed by dilatation and varicosity of the lymphatics (lymphangiectasis), lymphatic œdema, and sometimes by the development of lymphatic vesicles on the body surface, which may rupture (lymph scrotum, *see* Art. XXVI.); or communications may take place by the rupture of dilated lymphatics with the urinary passages (chyluria) or serous cavities (chylous ascites, chylocele, etc.). Lymphatic œdema may be followed by hypertrophy of the skin and subcutaneous tissue, and the development of elephantiasis of the scrotum or lower extremities (Elephantiasis Arabum, *see* Art. XXVI.).

Symptoms.—The presence of the adult parasite may be evidenced by attacks of lymyphangitis, leaving behind them definite indurated spots and the presence of embryonic filariæ in the blood. Chyluria may be intermittent and yet persist for many years. The urine—most characteristic after a full meal—is milky-white, when passed; setting into a mass, which again liquefies, a creamy material collecting on the surface and a deposit more or less pink, from the admixture of blood, falling to the bottom of the vessel. Similar coagulation occasionally takes place within the bladder, giving rise to painful and difficult micturition. Examination of the urine reveals fat globules in an extremely fine state of division, granular matter, leucocytes, often red blood discs, and filariæ. Albumen may be present in considerable quantity.

The filariæ should be sought either in the clots floating beneath the creamy layer which collects on the surface of the urine after it has stood for some hours, or in the deposit at the bottom of the vessel. It is almost useless to attempt to find filariæ in a drop of newly passed urine (Manson).

Diagnosis.—The presence of embryos in the blood or in chylous secretions is the crucial test of the filarial origin of cases of lymphangitis, lymphangiectasis, elephantiasis, chylous ascites, chylocele, or chyluria; but it must be borne in mind that all these conditions may be met with independent of the parasite, and that in every case due regard must be paid to the diurnal variation in the presence of filarial embryos in the cutaneous blood-vessels, and its occasional complete intermittence. According to Manson also, filariæ may still be discovered in lymph obtained from parts affected by elephantiasis, when none are to be found in the blood stream, their entrance into the general circulation being prevented by complete obstruction of the lymph stream.

Prognosis.—Filarial disease may persist for many years and patients usually die from other causes.

Treatment.—No method of destroying the adult parasite by internal treatment is known; but a promising result has been obtained by Maitland by excising an indurated mass in the course of the lymphatics containing the parasite. Although this operation did not prove radical, yet, as Maitland remarks, it shows that it is possible in some cases to localise the position of the parasites and to remove them. Beyond this, however, it points strongly to the common multiple existence of the worms, an unfavourable element to the success of the treatment. A number of cases of lymphangiectasis have also been treated by excision with no untoward local results, but up to the present no cases of radical cure have been recorded. The treatment of elephantiasis is referred to in Article XXVI. Prophylaxis naturally consists in the thorough boiling and filtering of water used for culinary or drinking purposes.

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XV. BILHARZIA HÆMATOBIA.

Ætiology.—A trematode worm, which gains entrance into the venous circulation, becomes encysted, and produces numerous ova, which are deposited in the tissues and escape from mucous surfaces, giving rise to symptoms the most striking of which is hæmaturia.

The parasite is common in Central and Eastern Africa, Madagascar, Mauritius, and Egypt. In the last-named country the disease produced appears to assume a particularly severe form.

Both the male and female worm are known. The male is white in colour, half an inch long, and about as broad as an oxyuris; the body is covered with microscopic tubercles, except about the anterior extremity and gynephoric canal. The female is darker in colour, especially at the posterior extremity, half as long again as the male, no wider than a fine thread, and covered with fine prickle-shaped projections. Both sexes are provided with two suckers: one at the anterior extremity, corresponding to the buccal opening, and one at the posterior end, serving for fixation, also with an alimentary canal and distinct generative organs.

The ova embedded in the tissues or as voided in the urine are oval in outline, measuring $\frac{1}{400} \times \frac{1}{600}$ of an inch, and provided at the narrow end with a spine $\frac{1}{2000}$ of an inch in length (Fig. 60). The latter is occasionally absent, and is said to be placed laterally in ova embedded in the walls of the large intestine (Manson; Zancarol). The shell is transparent, and within it the embryo may be seen in varying stages of development, the more mature specimens attaining dimensions of $\frac{1}{180}$ to $\frac{1}{160} \times \frac{1}{325}$ of an inch. When placed in water the shell bursts and the ciliated embryo, set free, assumes an elongated form and moves actively. It then either enters an intermediate host, as yet undiscovered, or dies in a few days.

Mode of infection.—The embryos are ingested with drinking water, the vehicle being possibly some small mollusc, such as the Cyclops conveying the embryo of the Guinea worm. The disease is never seen in Egypt amongst those who habitually drink filtered water.

The period of incubation is said by Brock to extend probably over four months. The mature worm is found in the human body, located in smooth-walled cavities in connection with the portal vein and its radicles, its occurrence in the systemic circulation being practically limited to the vesical veins. The female worm is rarely discovered at post-mortem examinations, and never in the portal vein, although the males are often found in this vessel to the number of several dozens, if it be opened previous to the removal of the liver from the body. No evidence exists as to the mode in which the ova are deposited in the tissues or reach the surface of the mucous membranes. They have been found in large quantities in the substance of the kidneys, ureters, bladder, urethra, prostate, seminal vesicles, mesenteric glands, rectum, and in warty vegetations of the skin of the perineum and in the neighbourhood of the external genitals. They have also been found in the liver, lungs (Mackie), and left ventricle of the heart (Griesinger). When old, the shells undergo calcareous degeneration. Empty shells have been discovered in the tissues; but there is no evidence to show that the entire cycle of existence of the parasite can be completed in the human body. The escape of the ova from the mucous surfaces is attended with hæmorrhage, and their presence in the tissues induces a condition of chronic inflammation. This is usually most marked in the bladder, but may extend thence to the ureters and kidneys, giving rise to hydro- or pyelo-nephrosis, and even destruction of the kidney. The basal and posterior part of the bladder are especially, sometimes exclusively, affected; here in the early stages inflamed ecchymosed patches, $\frac{1}{4}$ to $\frac{1}{2}$ an inch in diameter, are seen on the mucous membrane, covered with slimy mucus or yellow exudation. Later, soft granular deposits, encrusted with urinary salts, are found, and warty vegetations develop on the mucous membrane, or chronic irritation may lead to the development of papillomata, the walls of the viscus becoming thickened even to the extent of one inch or more, the tissues containing numerous ova. The ova not infrequently serve as nuclei for the formation of vesical calculi. Similar chronic inflammatory changes may extend to the ureters, these becoming thickened and dilated to the size of the little finger, and the mucous lining is often encrusted with fine gravel. The rectal mucous membrane when affected develops small polypoid growths, and later small round ulcers.

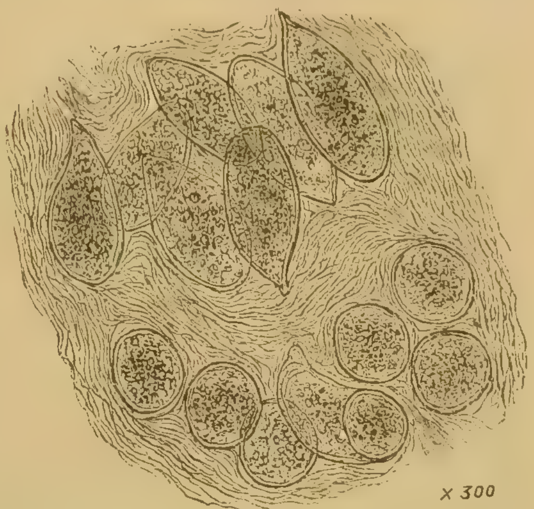

Fig. 60.—Ova of Bilharzia Hæmatobia in Tissue. Longitudinal and transverse sections.

Symptoms.—The disease is common in young males, but has been observed at any age (J. Harley). It is comparatively rare in females in Egypt, perhaps from the fact that they mostly drink in the house, and not in the fields, and hence drink water from the surface of jars in which the ova have settled to the bottom. The most prominent symptom is hæmaturia; but this may be preceded by uneasy sensations in the loins and perineum. The blood is either passed unmixed at the end of micturition, or is more abundant then if the urine is generally tinged. It may be passed in clots, containing large numbers of ova, pain being experienced during their expulsion; or when the blood originates in the ureters, the passage of clots may simulate ordinary renal colic. The hæmaturia may be intermittent, or the blood at times be so sparse as to be discoverable only by tests or microscopic examination. Pain due to subacute cystitis and frequent micturition, accompanied by a burning sensation, perhaps due to the passage of ova, are the signs which commonly bring natives to the surgeon, as they often think little of the hæmaturia. When dependent on the passage of clots or calculi, it closely resembles that observed in ordinary cases of stone. Symptoms of pyelitis or surgical kidney may develop later. The urine is strongly acid, of high specific gravity, contains blood, albumen, crystals of uric acid or oxalate of lime, epithelial cells, tissue *débris*, and numerous ova in the early stages. Later, the acidity and high specific gravity are less marked and tissue *débris* is more abundant. Ova are frequently to be found when the patient considers himself quite free from the disease. General symptoms are slight until the loss of blood is sufficient to tell on the system, when extreme anæmia, or pyrexia, dependent on inflammatory changes, may be met with. It is, however, remarkable how little the majority of the patients suffer from the loss of blood. When the rectum is affected, dysenteric symptoms occur.

Diagnosis.—This is to be made by inquiries as to the district in which the disease was incurred, and by detection of the ova in the urine or fæces. It may be necessary repeatedly to examine the excretions for the ova, as they are often sparse in number. The drops of blood which follow the flow of urine should be examined separately, or, if this be impracticable, the deposit at the bottom of the vessel should be examined after the contents have stood for some time.

Prognosis.—The disease has a tendency to wear itself out, if constant re-infection be not incurred, and is only immediately dangerous from the loss of blood. It may, however, continue for years, and the patient may suffer from secondary renal or vesical disease at a very late date. Death most commonly results from asthenia, the patient becoming worn out by his bladder trouble, occasionally aggravated by rectal tenesmus, or dysenteric diarrhœa may sometimes prove fatal.

Treatment.—No specific treatment is known. The use of iron and arsenic is indicated to combat the anæmia, while the bladder symptoms and tendency to the formation of calculus are to be treated with the ordinary remedies for those conditions from other causes. Violent exercise must be forbidden, and complete rest enjoined during attacks of hæmaturia. Liquid extract of male fern in doses of 20 to 25 minims thrice daily does temporary good by relieving the burning sensation attendant on micturition and the constant desire to pass water; moreover, during its exhibition, the ova in the urine are temporarily diminished in number. Methylene blue given in 5-grain doses as a wafer, repeated every few days, also occasionally gives marked relief, when pain is severe. Morphia may be needed to allay pain. In very severe cases preprostatic puncture of the urethra, followed by daily irrigation of the bladder, has proved successful as a palliative measure (Mackie).

In districts infected with the parasite care must be taken either to sterilise drinking water thoroughly by boiling, or to ensure efficient filtration. Bathing should be forbidden or discouraged, special warning being given of the danger incurred in swallowing water.

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XVI. MADURA FOOT, MYCETOMA.

Ætiology.—This is a chronic disease of the foot, and less commonly of the hands, due to the entrance of a vegetable organism, with consequent development of granulation tissue and suppuration, all tissues of the limb being invaded indifferently.

Madura foot is met with throughout India, but most commonly in the district of Madras, from which it has received its name. A case has been observed in Italy (Bassini), but otherwise it has not been seen in Europe. It attacks especially adult male natives, particularly those who work barefoot in the fields.

Originally confounded with tubercle, Carter, after careful investigation of the disease, ascribed its origin to the entrance of a vegetable parasite, and the discovery and investigation of the actinomyces led him to infer the presence of a similar organism in madura foot. Striking differences, however, exist between the two diseases, madura foot affecting external surfaces alone, while actinomycosis is common in the alimentary and respiratory tracts, and the organs and tissues in close relation with them ; and again, actinomycosis occasionally spreads by embolic metastasis to distant organs : an occurrence not observed in madura foot, and one which is, perhaps, the more striking, since the hyphæ of the organism have been observed within the blood-vessels by Boyce. Recent observers have studied the organism in preserved specimens in the light of present knowledge, but cultivation and inoculation experiments are still needed to clear up its actual nature.

Kanthack regards the fish-roe bodies as a form of actinomyces, and the black masses in their most perfect shape as also of this nature, the relation between the two forms being established by the occasional occurrence of a degenerated form of the yellow variety, not wholly unlike the black masses. Boyce and Surveyor, on the other hand, regard the yellow masses as consisting largely of caseous material, with the remains of a lowly-organised fungus, presenting in very many instances some of the characteristics of the actinomyces; while they describe the black particles as a curious metamorphosis of a large branching septate fungus. They suggest, therefore, that the two varieties are distinct, but consider both fungi pathogenic.

The division of the disease into two forms has originated solely from the different appearance of the contents of the abscess cavities; otherwise, the pathological process is practically identical in each.

Mode of infection.—Probably the development of the disease is always preceded by a slight injury or abrasion, but owing to the chronic course of the disease, this is often missed in the history.

Morbid anatomy.—The disease may be limited to the connective tissue planes, but in old cases more commonly invades all the tissues of the limb, inducing the formation of abundant granulation tissue, numerous abscess cavities, and fistulæ. The abscess cavities, lined with firm cicatricial tissue, may be isolated, or intercommunicate freely by tortuous fistulæ, which reach the surface. They contain either a dark granular substance, much resembling coarse gunpowder (black variety), or small aggregated particles resembling fish-roe (pale or ochroid variety). The fatty tissue of the limb is especially prone to invasion, the muscles less so. The short bones are usually the most markedly affected: they are softened and excavated, the cancellar walls are atrophied and opened out and the spaces filled with a greasy pulp; or smooth connective-tissue-lined cavities, identical with those seen in the soft tissues, may be developed. More rarely the affection extends to the long bones.

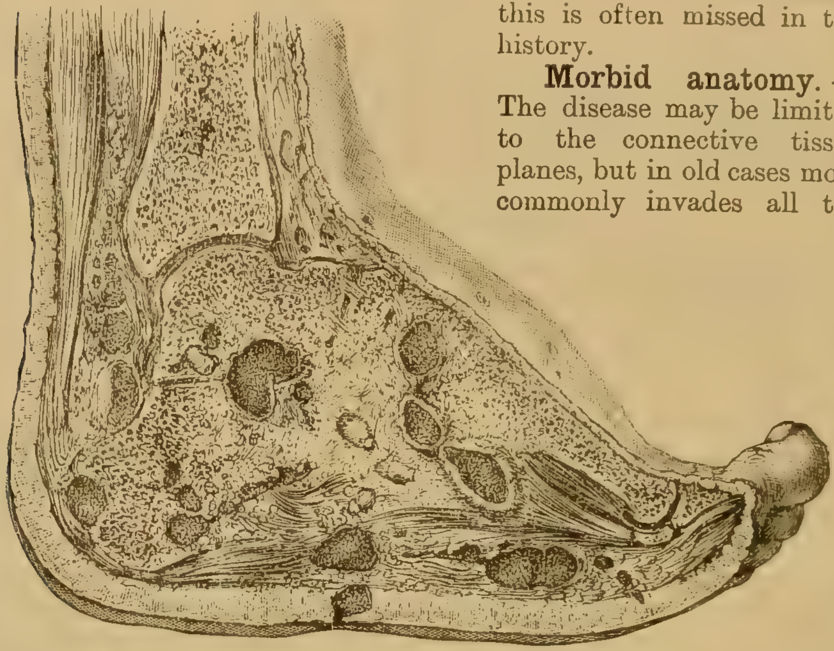

Fig. 61.—Sagittal Section of a Foot affected with Mycetoma. (St. Thomas's Hospital Museum.)

Symptoms.—The early development of the process is very slow. A small localised induration often between the toes, or upon some part of the dorsum or sole of the foot, is the first sign; the skin becomes discoloured and the formation of a papule or nodule followed by suppuration, ensues. There is often little pain, and the first pus offers nothing characteristic in appearance; later, the granules, or fish-roe particles, form a constant constituent. In some cases the discharge of black granules is foreshadowed by a black-mottled discoloration of the skin prior to the formation of the fistula. The formation of the first fistula is followed by the appearance of others, developed in a similar manner. The foot may enlarge to two, three, or four times its natural size, becoming riddled with fistulæ, "surrounded by raised margins, or opening on the summits of elevated tuberculations"; at times the openings are crater-like, or surrounded by mammillated projections, beneath which frequently minute yellow or black particles can be seen. In the black variety the orifices are often more or less completely plugged by irregular little aggregations of black substance, or granules, which can be picked out. The use of the foot in the earlier stages induces flattening of the arch and the toes become hyper-extended. (*See* Fig. 61.) The disease may extend upwards to the leg, or even higher. The progress is always chronic, but varies in rapidity in individuals; it is often one or two years before the foot is useless, and the disease may last for many years—even twenty to thirty. As in actinomycosis, infection of the lymphatic glands does not occur.

The local process is unattended by constitutional symptoms, but prolonged suppuration may eventually be followed by exhaustion or visceral disease.

Diagnosis.—In districts where the affection is endemic, attention to the history and the characters of the discharge should suffice to determine the nature of the case.

Prognosis.—Spontaneous recovery is unknown, but the disease offers no dangers to life beyond those attendant on prolonged suppuration.

Treatment.—Radical excision of the deposit appears to be equally successful in this disease as in actinomycosis. When the disease is too extensive for local treatment, amputation is the only resource; and great care must then be taken to ensure the complete avoidance of affected tissue, as recurrences have been observed in cicatrices after removal of a limb. The use of the sharp spoon may be indicated in some cases, and whenever local operations are performed a strong solution of carbolic acid should be applied, to obviate as far as possible the possibility of re-infection of the fresh surface.

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XVI. TUBERCULOSIS.

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Definition.—Tuberculosis may be defined as an infective disease, in which the tissue changes are due to the action of a specific micro-organism—the tubercle bacillus.

It may further be said that the tissue changes are in their general characteristics those of an inflammation of a low and usually chronic type, and that they are marked by the production of minute structures, called "tubercles." One distinctive feature of the inflammatory changes depending upon tuberculous infection is the appearance of caseation.

Examples of tuberculosis.—Tubercle in the lung produces phthisis; tubercle in the coverings of the brain, tuberculous meningitis; tubercle in the peritoneum, tuberculous peritonitis. Tuberculous ulcers are common in the mucous membrane of the bowel and bladder. The common skin affection, lupus, is due to tuberculosis of the skin.

That very widespread affection of the lymphatic glands, usually known as scrofulous or strumous disease of glands, is a tuberculous disorder. Tuberculous disease is common in the epididymis and testis. Tubercle often develops in bones and in synovial membranes, producing "white swellings" of a joint, caries of bone as illustrated by Pott's disease of the spine, and such affections as tuberculous teno-synovitis. There is, indeed, hardly an organ or a tissue in the body which may not be invaded by tuberculosis.

In certain organs it is comparatively uncommon; the most conspicuous of these are the ovary, the pancreas, the thyroid body, and the muscles.

The general features of the tuberculous process.—Speaking generally, the tuberculous process is slow and insidious. It is attended with the phenomena of inflammation, but these are often faint and subdued. The lesion may, and usually does, remain local. It has, however, a great tendency to extend locally, and slowly to infect the adjacent tissues. In Fig. 62 is shown an example of the deliberate and extensive spreading of a comparatively slight tuberculous affection.

It is very apt to be ill defined in its limits. Septic inflammation of a moderate degree is, on the contrary, usually precise in its limits. The damaged area is surrounded by a barrier, which tends to isolate it. Such a barrier is uncommon in tuberculous disease. The affected district fades off gradually into the healthy tissue, and it is not easy to say where one begins and the other ends.

In tuberculosis the inflammatory process is attended by a considerable exudation of a low type and by the formation of tissue of a flimsy and unstable character. For example, in tuberculous disease of such a joint as the knee, the tissues of the articulation are found to be infiltrated with a frail gelatinous tissue. To this condition the name of "pulpy or gelatinous degeneration" was at one time given. In such a vigorous inflammation as may follow injury of a joint, this substance would be represented by a healthy granulation tissue, by means of which the damage in the articulation would be isolated and the lesion repaired. In the tuberculous trouble the products of the inflammatory process are neither sound enough to form substantial connective tissue, nor simple enough to be absorbed, nor evil enough to break down into pus, and the compromise is the feeble, unstable, ill-formed, uncertain substance which is so marked a feature in the diseased joint.

One event in the tuberculous process is almost characteristic, and that is the appearance of *caseation.* Caseation is the outcome of a process of degeneration; the caseous material is wholly dead and is incapable of undergoing any but a chemical or physical change. It is absurd to speak of a caseous "deposit"; caseous matter is not deposited. It is the result of a decay of the materials concerned in the tuberculous process. It is equally absurd to speak of a caseous mass as suppurating or breaking down into pus. The cheesy nodule cannot form pus. It is only to be compared to a foreign body, to a slough, or to a sequestrum. It is incapable of active change. If suppuration take place, it is in the still living tissues which surround the caseous substance.

The common sequence of events in tuberculosis is to be well seen in tuberculosis of lymphatic glands. The part is invaded by the

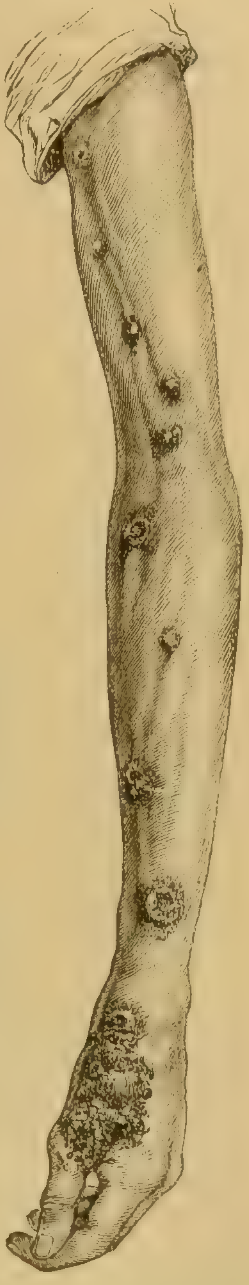
Fig. 62.—Tuberculous Lymphangitis of Arm, starting from Tuberculous Ulcer of Hand. ("Études sur la Tuberculose." Tome iii.)

bacillus. The tissues exhibit a characteristic inflammatory reaction in consequence. Caseation results; then may follow suppuration. The object of the pus formation is evidently to rid the body of the dead or necrosed matter represented by the caseous substance. The pus finds a vent and the caseous matter is discharged, and then the sinus heals up and the trouble is—so far as that part is concerned—at an end. It is desirable to note that caseation precedes suppuration. The caseous material met with in tuberculosis is *not* inspissated pus.

The tubercle bacillus is capable of inducing suppuration (pages 25 and 35). In large tuberculous abscesses it has been the only micro-organism found, and there has been an absence of the usual pyogenic cocci.

Repair after the apparent healing of a tuberculous lesion is usually feeble and often insecure. The disease is over, the once-infected area is no longer infected, all progress in the way of destruction has ceased, but the restoration of parts is of the flimsiest. The healing in tuberculosis contrasts forcibly with the firm, sound, solid cicatrix which follows after injury, and with the extensive and complete repair which commonly follows upon the removal of parts by operation. At the site of the tuberculous lesion there is no more done in the way of regeneration than is absolutely necessary. It thus happens that trouble is apt to recur at the site of an apparently healed tuberculous disease. A child has had hip disease, with abscesses. The sinuses have all closed, and the child is about again. Years go by, and then, after some little disturbance in health, the hip gives trouble again, sinuses re-open and discharge pus once more, and even after healing again there may be a second "breaking down."

Of this nature is the so-called "*residual abscess*"—the abscess which crops up again after months or years of apparent cure. It is called residual, because it probably has its starting-point in the residuum or remains of the original trouble. An example of the residual abscess is afforded by the abscess about a disabled hip, as already mentioned. It may also be illustrated by such a case as this: A child of twelve had caries of the spine and a psoas abscess. The abscess formed an opening in the thigh, and after discharging for many months, closed. The child was apparently sound. She grew up a vigorous and active woman, and ultimately married. Three months after the birth of her first child, and when she was twenty-five years of age, the psoas abscess appeared again in the same place, opened and discharged, and led to the patient's death.

The history of tuberculous disease is associated with frequent relapses and with many disappointments.

From a well-localised tuberculous focus the whole body may become infected, the tuberculous virus may be carried to almost every part of the organism, and the condition of acute general tuberculosis then results. The dissemination of the trouble is exactly comparable with the diffusion of septic matter in pyæmia, and the outbreaks of tubercle in distant parts are precisely to be compared with the secondary or metastatic abscesses in pyæmia.

It is well, however, to point out that general tuberculosis occurring as first described is rare—and indeed, considering the extraordinary frequency of local tuberculosis, it may be said to be exceedingly rare.

Another feature in tuberculosis is the trifling nature of the irritant or lesion whereby the mischief may be in the first place localised. Given an access of the tubercle bacillus to the body, and provided that that body is a suitable soil for the growth of the bacterium, it requires but a very trifling lesion to locate the disease. Thus hip disease and caries of the spine may follow upon quite slight injuries, tuberculous glands may be set up by some insignificant irritation of the surface, and phthisis may follow upon what may appear to be but a slight disturbance of the lung.

Finally, it is to be observed how readily tuberculous troubles will heal under suitable treatment, one essential in that treatment being the free removal of the tuberculous material. A tuberculous ulcer which has existed for months or years may heal up in a week or so after a vigorous application of Volkmann's spoon.

To make a summary of the matter, the tuberculous process has the following **general characteristics:** (1) It is slow and insidious. (2) It is attended with the phenomena of inflammation. (3) It has a tendency to extend locally, and to be somewhat diffuse in its outline and indistinct in its limits. (4) The exudation material produced is ill-conditioned. (5) Caseation is very common, and suppuration is very usual, although by no means inevitable. (6) Repair is often feeble and insecure. (7) A well-localised tuberculous disease may be the starting-point of a general acute disseminated tuberculosis. (8) The tuberculous process may be easily induced, provided that the soil is suitable and ready, and that the bacillus has access to the part. (9) Tuberculous affections respond readily to treatment when a free eradication of the diseased tissues is possible.

The tubercle.—The term "tubercle" was originally applied to a certain naked-eye appearance—to minute spots of diseased tissue which were conspicuous as nodules or tubercles. The association of a caseous change with these little bodies was early appreciated.

A better limitation of the word was arrived at when it set forth that some of these nodules were grey and clear, while others were yellow and opaque. Thus arose a division of tubercle into the *grey* and *yellow* varieties. The yellow, or so-called crude, tubercles were for the most part caseous masses, or, at least, masses advanced in that degeneration. With regard to the grey variety, it was found that these, when met with in the lung, were often made up solely of little masses of alveolar epithelium, the results of a lobular catarrh. All such nodules, therefore, had to be eliminated, and the term was then restricted to such grey semi-transparent bodies as were not merely masses of catarrhal exudation, and which, while retaining the size of a millet-seed, were hard and firm. The name of *miliary tubercle* was given to them, and in the disease known as

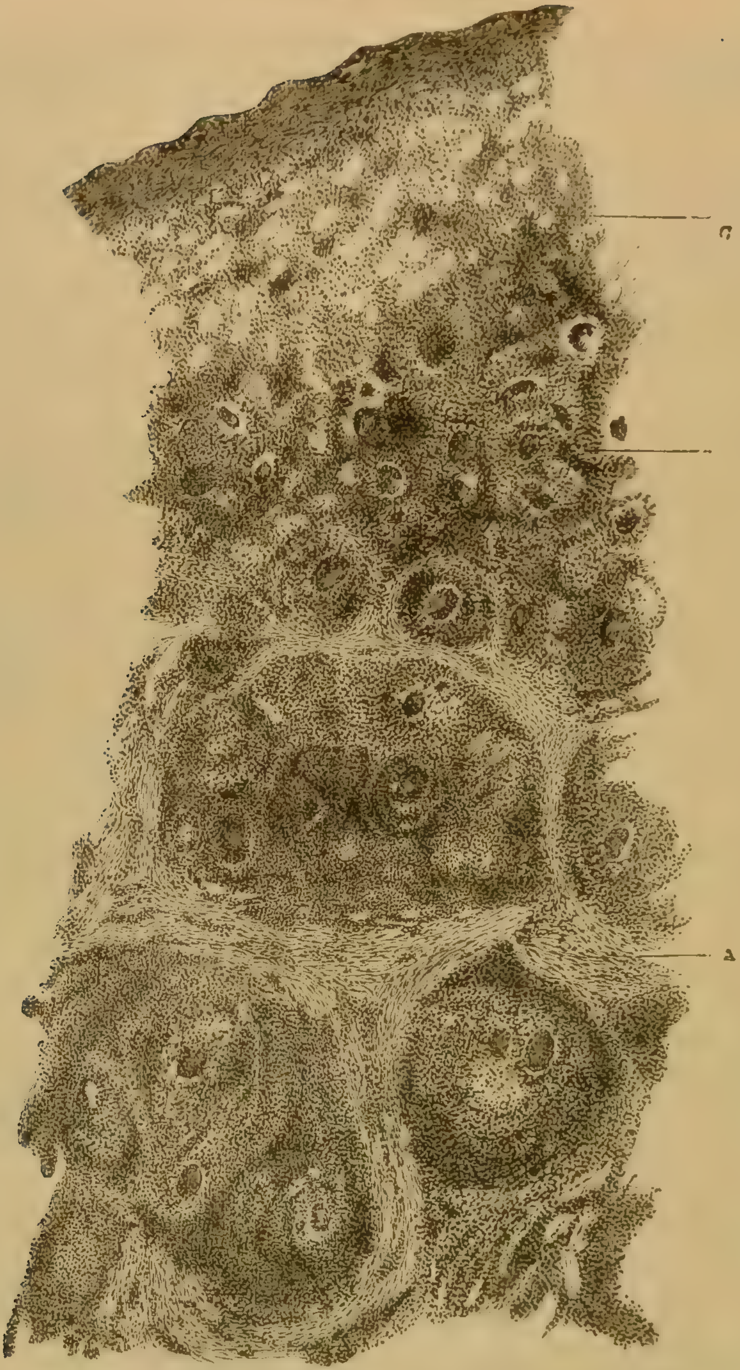

Fig. 63.—Tuberculous Disease of the Synovial Membrane of the Hip-joint.
A, Advanced stage; B, less advanced stage; C, earliest stage.

acute miliary tuberculosis they were considered to be met with in perfection. In time, however, more subtle tissue changes were noted, which were regarded as tuberculous, but which were not associated with the appearance of these distinct grey masses. In the place of such masses certain microscopic nodules alone were detected, which were found to possess a fairly simple structure; and as it was observed that certain of the grey miliary tubercles, visible to the naked eye, were made up of a collection of these microscopic nodules, the latter were distinguished by the term *submiliary tubercle.* It is to these microscopic nodules only that the term "tubercle" is, in its strictest sense, now applied.

The structure of the tubercle.—The tubercle as it is exposed under a microscopic slide is too small to be seen by the naked eye. The groups and lines into which these bodies very usually arrange themselves are, however, visible enough.

In a general view of a tuberculous tissue under the microscope, it will be seen that the tubercles are scattered fairly evenly over the field, and that they tend to form well-defined rounded groups or clusters which are more or less emphatically circumscribed (Fig. 63).

The changes visible may be said in general terms to resemble those of inflammation, rendered remarkable by the presence of the minute formations or tubercles.

The individual tubercle is rounded, is made up of cells, is clearly differentiated by staining, is void of blood-vessels, and is prone to exhibit a rapid caseous degeneration. It will be noticed that as the centre of the tubercular district is approached, the tubercles and the clusters into which they are gathered become usually larger and larger (Fig. 63, A). On the outskirts of the infected area, on the other hand, the tubercles are small, and their disposition to form into groups is hardly noticeable (Fig. 63, B); while at the extreme periphery no distinct tubercles are to be seen, although their presence is suggested by certain casual groups of cells (Fig. 63, C).

The districts marked A, B, C in Fig. 63 serve to display the three grades of tubercle, and to show its progress and growth from its earliest appearance to its most complete development. The three perfect groups or clusters of tubercles shown in the most advanced area (Fig. 63, A) would be apparent to the naked eye as actual grey nodules, or as "miliary tubercles."

The tubercle appears to begin by a proliferation of the fixed cells of the part, these being represented by the endothelial cells of the blood-vessels or lymphatics, and by the cells of the connective tissue.

There is by this means produced a little ill-defined mass of highly nucleated cells, which are usually spoken of as epithelioid, and which are very often located around or near to a blood-vessel (Fig. 64). With these conspicuous bodies are associated a greater number of ordinary leucocytes.

So far there is nothing distinctive about the morbid change. At a very early period, however, the proliferation of the epithelioid cells proceeds in such a manner as to produce the appearance known as giant cells. The individual features of these remarkable bodies are at first indistinct. Their earliest manifestations are shown in Fig. 64.

The introduction of the giant cell element gives to the tissue change a distinctive character, and Fig. 64 shows the earlier tuberculous phenomena, such as may nearly always be seen at the periphery of an obviously tubercular focus.

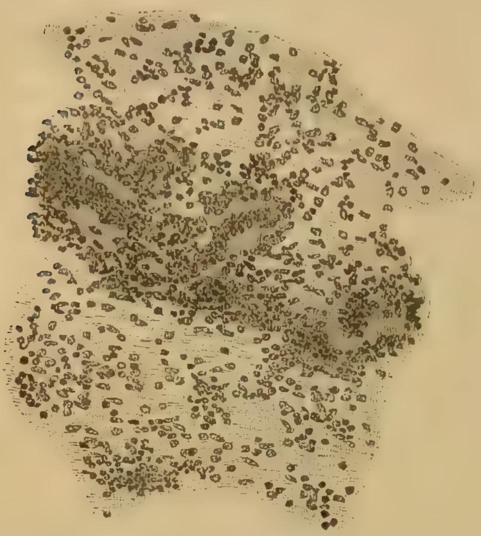

Fig. 64.—One of the Cell Clusters from Area c Fig. 63.

Beyond this stage, in acute cases, the process of tuberculosis may not extend, and after no more elaborate development than has been here shown, the affected district may pass on into caseation.

In the more usual or chronic progress of tubercle formation, on the other hand, a further development takes place.

The giant cell becomes more conspicuous and more clearly defined. As a rule, with each tubercular focus but one giant cell is associated; or if more than one be present, a particular cell is predominant. It is disposed in the centre of the cell colony, and what appears to be a reticulum may commonly be seen to extend from the mass forming the giant cell into the midst of the surrounding cells (Fig. 65). The so-called giant cell is—when compared with the structures in its vicinity—of enormous size. Its nuclei are composed of epithelioid cells, which are often arranged in a curved row about the periphery of the mass, or are huddled together in a semi-lunar cluster at one end of it (Fig. 65). On the other hand, the nuclei within the cell may be arranged in no order, and appear simply as a compressed but well circumscribed crowd.

In the cell bodies which surround the giant cell it may be apparent that the epithelioid cells are more numerous near the centre, and the leucocytes more numerous about the periphery. The whole collection forms a more distinctly rounded focus, and under a low power of the microscope stands out more clearly as a nodule.

Beyond this stage, again, the process may not extend; but as soon as the degree of the development just described has been reached, disintegration may set in. In very many of the commoner examples of surgical tuberculosis, the tubercle is a no more elaborate structure than this; and Fig. 65 may serve to illustrate the form which is perhaps most common in tubercular gland disease.

In the more chronic cases a still further elaboration of structure is, however, to be met with. This takes the form of the complete tubercle: a structure sufficiently large and well defined to be visible under a quite low magnifying power. The final stage is shown in A, Fig. 63, and in detail in Fig. 66.

Here is a well-marked rounded mass, well differentiated from the surrounding tissues. It is made up of cells, and is void of blood-vessels. The cells are very evidently of three kinds, and exhibit some method in their arrangement. In the centre is the giant cell, with its very numerous oval nuclei. From its margin proceed radiating processes, which join in an ill-marked reticulum about which the tubercle appears to be built up.

In the second place, proceeding towards the periphery, is a cohort of epithelioid cells, which surround the giant cell, and are best seen in its immediate vicinity. They form an ill-marked zone in the round area of the tubercle.

Thirdly, about the periphery of the district is a host of round cells or simple leucocytes, which crowd the outskirts of the tubercle and insinuate themselves in diminishing numbers towards the centre.

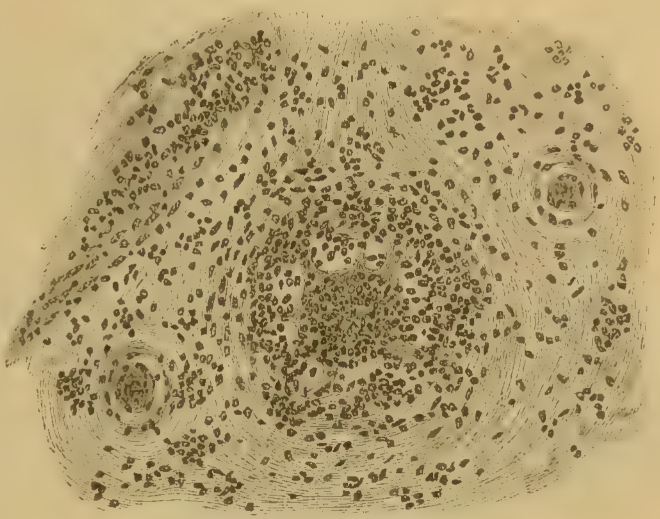

Fig. 65.—Tubercle from Area B, Fig. 63.

It will often be observed that the cells are not precisely arranged in definite zones, although their inclination towards certain parts of the tubercle is very apparent. It will be noticed also that the reticulum of the neoplasm has a tendency to concentric arrangement at the periphery of the mass.

Lastly, it should be borne in mind that the giant cell may be absent or obscured, or be found near the outskirts of the collection; while now and then two or more smaller giant cells stand in the place of the central figure. The tubercle is often obscured by inflammatory changes, which disturb the surrounding tissues and invade the precincts of the little growth itself.

Unlimited discussion, which need not here be considered, has taken place as to the exact nature of the giant cell. One fact would appear to be established; and that is, that giant cells do not all originate in the same way.

Certain of them are obviously but coagula of fibrin and lymph which have engaged the products of a rapidly proliferating endothelium in a blood-vessel or lymph passage. Others evidently result from changes in the fixed cells of the tissue.

Giant cells are not so characteristic of tubercle as is the bacillus, since they have been found incidentally in other lesions, such as chronically inflamed connective tissue.

The bacilli are most common and most numerous in the giant cells. They may not be readily detected in the earliest stages of the tubercle formation, but they are fairly well seen at a later period, when the giant cells are in evidence, and are often most distinct when the structural details of the part have been obliterated by caseation.

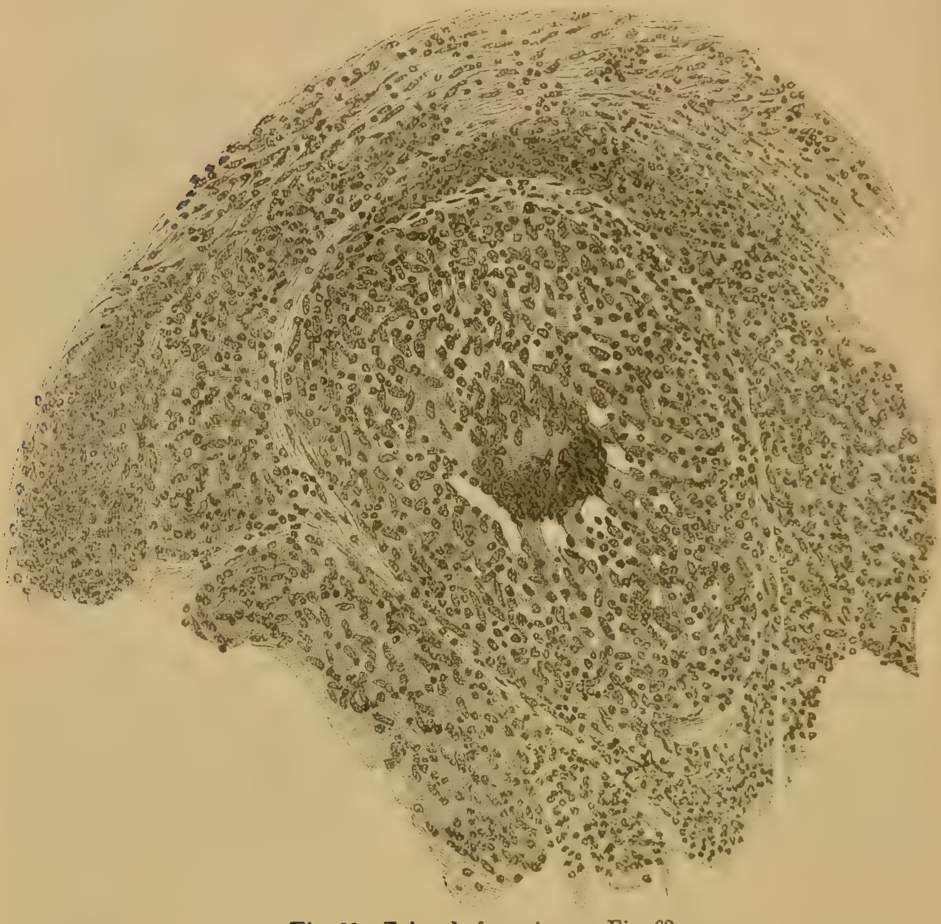

Fig. 66.—Tubercle from Area A, Fig. 63.

It only remains to be said that the tuberculous process, when once established, is liable to indefinite extension. It usually spreads along the lymphatic channels, and follows lymphatic vessels. In gland disease, tubercles may be readily found in the lymph canals which connect one infected gland with another; and it may be possible to prove that the disease has been brought to the gland by lymphatics which have their origin in a tubercular lesion of the periphery

In tuberculosis of the lung and of other organs, the part played by the lymph spaces in the advancement of the disease is often to be made manifest.

The tuberculous process has also a tendency to spread over any surface upon which the infective process may once have obtained a hold. Thus, it may extend indefinitely over the pleura or peritoneum, or proceed without check along the lumen of the bronchi, the vas deferens, or the ureter.

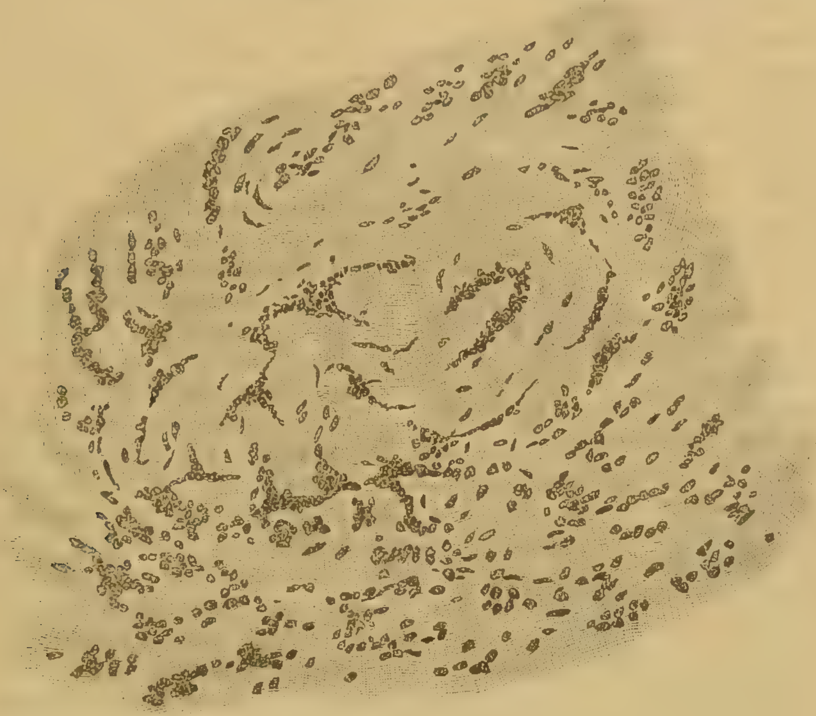

Fig. 67.—Margin of a Caseous Patch from a Case of Tuberculosis of a Synovial Membrane.

It has been noticed that the process of extension is apt to be arrested by any membrane which may intervene, unless the membrane itself be destroyed by necrosis. Thus, the tubercular mischief in a gland would appear to be arrested by the capsule; and tuberculosis of the lung does not commonly extend to the pleura.

The end of the tubercle. (1) *Caseation.*—Caseation usually brings the process of tubercle formation to a close, and is, indeed, a distinctive feature of tuberculosis. It is true that a caseous degeneration has been noticed in certain syphilitic new formations and in some other isolated conditions, but the instances are all exceptional.

The process is one of necrosis and fatty degeneration. The caseous patch is a sequestrum; an area of absolutely dead tissue, which is capable of no other than chemical changes (Fig. 67).

Caseation begins in that part of the infected district which is the most remote from the blood supply. The drier the part, the more characteristic is the degenerative change. Caseation is rarely observed in such a tissue as an œdematous lung, or in a part which has remained long engorged with blood. Where bacilli are numerous, and where the infiltration by leucocytes is considerable, caseation is rapid and well marked. When the opposite conditions obtain, the degeneration is less quickly moving. In the most acute cases there is no time for this leisurely form of decay, and the diseased part may assume at once the phenomena of necrosis or sloughing, or of acute softening.

Caseous matter—as it appears to the naked eye—takes the form of deposits or patches of a bright pale yellow or buff colour. These patches are homogeneous, well circumscribed, and firm, and as displayed in a section are very aptly to be compared to cheese. In the lymphatic glands the caseous districts stand out with remarkable clearness from the surrounding tissues: not as oases in a desert, but as minute deserts in a vast oasis.

Caseous matter may remain for a long time unaltered. It may accumulate, and so produce large masses, as is sometimes seen in the brain, and in a minor degree in much enlarged lymphatic glands.

It may in due course undergo the purely physical and chemical process of liquefaction, and appear when exposed as a collection of creamy matter. Such collections have been inaccurately described as purulent, and the degenerated tissue has been spoken of as suppurating. Not infrequently the centre of a caseous area is found to have become softened, while the periphery remains firm.

Cheesy foci may act as foreign bodies, and may encourage suppuration in the living tissues which surround them.

In certain conditions it would appear that caseous products are capable of being to a certain extent absorbed; but there is no evidence to show that this method of elimination is ever extensively in progress, or that it can be regarded as a common means of ridding the body of this degenerate material.

A caseous patch may become the seat of some degree of calcareous infiltration and so be rendered still more inert.

Suppuration may, however, be aroused around a calcareous nodule which has been for many years in a state of perfect peace, and from the resulting abscess considerable calcareous fragments may be discharged.

Finally, caseous material may become in due time encapsuled. The capsule in such a case is formed of fibrous tissue developed from the more or less uncontaminated structures which surround the tuberculous area. This process of capsule formation is distinctly of a reparative character. It allows the caseous mass to be enclosed, to be shut off from the rest of the body, and indeed to be isolated, as one would isolate an infected person.

(2) *Fibrous transformation.*—It has been already said that caseation represents the usual mode of ending of the tubercular process.

In comparatively rare instances, however, a transformation into fibrous tissue obliterates the phenomena of disease, and leads to a condition which is one of cure, although not precisely one of recovery. The *status quo ante* is never established. The fibrous change is seen in the most chronic and most gently progressing cases.

In the place of elaborate tubercle formation there is simply left an ill-defined mass of fibrous tissue—a cicatrix, in fact. The process differs in no way from that observed in inflammatory conditions of a less destructive kind. A scar is produced and that scar, as a rule, remains sound.

In instances in which this rare change is noted the tubercle is eradicated, the disease is healed, and the tubercle bacillus is no longer to be found.

The bacillus.—The bacillus of tubercle has been already described on page 34 of this volume. A few points need only here be alluded to.

The tubercle bacillus is very persistent, and has great powers of resistance to destructive agencies. Cornil and Babes show that it will live for seventy days at ordinary temperatures in sterilised Seine water. It retains its infective power after very long exposure, after having been dried and kept for months, and after having been placed in a putrid solution. Heat destroys it most satisfactorily, but germicide solutions to be destructive have to be strong, and carbolic acid would appear to be more potent in this connection than corrosive sublimate. It is very resistant to the action of the digestive fluids. It grows best at about the temperature of the body. It needs water for its development. It is an obligate parasite, and can only flourish on the living body of higher organisms.

The bacillus is introduced into the body in many ways. It is inhaled with the air. It is swallowed with the food. It finds an entrance through the mucous membrane of the mouth. It may be conveyed by an inoculation through a breach in the skin. Its presence has been demonstrated in the air and dust of rooms occupied by phthisical subjects, in the milk of tuberculous cows, and in the discharges from tuberculous patients.

It is met with in all forms of tuberculous disease, although its distribution is very irregular. The number of bacilli found in the tissue is not always proportionate to the severity of the local changes. The parasite is most particularly to be met with in advancing lesions. It would appear to be freely transported by the blood. It has a peculiar affinity for certain parts, such as the serous membranes, the lung, the spleen, the kidney, the intestine, and the tissue of bone.

The transmission of tubercle.—The demonstration of a bacillus as an essential feature in the entourage of a tubercle would of necessity involve a question as to the possible transmission of tuberculosis from one individual to another.

It will be convenient to consider the subject of transmission under the following heads:—(1) By inoculation; (2) by respiration; (3) by cohabitation; (4) by feeding.

The evidences of transmission may be very briefly arranged into such as are furnished by experiments on animals and such as are derived from clinical experience.

(a) Experiments upon animals. (1) *Transmission by inoculation.*—The fact that the disease could be communicated by inoculation was known for some time before the discovery of the bacillus.

In the production of tuberculosis in animals by inoculation, the materials used in the experiments have been cultivated bacteria, or the products of tuberculous disease, such as sputum and the degenerate tissue of disordered glands, lungs, or joints.

The infecting material produces at first only a local tuberculosis, which may be followed by a more general dissemination of the disease. That is to say, the first eruption of tubercle takes place at the point of inoculation, whether it be the skin, the peritoneum, the interior of a joint, or the anterior chamber of the eye.

If the infective matter be introduced, with suitable precautions, into the blood stream, a more or less general dissemination of tubercle results.

In the spreading of the tuberculous process from the original seat of inoculation, it is very noticeable that the lymphatics play a conspicuous part in the propagation or extension of the disease. (*Compare* Fig. 62.)

In the general dissemination of the disease throughout the body, the bacillus appears to show an undoubted preference for the tissue of the lung.

The disposition of the blood stream will also serve to explain the frequency with which the lung is found to be involved in cases where the infection of the body is becoming general.

The capacity or virulence of the material of infection shows, as may be imagined, pronounced differences. Inoculations with material from caseous glands will not produce so rapidly fatal a tuberculosis as will inoculations with the tissue of miliary tubercle. A fragment of caseous bone from the finger of a tuberculous child introduced into the peritoneal cavity of a guineapig was followed by an outbreak of tubercles in the mesentery and mesenteric glands, and later in the kidney and lungs. The scabs of eczema from the same child, introduced in the same way, produced no result in the animal experimented upon.

(2) *Transmission by respiration.*—Animals made to inspire tuberculous matter introduced into the atmosphere of a chamber by means of a spray have developed tubercles in the air-passages and lungs. These experiments are much modified in their results by the manner of their performance. If the infecting material used be fresh sputum from a phthisical patient, very freely divided by being introduced into a spray apparatus, the results will be as above described; but if such sputum be dried and converted into dust, and then made to circulate in the air of a closed chamber, the animals contained in that chamber will not become tuberculous.

(3) *Transmission by cohabitation.*—This has been well demonstrated in the case of rabbits and guineapigs. Animals affected with tuberculosis have been put to live with healthy animals, and have been surrounded by healthy conditions. In process of time, the sound rabbits and guineapigs have developed tuberculous disease and have died.

(4) *Transmission by feeding.*—Certain animals fed upon tuberculous material have developed tubercle in the intestine and mesenteric glands. In some of the instances, tuberculous matter, such as sputum or disintegrated tissue containing tubercle, has been mixed with the animal's usual food. In other instances the food substance has been composed of the flesh of tuberculous animals or the milk of cows suffering from tuberculosis.

Speaking in general terms of these experiments on animals, it is to be noted that the susceptibility of different animals to the tuberculous virus differs very considerably. Rabbits, guineapigs, apes, and ruminants are peculiarly prone to tuberculosis, and readily develop the manifestations of the disease after inoculation. The carnivora, on the other hand, may be said to exhibit but a comparatively slight susceptibility to the disease.

It is needless to say that the results of inoculation experiments are much influenced by the concentration of the virus, its amount, and the site and method of its introduction.

(**b**) **Clinical experience.**—A study of the transmission of tuberculosis in the human subject, as gained by clinical experience, soon makes it evident that data derived from laboratory researches and experiments upon animals must be accepted with considerable modification when it is sought to apply them to the disease in man. The animal man, viewed as a *corpus vile*, does not respond very readily to the tuberculous virus when it is introduced into the body by an unintended experiment.

(1) *Transmission by inoculation.*—Considering the wide distribution of tuberculous disease, and the fact that in many forms of it a discharge exists in which tubercle bacilli are more or less numerous, it is remarkable how very rare are examples in man of inoculation with tuberculosis through the medium of the skin or mucous membrane. It is evident that to effect much inoculation the circumstances must in one way or another be most exceptional.

The pathologist at a large hospital for consumption must have his hands soaked for hours at a time with tuberculous pus. His knuckles cannot always be free from abrasions and scratches; and yet how very rarely does any development of tubercle occur at the undoubtedly inoculated spot. It is true that a certain form of "anatomist's tubercle" has been shown to be, in actual fact, tuberculous; but the comparative rarity of even that readily-cured affection is noteworthy.

As examples of the communication of the disease by inoculation, the following may be given.

A servant-maid wounded her finger in cleaning the spittoon of her master, who was dying of phthisis, and in whose sputum bacilli were found. An indurated ulcer formed on the finger, the tendon sheath became involved, and the axillary glands enlarged. The finger was amputated and the glands were excised. The morbid changes were found to be tuberculous and the bacillus was discovered in the parts removed. The girl made a sound recovery. The case is recorded by Tscherning.

Leloir details a case of lupus of the nose in a young girl who lived with a sister, the subject of phthisis, and often used her handkerchief.

A girl tatooed on the arm by her lover, who was dying of phthisis, developed lupus at the inoculated spot.

Unna describes lupus of the lobule of the ear in a girl of thirteen, who wore the earrings of a friend who had died of phthisis.

Several examples of the communication of tuberculosis by the Jewish operation of circumcision have been recorded.

Jeanselme gives the case of a woman who developed tuberculous ulcers of her fingers after long attention upon her husband, who had a fistula (which she dressed), and who died of phthisis.

(2) *By respiration and* (3) *by cohabitation.*—A certain number of instances have been recorded in which it is more or less apparent that tuberculosis in the form of phthisis was communicated to a sound individual. These examples have been mainly noticed in the persons of husbands and wives, or intimate friends, or nurses and patients. The cases, however, are quite uncommon, considering the wide prevalence of the disease. Jousset would place them as low as 2 per cent. This method of communicating tuberculosis is practically unknown in hospitals for consumption. It is obvious, however, that tuberculous diseases are more common in crowded communities; and one can imagine that a wife in poor circumstances, who is in constant attendance, in a close ill-ventilated room, upon a husband who is dying of phthisis, is in a most favourable position for acquiring the disease.

A confined atmosphere, the lack of fresh air and of sunshine, are favouring elements in the spread of tubercle.

(4) *By feeding.*—This method of communicating tubercle is in the human subject exceedingly rare, and is doubted by many. Jousset affirms (writing in 1893) that no case has been demonstrated. It would appear that the bacilli of tubercle cannot live in the healthy stomach, and cannot survive the heat involved in the cooking of meat. The communication of the disease to a child by means of milk from a tuberculous cow has not been established beyond doubt. Niepve gives the case of an infant born of healthy parents who was suckled by a tuberculous nurse who had bacilli in her milk; the child soon died of tuberculous meningitis.

The tuberculous individual.—Two things are needed to set in action the tuberculous process. There must be the *seed*, and there must be a *suitable soil.* The seed is provided by the tubercle bacillus, and among the haunts of men this bacillus must be fairly liberally distributed. The more closely human beings are packed together the more common may the bacillus be expected to be. In spite of the very general diffusion of the bacillus in our midst, it is remarkable that tuberculosis, although a common affection enough, is by no means common to all men. Some individuals are more exposed to infection than others, it is true; but that does not explain the distribution of the disease. Were the seed alone the necessary thing, then all the nurses in hospitals for consumption should become tubercular, and from such institutions tuberculosis should spread as from a centre.

Tuberculous disease, however, does not spread in this way, or only in a few very rare accidental instances, which have been already alluded to.

It is evident that the soil is of much more importance than the seed—that there must be a large proportion of a population who are practically non-inoculable, while there is a proportion whose bodies form a suitable medium in which the bacillus can grow. The tuberculous individual is the one whose tissues form a suitable soil for the development of the bacillus. The non-tuberculous individual is the one in whose tissues this micro-organism cannot obtain a hold, it being assumed that both are equally exposed to infection.

Tuberculous disease may be acquired, but it cannot be *inherited;* that is to say, the parent cannot hand down to its offspring the tuberculous process in its complete condition with its bacilli, its tubercles, and its caseating districts. What the parent can unfortunately transmit to its offspring is a body whose tissues form a suitable soil for the development of the bacillus, should that microbe ever reach it in sufficient strength. It is only in this sense that tuberculosis can be said to be inherited. What is inherited as a matter of fact, is not a disease, but a predisposition.

In a large number of tuberculous persons this inheritance is marked. One or both parents have died of phthisis, or they are themselves sound, but there is phthisis in other members of the family; or in the same family there is a history of enlarged glands in the neck, of joint disease, or of bone affections.

On the other hand, the state of body needed to form a suitable soil for the bacillus may be *acquired.* This is well seen in what is known as "penitentiary" or "workhouse" tuberculosis. A number of children find their way to these institutions. Their parents may have been entirely free from tuberculosis, although possibly not perfect in health in other respects. The children are often brought up under unhygienic conditions, with insufficient exercise, insufficient sunlight, insufficient air, and insufficient food. A small proportion of them become tuberculous for no other reason than that their surroundings during the period of growth have been such as to render their tissues a favourable soil for the bacillus of tubercle. Had they been placed in happier circumstances, their bodies would very possibly have remained an unsuitable medium for the growth of this micro-organism.

It has been said by some writers that any condition of ill-health may render a patient tuberculously disposed, but this is not so. The distribution of tuberculosis is not identical with the distribution of mere ill-health. There is much evidence to show, however, that there is a certain amount of truth in the statement. In support of this, the following illustrative cases may be given as representative of quite considerable classes.

(1) A medical man had had three perfectly healthy children, and then acquired syphilis through a scratch on the thumb when performing an operation. There was no tuberculous history in his own family, nor in his wife's. He lived apart from his wife for two years. A fourth child was then born. It showed no signs of inherited syphilis, but it became the subject, when twelve years of age, of most extensive tuberculous disease.

(2) The youngest child of a large family was the only one that exhibited any signs of tubercle. The father and mother were entirely free from that affection, but the child in question was the only child born after the father had developed Bright's disease.

(3) A woman of 20, born of perfectly healthy parents, and with no tuberculous history in the family, developed a considerable mass of tuberculous glands in the neck. Until she was 18½ she had been living in comfortable circumstances. She then became very poor, and had to support herself. After working for some months in a close ill-ventilated room the glands appeared.

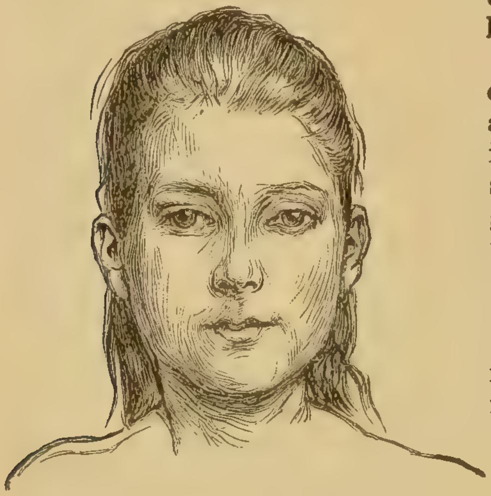

Fig. 68.—The Fine Type of Tuberculous Individual.

If by the term a "tuberculous individual" it is intended to imply a person prone to tubercle formation, it will be evident from what has just been said that that individual can hardly be expected to present any common general characteristics. There is, indeed, no *physiognomy* peculiar to and diagnostic of tuberculosis.

While a large proportion of the patients present no characteristics which can be accepted as the basis for a type, it will be evident—especially when children and young adults are the subjects of the inquiry—that there are some features met with so often among the tuberculous, that it is possible roughly to classify the majority of the individuals in two classes.

The distinguishing characteristics of the two classes are, of course, not always so marked as they are in the following description :—

1. **The fine or sanguine type.**—The children have fine and regular features, well-shaped limbs, and delicate hands (Fig. 68).

The skin is clear, white, and thin, and marbled with venules. The complexion is usually fair. The face is oval, the lower jaw small, and the lips thin. The eyes are bright and covered with long lashes, and the hair is often remarkably fine and silken. In the younger children there is not infrequently a faint growth of downy hair over the forehead and on the backs of the fore-arms.

The teeth are, as a rule, white and well formed, but brittle. Not infrequently they are noticeable by the large size and square outline of the upper central incisors.

These children are sprightly and emotional; full of life and, from the modern standpoint, would be called neurotic.

This is the type which is *inherited.* These are the children whose parents have died of phthisis, or who have a strong tuberculous history in their families. They are born with a predisposition, and are congenitally a favourable soil for the bacillus.

These are the children who may develop phthisis, tuberculous meningitis, or peritonitis. They often have enlarged tonsils, post-nasal growths, and enlarged glands. They are liable to catarrh. They occasionally present lupus. They are attacked with tuberculous bone and joint disease, but not with marked frequency. Phlyctenular ophthalmia is not common among them, nor are skin affections.

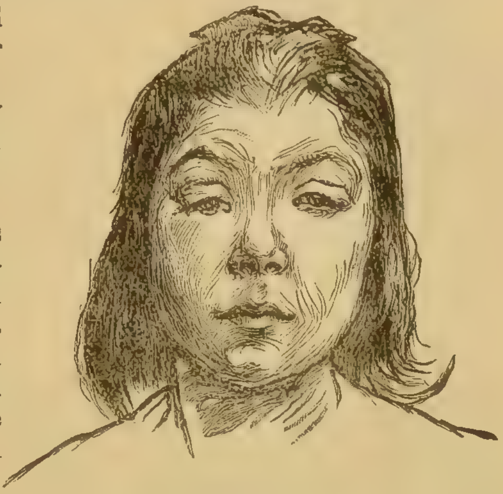

Fig. 69.—The Coarse Type of Tuberculous Individual.

2. **The coarse or phlegmatic type.**—In this class are comprised individuals who are, as a rule, short and bulky, with coarse limbs, large hands and feet. The face is broad, the lower jaw heavy, the malar bones often prominent, and the features generally coarse and irregular (Fig. 69). The nose is usually thick, the lips tumid, the lobes of the ears large, and the neck unshapely. The teeth are often ill-formed, and soon become carious. The skin is coarse, harsh, and thick. The amount of subcutaneous cellular tissue is considerable, and often sufficient to conceal the muscular outlines of the body. The skin in the previous type is fine, and it is possible to pinch up with the fingers a little portion of it; but in these individuals none but a large fold of skin can be picked up, as it is so coarse. The children of this class appear dull, flabby, and heavy-looking. They are apathetic and without vivacity, have little muscular power, and are soon tired.

Their circulation is remarkable. The blood appears to stagnate in exposed parts, the cheeks often assume a bluish or mottled aspect, the extremities appear swollen, and the skin itself feels often chilled and clammy. These children are very liable to chilblains.

They are the common subjects of glandular swellings, of enlarged tonsils, of post-nasal growths, of phlyctenular ophthalmia, of lupus, and of muco-purulent discharges from the nose, ears, or vagina. They are often attacked by lupus and are often the subjects of bone and joint disease. They seem prone, indeed, to the surgical forms of tuberculosis, and are not so commonly the subjects of phthisis, meningitis, or peritonitis.

This is the type of tuberculous individual whose peculiarities are probably *acquired.* To this type belongs "parochial struma" and the tuberculous affections common in the slums of great cities.

Age.—Tuberculous disorders may appear at any age, but they distinctly affect certain periods of life.

Speaking especially of the surgical forms of tuberculosis, it is to be observed that the largest number of cases belong to the ages between five and fifteen. Another period of frequency would appear to be between twenty and twenty-five, and another period a little after thirty.

The term "*senile scrofula*" or "*senile tuberculosis*" is applied to instances in which tuberculous diseases make their appearance in old people. Such instances are rare. In sixteen cases reported by Bourdelais, five patients were between sixty and seventy, seven were between seventy and eighty, two were over eighty, and the remaining two were under sixty. Senile tuberculosis is as common in females as in males. In some instances there is a history of a tuberculous affection in early life, but as a rule the disease is primary, and represents the first appearance of tubercle. The diseases to which the subjects of senile tuberculosis are liable are identical with those met with in earlier life. The commonest manifestation is bone disease, which is often about the wrist. In other instances there have been joint disease, lupus, tuberculous ulcers, or glandular enlargements.

Environment.—In very general terms, it may be said that tuberculous affections are encouraged in those who live in closely-confined districts, and whose dwellings are dark, damp, ill-ventilated, and removed from a free access of fresh air.

It would appear, also, that a district lying high and dry and bare has a more favourable influence than has a low-lying district, where the atmosphere is laden with moisture, and where vegetation is luxuriant, or at least plentiful.

Tuberculosis and scrofula.—For some centuries a great war of words was waged over the relations of tubercle and scrofula. The term "struma" or "scrofula" was derived from the common appearance presented by enlarged glands in the neck, and as years advanced the expression was loosely associated with other vaguer conditions of mere ill-health. The diseases which were usually termed scrofulous were the following, and it will be observed that they simply embrace the common external manifestations of tuberculosis:—phlyctenular ophthalmia, chronic otorrhœa, enlarged glands, "white swellings" of joints, bone disease, "strumous ulcers," cold abscesses, lupus, and certain other less well-defined conditions. At one time the manifestations of hereditary syphilis were included under the term scrofula; and, indeed, that term was employed to cover any condition of ill-health in a child to which a more precise term could not be applied.

It has now been shown that there is no disease requiring the special designation scrofula. The term was a convenient term to cover ignorance. It has served its purpose and may now be most profitably abandoned. The diseases commonly called scrofulous have been long since shown to be of a tuberculous nature. There can be no object in still retaining the term, even if limited in application to the commoner external manifestations of tuberculosis.

It was to the coarse or phlegmatic type of tuberculous individual that the terms "strumous" and "scrofulous" were usually applied.

The general treatment of tuberculosis.—It will be only appropriate to refer to those forms of tubercular disease which come under the surgeon's notice. A considerable part of the treatment of such forms of tuberculosis depends mainly upon obvious measures of common sense.

(1) Any irritating or localising cause of the trouble should be sought for and, if possible, removed. Thus, in a case of enlarged cervical glands, hypertrophied tonsils and post-nasal growths should be got rid of, diseased teeth should be treated, and attention should be given to phlyctenular ophthalmia, to discharges from the nose or ear, to eruptions on the scalp, and such other disturbances in the periphery as may tend to induce secondary tuberculous changes in the glands.

(2) In the second place, the involved part should be kept at rest. This is very obvious in the case of diseases of bones such as those of the spine, and in joints such as the hip or knee; but it is not so often insisted upon as it should be in connection with glandular affections. When the glands are in the axilla the arm should be kept at rest; and when they involve the neck, that ever-moving part of the body should be fixed by some simple collar or stock.

(3) In the third place, the tuberculous disease should be thoroughly eradicated in suitable cases. In estimating the suitability of a case, it would be assumed that the local disease was well advanced, that it had progressed as far at least as caseation, that its removal could be entirely effected without undue risk, and that the prospects of spontaneous recovery were either hopeless or, at least, uncertain and remote. In connection with this point a considerable degree of discretion must be exercised. There can be little doubt that a long-abiding tuberculous ulcer, a caseous tonsil, or a broken-down or softening gland had better be disposed of by operation with as little delay as possible. But, on the other hand, it must be remembered that under the expectant treatment tuberculous troubles have exhibited remarkable powers of recovery.

Glands, after remaining in evidence for months, may disappear;

joint affections, if properly treated, do by no means ill; and the surgeon must decide in relation to each individual case how much or how little is to be expected from natural processes. I would lay stress upon this, because there is no doubt but that glands are now and then removed which would have subsided without operation, and joints are occasionally excised which would have recovered if treated by long-continued rest under favourable conditions. I have seen more than one case of tuberculous orchitis which has been condemned to castration recover after a moderate scraping away of the evident disease and a residence in a favourable climate. There appears often to be a cycle in tuberculous diseases. There is an outburst of the disease, and after it the trouble tends to clear up and get well, and the patient remains free until the next uncertain period of eruption.

(4) Finally, considerable attention should be paid to constitutional treatment. There are no drugs which are specifics against tubercle, and three-fourths of the medicines given are administered not to treat the disease, but to soothe that inherited craving in the human race for physic, which is almost as uncontrollable as the craving for drink.

The principal elements in the treatment are plenty of air, plenty of sunshine, and plenty of good food. Let the patient escape from the crowded city and live in the country; let him sleep with his bedroom windows open, winter and summer. So long as mere movement will not be in itself injurious, let him be moved out into the open air. Let him live in the open air.

Let the atmosphere be dry. Patients do not do well in densely-wooded districts, in valleys, by the banks of large rivers, and in moist places; they do better by the sea, in districts with a low rainfall, with a porous soil, with spare vegetation, and with an unimpeded rush of air from the sea. Sunshine and dry air are deadly to the bacillus, and light and oxygen are of more value than all drugs. In England there is some difficulty in spending much time in the open air in the winter; although I have had patients with high temperatures and suppurating wounds out of doors every day in the winter, in spite of snow, and frost, and rain. If the patient can winter in a more suitable climate, let him go to a place where the air is dry and the sun bright, and where he can be out of doors all day. Thus it happens that surgical forms of tuberculosis do as well at St. Moritz and in Canada as at Cairo and California. These are places with this at least in common: there are facilities and inducements to be out of doors in the winter months.

In the matter of drugs, cod-liver oil may be given as a food during the cold weather; iron may be needed when there is anæmia, and is conveniently given in the form of Flitwick water; arsenic and quinine are now and then of value, and in some instances iodide of potassium is of service. Iodine is conveniently employed in the form of Kreuznach water, and the special treatment carried out at Kreuznach is often of decided benefit in the early stages of gland disease.

Certain tuberculous affections.—It will be convenient to describe in this place certain tuberculous troubles which are not readily included in other Articles.

The tuberculous ulcer.—This ulcer, when met with on the *skin*, is usually dependent upon the bursting of a tuberculous abscess, or is found about tuberculous sinuses, or has followed the breaking down of a "strumous node." (*See* page 122.)

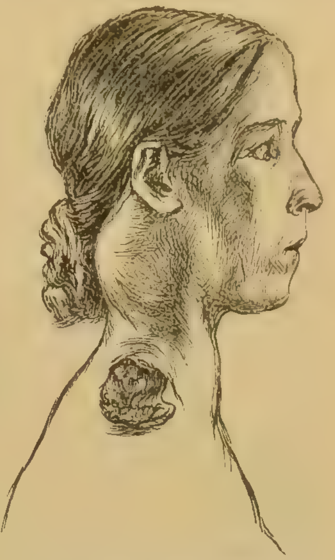

Fig. 70.—Tuberculous Ulcer (with Gland Disease) in a Patient dying of Phthisis.

They are common, therefore, on the neck in connection with suppurating gland disease, about diseased joints and bones in association with discharging sinuses, about chronic abscesses—such as the ischio-rectal —which have found a vent through the skin, and in other parts in which there has been infection of the skin. They may be primary, and due to inoculation (*see* page 353); but, as will be already evident, the tubercular ulcer of the skin is usually secondary (Fig. 70). The ulcer, when fully developed, has these characters. It is of irregular outline; the edge is neither raised nor indurated, but is flat and thin. The skin around the ulcer is a purple colour, and the margin of the ulcer is undermined so far as this discoloured integument extends. The degree of the undermining is often considerable, and can be estimated by a bent probe (Fig. 19). The base of the ulcer is commonly irregular and dirty, exhibiting no disposition to healthy healing, or is covered over with pale and œdematous granulations. These granulations may project above the surface of the surrounding skin. The undermined skin around the ulcer is apt to give way in places, and in this way holes and irregular breaches in the skin in the vicinity of the main ulcer may be produced. It is the insidious undermining of the integument, and the absence of a protecting barrier of sound lymph, which give the main features to the tuberculous ulcer.

The *scars* that result from this form of ulceration vary, and are most commonly drawn attention to when situated in the neck. In some instances the resulting cicatrix is surprisingly soft, simple, and pliant. In other instances the scar left is irregular, hard, bossy, raised, and unsightly, by reason of its purple colour. Now and then, as the result of the undermining and irregular destruction of the integuments, the scar may be marked by tags of skin or bridges, or bars of skin not unlike the columnæ carneæ of the heart. Often the scar shows—when there has been deep suppuration—a severe degree of contraction, whereby unsightly depressions are produced on the surface. In many instances I have made microscopical sections of exuberant scars from tuberculous subjects, and have found that they seldom exhibit any tuberculous change, but are rather the seat of a cheloid formation. Cheloid is well known to be liable to occur in or about the neck, and especially to follow wounds that have been slow in healing or long irritated.

The tuberculous ulcer of the *mucous membrane* is of common occurrence, and, like the similar lesion on the skin, is very usually secondary. It is common at the orifices of the body, and is not infrequently associated with grave tuberculous disease elsewhere, as illustrated by the ulcers of the mouth in advanced phthisis, and of the anus in tuberculous disease of the colon or rectum.

The tuberculous ulcer is met with on the lips, the palate, the tongue, the lining of the buccal cavity, the larynx, the alimentary canal, the bladder and urethra, and, indeed, no mucous surface can be said to be exempt from it. In appearance the ulcer is very like that met with on the skin. The edges are neither raised, indurated, nor everted. They are, on the other hand, flat, thin, sharp-cut, and bevelled or undermined. The surface is uneven, and covered with yellowish-grey sloughy-looking mucus, or with pale and weak granulations. In depth the ulcers vary greatly.

The *treatment* of tuberculous ulcers is mainly local. The whole of the diseased tissue should be destroyed by the cautery and the sharp spoon. All purple undermined skin should be sacrificed if sound healing is hoped for. The scraping should extend up to the region of undoubtedly sound skin.

After the little operation the part should be dressed with iodoform.

The tuberculous node.—This is known also as the *strumous node*, and as the *scrofulous gumma.*

The term is applied to a localised tuberculous focus in the subcutaneous tissue. In a tubercular subject—and not usually in the subject of actual tuberculous disease—a small nodule is felt under the skin. It is commonly fixed, is not painful, but may be tender. The skin over it is normal. The little swelling, which is hard at first, increases and becomes softer. The skin covering it becomes adherent and reddish. Later, it assumes a deep purple colour, and beneath the discoloured area the now fairly extensive swelling is felt to fluctuate. The skin in due course gives way in one or more places, and there results a tuberculous ulcer with undermined edges. Most of these deposits in the subcutaneous tissue are of small size. If, however, the giving way of the skin be long delayed, the undermining of the integument may extend over several square inches.

Certain of these "nodes," especially when on the skull or over the shin, originate in the periosteum, and are evidently from the first deeply placed.

They follow the course already indicated, save that on the scalp the skin undergoes little change in colour; and when they have burst, or have been evacuated, an area of bare bone is demonstrated at the base.

The *treatment* consists in an early incision and an early evacuation, by scraping of the diseased tissue. All quite disorganised skin should be destroyed. A dressing of iodoform is then applied.

XVII. RICKETS.

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Ætiology.—Rickets or Rachitis is sometimes called the English disease, in honour of its discoverer, Francis Glisson (1650).

Rickets is a general disease, the most striking surgical features of which are seen in the changes occurring in the bony skeleton. It is met with mainly among the children of the poorer classes of large towns, and is essentially a disease of malnutrition, induced by improper feeding and exaggerated by imperfect hygienic surroundings. Softness of the bones may be produced experimentally in animals by the exclusion of lime salts from the diet, and well-marked rickets has been observed in lambs where the food of the ewes has been deficient in this element; again, it has been seen in young lions fed with lean meat without bone, and in young monkeys placed upon a purely vegetable diet (Sutton). In all these instances the disease has been readily cured by the addition of the deficient elements to the food.

Heredity plays no part in its occurrence, although it is frequently met with in large families; and here the younger members are the more frequently and severely affected. Children suckled by their mothers are rarely attacked; but these may suffer if lactation be unduly prolonged, if the mother be out of health, or become again pregnant while suckling her child.

Hand-feeding is the most prolific source of the disease: patent foods, containing large quantities of starch and flour, and deficient in a proper proportion of animal fat (Cheadle), proteid, and earthy salts, being often employed. Poor milk, skimmed, and thus deprived of its normal fatty element, may produce the disease; again, the development of an attack is often noticed subsequently to attacks of diarrhœa and vomiting, induced by feeding with undiluted cow's milk, leading to defective assimilation of the fatty and proteid elements.

Syphilis is in no sense a cause of rickets, but as a debilitating disease may perhaps predispose to its occurrence, and also modify in some degree its course. It is almost certain that craniotabes and visceral enlargements occur in both diseases, since the exhibition of mercury readily affects these conditions in children with a syphilitic history, while in those in which such history is absent no improvement occurs. A strong proof also of the occurrence of craniotabes in rickets, apart from syphilis, is found in the fact that it is met with in a well-marked form in animals, such as monkeys (Sutton), in whom syphilis is unknown. Rickets may be associated with *scurvy*, the two conditions being induced by the same cause—*e.g.* improper feeding. The relation of rickets to the *tubercular diathesis* is not very close: the same conditions predispose to both; but it is uncommon to find rickety children affected with the ordinary forms of surgical tuberculosis, such as joint disease or lupus. On the other hand, the common occurrence of bronchial catarrh is, no doubt, of importance in the incidence of pulmonary tuberculosis.

Time of appearance.—The disease may rarely be congenital, and marked epiphysial enlargement may be noted as early as the third month. It most commonly becomes apparent, however, about the sixth month, and rarely, if ever, occurs after the period of eruption of the milk teeth. Cases designated late rickets are met with; but these are probably recurrences of early attacks which have passed unnoticed. Even when the disease has been subjected to prolonged courses of treatment, and appears thoroughly cured, a predisposition to recurrence exists (Henoch).

Morbid anatomy.—In rickets, the process of ossification of the skeleton is disturbed by irregular progress of the preparatory changes occurring at the epiphyses and in the subperiosteal layer, and the deficient deposition of lime salts. An ill-developed substitute takes the place of normal bone, while an increase in the centrifugal absorption accompanying the natural process of formation of the medullary cavity and the modelling of the bones removes normal osseous tissue developed previously to the commencement of the disease. The degree to which these changes progress varies, and in severe cases a comparatively soft yielding column takes the place of a properly resistant bone.

In the normal course of development of the skeleton, certain physical forces—especially muscular action—influence the contour of the bones, producing regular curvatures and eminences, both in individual bones and in those arranged in series. Such curves normally serve the purposes of endowing the bones with strength to resist intrinsic and extrinsic force, give elasticity, and provide for the exertion of more effective leverage by means of the muscles. Rickets occurs during the active development of these changes of form and outline, and the inefficient resistance offered by the affected bones leads to exaggeration of the eminences and curvatures.

The *primary deformities* in early rickets, therefore, consist almost entirely in the exaggeration of normal curves, while to these, various deformities dependent on pressure exerted by the weight of the trunk and extremities in the acts of sitting, crawling, or walking, are superadded at a later date. Deformity thus produced is rendered the more striking by limited growth in length of the bones, the temporary disturbance of the normal ossification leading to permanent shortening, which, in the case of such bones as the femur and humerus, may amount to a quarter of their proper proportional length (Humphry).

The *periosteum* of rickety bones is soft, reddened from increased vascularity, thickened, and when stripped from the bone is rough and irregular, bearing numerous spicules and fragments of ill-formed osseous tissue on its inner aspect. Beneath it are found a varying number of concentric layers of reddish or pale spongioid bone, resembling pumice-stone in consistence, which can be readily cut without injury to the knife. When the disease is far advanced, tissue of this nature, completely unossified, may occupy the whole thickness of the bone between periosteum and medullary cavity, and also more or less fill up the concavity of any curvature existing. In this condition and its degrees, the ease with which curvatures or fractures from slight degrees of violence may occur is readily understood; while the abnormal thickness of the periosteum explains the tendency to fracture of the green-stick variety, and the softness of the bone to the frequent inability to elicit ordinary crepitus during life.

The earliest gross changes are, however, noted in the *epiphyses*. These are enlarged, thickening existing both on the proximal and distal sides of the line of cartilage. On section, the cartilage is semi-transparent or gelatinous-looking, reddish from abnormal vascularisation, and irregular at the periphery, in place of maintaining the normal even line (Fig. 71). Microscopic examination shows the deeper or osteogenetic layer of the periosteum to be chiefly affected, and the layers of spongioid bone beneath to consist of calcified islets, arranged radially to the surface of the diaphysis. Between the islets of bone are large red medullary spaces. In the earlier stages of the disease these changes are limited to a superficial layer, ensheathing normal bone; but the latter may gradually disappear with the progress of the disease, and the natural process of modelling and enlargement by absorption from within. A similar layer of spongioid bone may then line, or more or less completely obliterate the medullary canal.

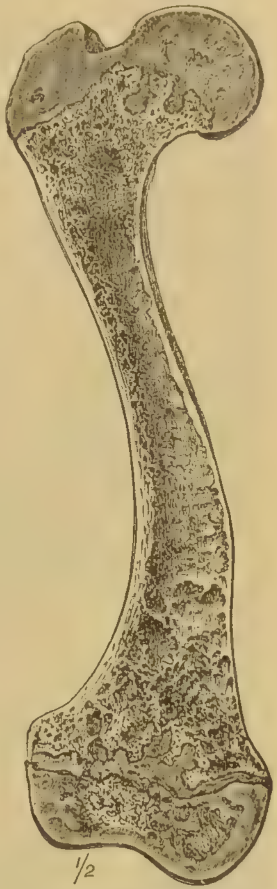

Fig. 71.—Section of Rickety Femur, showing curves and irregularity of epiphysial cartilage. (St. Thomas's Hospital Museum.)

The epiphyses present the following abnorma appearances on microscopic examination (Fig. 72).

The cartilage cells are seen to have multiplied in an excessive and irregular manner (Fig. 72, A), and between the columns of cells so formed large vascular medullary spaces exist (B). The next layer consists of unossified bone tissue (C), in places intermixed with small areas of cartilage cells, which have undergone premature calcification (D). Beyond this are islets of fully-developed bone, still surrounded by bone tissue, as yet uncalcified, enclosing abnormally large and vascular medullary spaces (E).

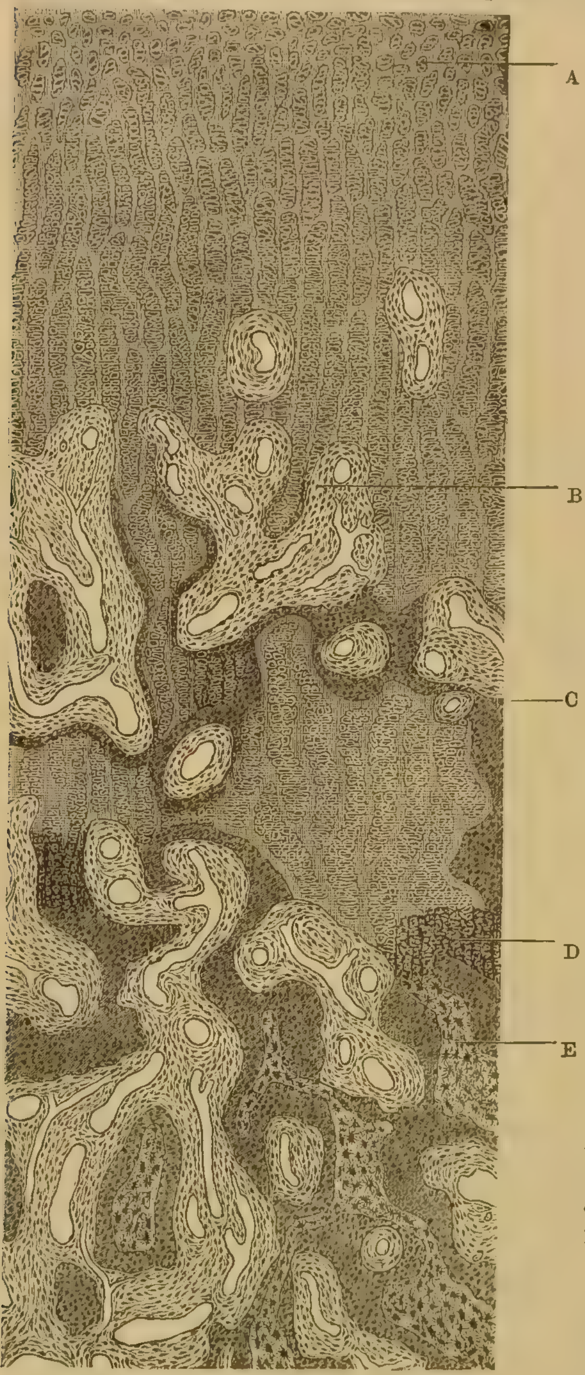

Fig. 72.—Section of an Epiphysis in Rickets. (Modified from Ziegler.)

A, Irregular multiplication of cartilage cells; B, vascular medullary spaces; C, unossified bony tissue; D, calcified cartilage cells ;E, imperfect bon

Chemical examinations of the bone in rickets have shown it to be very deficient in lime salts, the proportion to the organic constituents being reduced in marked cases to as much as $\frac{1}{5}$ in place of the normal $\frac{2}{3}$.

The liver and spleen are occasionally much enlarged, and a less marked change is observed in the kidneys and lymphatic glands. The enlargement depends mainly on an increase of the fibroid elements, with some overgrowth of the glandular tissue (Dickinson). The voluntary muscles are pale and flabby, in the heart fatty degeneration has been noted, and the blood is deficient in red discs.

The *tendency* of the disease is towards spontaneous cure; this is effected by obliteration of the abnormally large medullary spaces, both at the epiphyses and in the subperiosteal layer, by extremely dense bone, the process of ossification resembling that observed in the consolidation of callus in fractures. Abnormal vascularity is, however, long retained, as is often evidenced by the free hæmorrhage during osteotomy from bones apparently already dense and strong. The spongioid bone deposited in the concavities of the curvatures ossifies in like manner, and thus strong bony buttresses are produced, the bones being often further strengthened by narrowing or obliteration of the medullary canal.

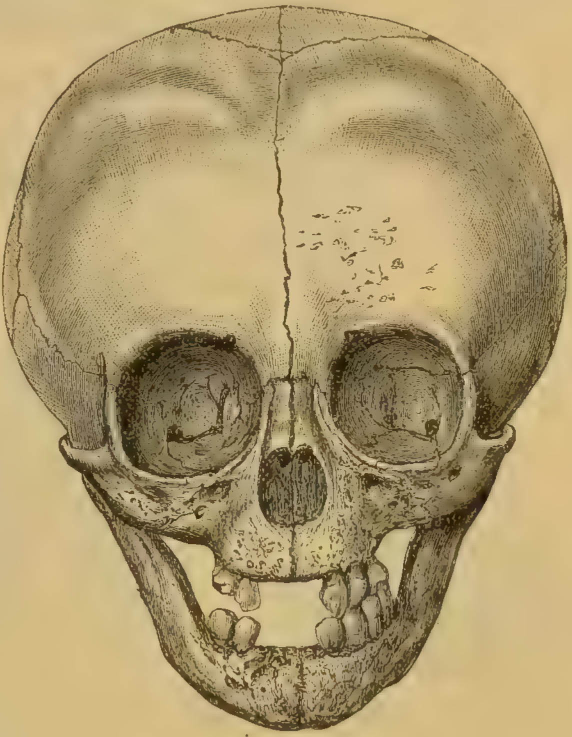

Fig. 73.—Skull of Rickety Child, illustrating abnormal preponderence of cranium, defective development of face, rapid decay of milk incisors, and angular form of mandible. (St. Thomas's Hospital Museum.)

Special deformities. Skull.—Changes in the bones of the head, particularly in those ossified in membrane, are the earliest to occur, commencing even in the third month. Longitudinal prominences in the marginal growing portions of the flat bones mark out the lines of the sutures; and this is particularly well seen at the coronal suture. The sutures themselves are deficient in firmness and resistance, and the fontanelles remain open until a late date. The large anterior fontanelle often closes only as late as the sixth year. Certain parts of the skull — notably the frontal and parietal bones—gain much in thickness, while the occipital may remain abnormally thin. The incapacity of the imperfect bone to resist pressure may be evidenced by the development of small soft areas from $\frac{1}{10}$ to $\frac{1}{2}$ an inch in diameter, in which the skull may be thinned, or true osseous tissue may be entirely absent. These soft yielding spots are most common in the region behind the parietal eminence, and in the occipital bone. They occur, however, rarely in the temporal, and even in the frontal bone. They are usually not numerous, but have been met with to the number of thirty. This condition is known under the name of *craniotabes*, given to it by its original describer (Elsasser). The occurrence of the disease during the first year of life, normally a period of especially active growth of the facial portion of the skull, leads to very characteristic changes in the head. The cranium maintains its early preponderance in size, and the forehead appears abnormally prominent, while the face is small and peaked (Fig. 73). The deformity most characteristic in the *facial bones* is that observed in the mandible; defective development of the outer wall of the bone interferes with the acquisition of the proper arched form by the body, the incisor teeth being arranged transversely, and from them the remaining teeth diverge obliquely backwards, the alveolus being somewhat inverted posteriorly, so that the teeth, instead of being vertical, point inwards. The lower margin of the body is everted (Fleischmann). *Dentition* is much delayed, the first tooth often not appearing before the twelfth month, and subsequent eruption is irregular and retarded; the enamel is defective, striped, and pitted, and the teeth readily wear away to the level of the alveolar margin.

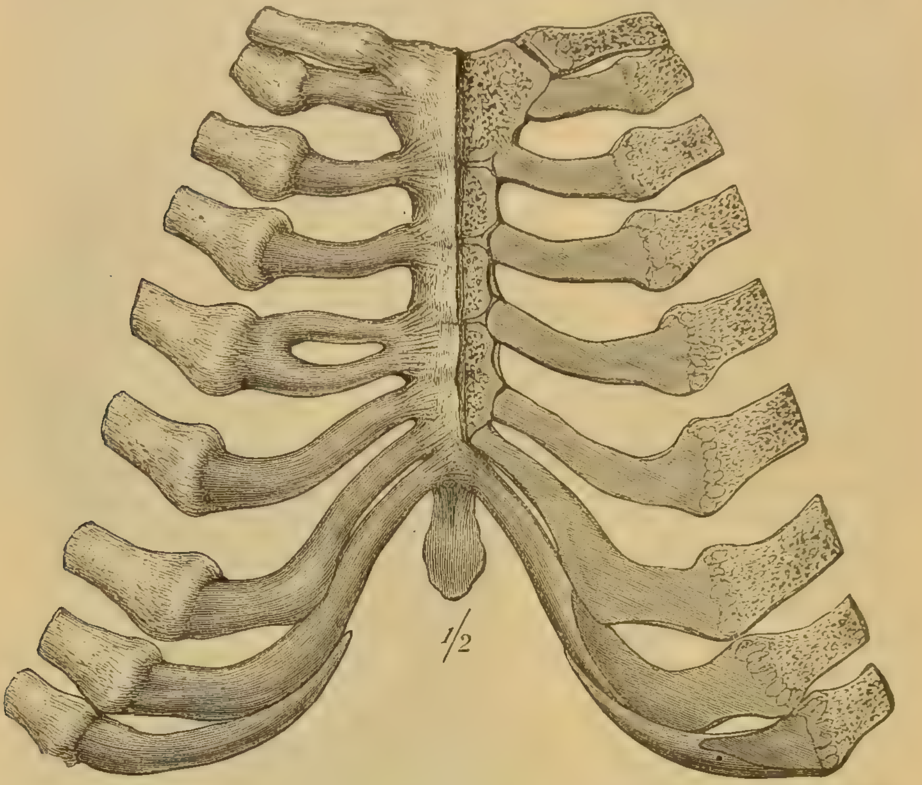

Fig. 74.—Sternum, Costal Cartilages and Ribs in Rickets. The section on the left side shows the irregularity of the epiphysial ossification. (St. Thomas's Hospital Museum.)

Spine.—The earliest and commonest change is an increase in the great dorsal accommodation curve, in which the lumbar spine may also be involved (kyphosis). This is compensated for later by an increase in the cervical, and less in the lumbar convexities, the change in the cervical spine often causing the child to hold the head abnormally tilted upwards. At a later date—from one-and-a-half to four years—lateral curvature may develop; in the earlier period this is often an extensive curve, with the concavity to the right, due to the child leaning to the right to rest the head on the nurse's shoulder, when held in the habitual position on the left arm of the bearer. This curve may be accompanied by slight or no rotation of the vertebral bodies. When developed subsequently to the assumption of the erect position, typical scoliosis may be met with; the deficient resistance offered by the softened bones and ligaments being especially favourable to the influences usually producing an habitual scoliosis.

Thorax.—Changes in the thorax occur early—usually about the sixth month—closely following those observed in the head. When well marked, the shape of the chest is approximated somewhat to the quadrupedal type. Beading of the epiphyses, involving both rib and cartilage, especially on the pleural aspect, occurs, producing the so-called *rickety rosary* (Fig. 74). The ribs, by reason of their deficient power of resistance, fall in just beyond the line of junction with the cartilages, partly from the effect of the atmospheric pressure during inspiration, partly from the lateral pressure exerted upon them by the hanging upper extremities. The ribs, from the second to the eighth, are the most affected, failing to attain a proper length, while the angles become less obtuse than normal. The sternum at the same time projects or, in rare cases, sinks bodily. The outline of the lower part of the thoracic wall is influenced by the pressure of the solid viscera underlying it; thus the heart, liver, and spleen all support the ribs, and tend to produce irregularity of contour, especially the liver and spleen when they are much enlarged. The defective growth of the ribs causes serious permanent diminution in the vital capacity: a serious matter when pulmynary affections supervene.

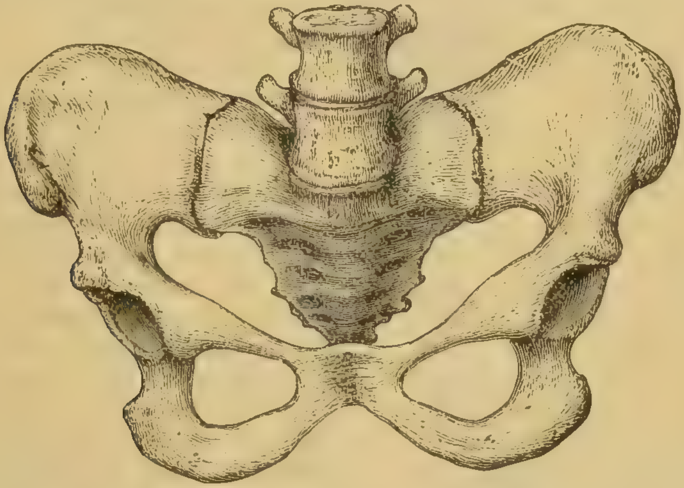

Fig. 75.—Rickety Pelvis. (From St. Thomas's Hospital Museum.) The great prominence of the sacro-vertebral angle is not well shown in this position of the pelvis.

Pelvis.—The downward pressure of the spine gives rise to increase of the sacro-vertebral angle, while the lateral pressure exerted by the femora or the acetabula flattens the lateral wall of the pelvis. The weight of the abdominal contents expands the ilia, while the ischial tuberosities are inverted. In this way the triangular or heart-shaped pelvis is produced; when extreme, one of the most serious bony deformities in women, as affecting parturition (Fig. 75).

Long bones.—The notable changes are enlargement of the epiphyses, exaggeration of the normal curves, the development of pressure curves, and of muscular apophyses in certain positions; and later, deficiency in proportional length.

In the *upper extremity* the lower epiphysis of the radius and those about the elbow usually exhibit the most marked enlargement: perhaps because most readily investigated by palpation. The curves of the clavicle may be exaggerated, and an abnormal outward curvature of the humerus, the greatest convexity of which corresponds to the insertion of the deltoid muscle is not uncommon. In children who, from inability to walk, have crawled freely, the bones of the fore-arm are often strongly curved outwards, especially in the lower third. The production of this deformity is readily explained by bearing in mind the direction of the pressure brought to bear on the spreading pronated fore-arm and hand in this form of progression.

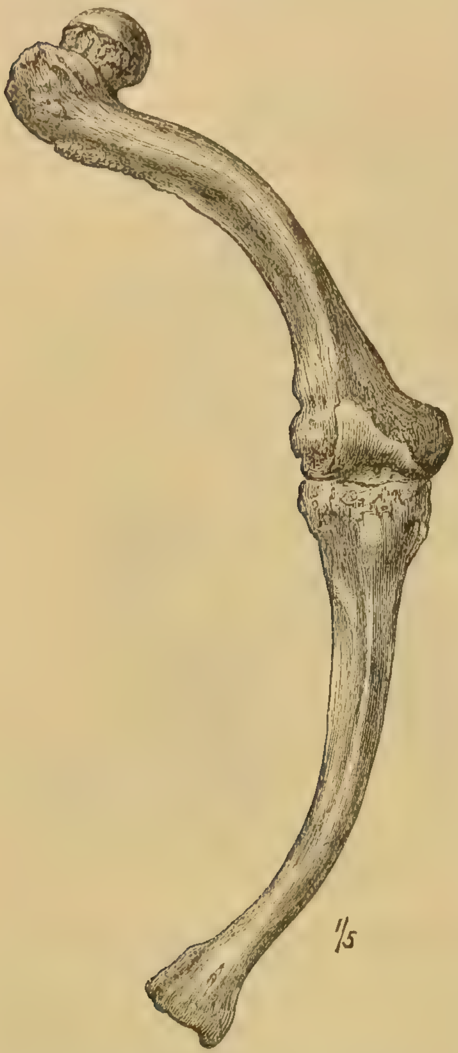

Fig. 76.—Adult Femur and Tibia in a case of cured Rickets. Note angle of femur, gluteal trochanter, overgrowth at inner section of lower epiphysis of femur. curves of both bones, and buttress to curve of femur.

In the *lower limb* epiphysial enlargements are readily detected at the ankle and about the knee, but in slight cases they are less marked than in the upper extremity. The degree of deformity varies greatly. In children who have been unable to walk during the most active part of the disease, little deformity or varying slight exaggerations of the normal curvatures are met with. When, on the other hand, standing and walking have been possible, very marked changes occur.

In the femur the angle of the neck is reduced: the shaft forms one large anterior curve; and in cases of knock-knee there is marked proportional overgrowth at the inner segment of the lower epiphysial line. A more or less well-marked abnormal muscular apophysis may represent the gluteal trochanter of the horse (Fig. 76).

The tibia and fibula acquire a forward, or forward and outward, bend in the lower third, and there may be marked proportional overgrowth at the inner segment of the upper tibial epiphysis. Much more rarely a marked bend, either forwards or backwards, may be developed at the upper epiphysis. The development of *bow-leg* or *knock-knee* depends mainly on the age of the patient and the degree of walking power attained. Bow-leg is the rule in younger children, as at this age the gait is rather rolling or waddling than ordinary walking. A constant effort to avoid falling forwards exerts a strain on the lower third of the leg, which accounts for the anterior curvature of the bones, while a similar corrective lateral effort to preserve the centre of gravity of the body leads to the accompanying external bend, producing bow-legs (Volkmann). The outward curve of the leg may reach such a degree that the external malleolus rests on the ground, the foot being strongly inverted; and in cases of extreme anterior curvature, the foot may need to be so much flexed as to touch the front of the tibial curve when the child is in the erect position. In older children, in whom ordinary walking has been acquired, the development of genu valgum is more common; and this is to be explained on the stand-at-ease theory, the weight of the trunk being transmitted through the outer femoral condyle of the limb on which the body mainly rests: growth is arrested at the outer segments of the femoral and tibial epiphyses, while it occurs freely at the inner segments. In rickety genu valgum the change may be almost equally developed in the two bones, or may be more marked in the tibia; and in these circumstances a definite angular projection or a prominent bony growth—not unlike an exostosis—sometimes emphasises the position of the tibial deformity.

Symptoms.—The early symptoms of rickets are insidious and ill-defined; and infants most frequently come under the notice of the surgeon, in consequence of inability to sit upright, backwardness in walking or standing, or the loss of previously-attained power in these respects. Such infants are often apparently well-nourished, but on closer examination prove to be fat and flabby, the rosy shining cheeks being due to the presence of dilated capillaries. The skin is often glazed and shining, sometimes clothed with very fine scales, especially if sweating is profuse. In other cases anæmia and emaciation are well marked. The child is restless and sleeps badly.

General tenderness is evidenced by the tendency to throw off the bed coverings, and local tenderness may be deduced from watching the children. Cases of severe craniotabes often sleep only when sitting up in the arms of the mother, so that the head is relieved from pressure; and older children often lie on their stomach, resting the less tender frontal region on the pillow. Again, lifting the child by the hands placed round the chest often produces evident suffering. In many cases, however, these local signs are insignificant. The head sweats profusely, often soaking the pillow, while the greater part of the trunk and extremities remain dry. At times eruptions of sudamina are seen, especially on the flexor aspects of the limbs. The general temperature is, usually, little raised. General pallor of the mucous membranes is common.

In some cases, gastric catarrh, accompanied by flatulence, vomiting, and diarrhœa, the stools being clay-coloured and offensive, are prominent symptoms; but in others digestion is apparently good.

The urine contains an excess of phosphate of lime, which may be increased by the use of the earthy phosphates medicinally. The spleen and liver are occasionally much enlarged. Bronchial catarrh is a frequent complication; and this, especially in conjunction with other infantile diseases, is the most common cause of death in rickety children.

Rickets is also the most common cause of laryngismus stridulus, and a fertile source of infantile convulsions. The varying deformities resulting from the disease have already been dwelt upon, and careful inspection and palpation will reveal them in varying stages of development. Signs are usually earliest in the skull, then in the thorax and later in the spine and extremities. Enlargements of the epiphyses in the upper extremity—particularly at the lower end of the radius—are more marked than in the lower; on the other hand, marked deformities from bending and unequal growth at the epiphysial lines are more common in the lower extremity.

Diagnosis.—In the early stages, especially when craniotabes is a prominent symptom, care must be taken to discriminate rickets from infantile syphilis. The history, both parental and of the child, together with the possible presence of co-existent undoubted signs of syphilis, usually allows a correct diagnosis to be made. The apparent prominence of the head occasionally leads to suspicion of hydrocephalus; while loss of power, more or less sudden, in one or more limbs may suggest infantile paralysis. Careful inspection and palpation of the head and epiphyses is the best safeguard against error in such cases.

The frequency of rickets as a cause of laryngismus stridulus must be borne in mind in any case of difficult breathing of a laryngeal nature.

Prognosis.—The disease is rarely fatal in itself, but many children succumb to the commoner complications or intercurrent diseases. The amount of permanent deformity left varies much in degree, in accordance with the severity of the attack, the efficiency of the treatment adopted, and also in the different bones affected. Consecutive growth tends to reduce curvatures of the long bones, so that at seven or eight years they may be hardly apparent; and, considering the frequency of these deformities, it is very striking how few instances of a severe nature are met with in adult life. Severe knock-knee, or bow-leg, or general stunting in growth may persist, even when great muscular power is regained. In the spine and pelvis the possibility of relieving the bones from pressure is less easy, and well-marked deformities of these parts are more likely to be permanent. The importance of contraction of the female pelvis in parturition has been already referred to.

Treatment.—Rickets, even when severe, is readily cured by suitable feeding of the child. If the mother be unable to afford adequate nourishment, cow's milk should be substituted up to seven months. This should be mixed with a third part of water, and a little sugar added. Later the milk may be unmixed, and eggs, a moderate amount of farinaceous food, and pounded meat, or raw meat juice, may be added. Cod-liver oil, or fresh cream (Cheadle) and vinum ferri, may be given medicinally. The administration of phosphate of lime is of little use, being mainly excreted with the urine—a proof that want of power to assimilate the lime salts is rather the cause of defective ossification than deficiency of lime salts in the diet.

The condition of the muscles may be improved by massage; tepid salt baths may be given, and the child should be clothed in flannel, so arranged that it cannot be thrown off at night. Every effort must be made to improve the hygienic surroundings of poor children; in the better classes, bracing air and the sea-side may be recommended.

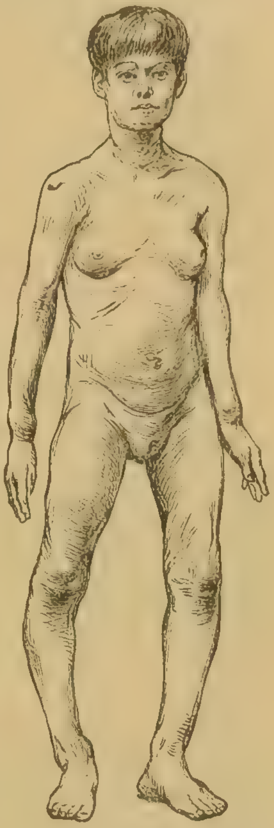
Fig. 77.—Late Rickets. Female. Aged 12. (Clutton's case.)

The first point in the treatment of the various deformities is the prevention of their increase. Thus, infants with a weak spine must be kept as much as possible in the supine position, those with bent fore-arms must be prevented from crawling, and those with bent legs from walking or standing, except in moderate amount. If the curvatures are severe, splints should be applied to bow-legs on the inner, to knock-knees on the outer, side of the limb. In severe cases below six years of age osteoclasis may be indicated if all signs of active disease have disappeared; when this is necessary, it should be effected by consecutive attempts with moderate force, made on the same occasion, rather than by one severe effort. After six years of age osteotomy may be called for.

Late rickets.—The condition known under this name differs in no essential particular from the infantile disease. In Drewitt's case examination of the bones showed the subperiosteal and epiphysial changes to be identical. The causation is not so distinctly to be traced, but there is much reason to believe that the disease is merely a recrudescence of an earlier attack which has passed unnoticed. As has been pointed out by Clutton, the cases differ from those in which bending of the bones occurs between the ages of six and nine, since in the latter the history of rickets is a continuous one. The bending of the bones also differs from that observed at puberty or later, as in the so-called adolescent knock-knee and bow-leg, since it is accompanied by the enlargement of the epiphyses observed in infantile rickets (Fig. 77).

Late rickets usually comes under observation in children from nine to thirteen years of age. The curvatures and epiphysial enlargements are more striking in the lower extremity, but the whole skeleton may be involved.

As in the infantile disease, the treatment depends entirely on attention to diet and the administration of cod-liver oil and iron. The deformities must also be treated on the same principles.

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Drewitt : Pathological Society's Transactions, vol. xxxii., 1881, pp. 387, 391.
Lucas : *Lancet*, vol. i., 1883, p. 993.

Scurvy rickets.—This disease consists in a combination of the two affections, and is of especial interest from the support it gives to the theory of improper feeding as the causation of rickets. The want of milk, poor cow's milk, or artificial foods, which give rise to rickets, are equally deficient in the anti-scorbutic elements necessary for a perfect food, and hence are responsible for the two conditions. The disease is more common among the children of well-to-do parents, from the fact that these are more often hand-fed with patent foods. (Barlow.)

The pathology of the disease is that of rickets *plus* scurvy ; and this is evidenced by the presence of more or less extensive subperiosteal hæmorrhages, and hæmorrhages from mucous surfaces, into the muscles and beneath the skin. A special peculiarity of scurvy rickets is the tendency to separation of the epiphyses : an accident not observed in pure rickets, though not uncommon in the epiphysial disease of infantile syphilis. The disease is most commonly met with in children from ten to twelve months of age. According to Barlow, it may arise at any period after four months, and especially between nine and eighteen months.

Symptoms.—The onset of the disease is an acute one. The child becomes ill, and in a few days the limb or limbs swell and are excessively tender to the touch, and the temperature may rise to 101° Fahr., or more. The commencement is often heralded by the appearance of ecchymoses beneath the conjunctiva, the skin of the eyelids, or of the limbs. Proptosis from hæmorrhage beneath the periosteum of the orbit has been observed in a considerable number of cases. In some cases subcutaneous discoloration of the affected limbs is a prominent symptom. Beyond this, in about half the cases, the gums are spongy and bleed readily, the condition, however, affecting only those parts of the alveolus from which the teeth have already protruded (Barlow). Bleeding may occur from the nose, rarely from the rectum, and hæmaturia is not uncommon.

Tenderness is evidenced by the disposition of the child to keep the limbs so still as sometimes to simulate paralysis, and by signs of great pain when the limbs are handled. The lower limbs, and especially the femora, are most frequently affected; but the condition may be more or less general, involving the scapula, upper limbs, and thorax. Sinking of the sternum *en bloc*, due to separation of the ribs from the costal cartilages, is of such comparative frequency in this disease as to be in some degree pathognomonic (Barlow). The swelling of the limbs usually commences in the neighbourhood of the epiphyses; it may be limited, but more commonly involves the whole segment corresponding to the affected bone; it is firm and tense, not pitting on pressure. The limb is white, except when ecchymoses are present, and hot to the touch. Occasionally separation of the epiphyses occurs, or fracture of the shafts, and then soft crepitus is elicited on movement.

The disease is readily recognised by attention to the symptoms of scurvy. It is accompanied by symptoms of severe illness, great anæmia, and rapid emaciation, and may terminate fatally; relapses may occur or the course become chronic. When uninfluenced by treatment the average duration is from two to four months (Barlow).

Treatment consists in the same attention to diet that is needed in rickets, lime-juice or fresh orange-juice being added. The limbs affected should be treated with cooling lotions and supported by bandages; if separation of the epiphyses or fracture occurs, splints are necessary.

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T. Smith: Pathological Society's Transactions, vol. xxvii. p. 219.
Cheadle: *Lancet*, vol. ii., 1878, p. 657, and vol. ii., 1882, p. 48.
Barlow: Medico-Chirurgical Society's Transactions, vol. lxvi. p. 159, and *Lancet*, vol. ii., 1894, p. 1075.

XVIII. HÆMOPHILIA.

By FREDERICK TREVES, F.R.C.S.,

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HÆMOPHILIA, or the "bleeder disease," is defined as a congenital and habitual hæmorrhagic diathesis. The subjects of it are liable to severe and obstinate hæmorrhages, which may occur spontaneously, or may follow injuries, often of the slightest kind.

General phenomena.—The disease is noticed in the patient from the commencement of life; it may be said to exist so long as he exists, and does not appear (except in some rare and imperfectly understood instances) to be ever acquired.

The malady is in nearly every case *hereditary*. Grandidier speaks of it as "the most hereditary of all hereditary diseases," and the subjects of it will generally be found to belong to definite "bleeder families." It attacks the individual members of a family extensively, and it is estimated that there will be three bleeders to every family affected. Hæmophilia is nearly twelve times more common in *males* than in females. The disease assumes a much less typical form in women.

The disease is *transmitted* in a remarkable way. It is handed down, not by the bleeders themselves, but in nearly every instance by the non-bleeder members of the family, and almost exclusively by the females. Thus, if there be six children in a bleeder family (three boys and three girls) the boys will be bleeders but the girls most probably will not be affected. If they all marry and have children, the children of the bleeder males will most probably be non-bleeders, whereas the children of the females will almost certainly present the disease.

Hæmophilia has a peculiar *geographical* distribution. It is common in Germany, and many cases have been recorded in Great Britain, in North America, in Norway and Sweden, and in France. The disease does not appear to be met with in Italy, Greece, Turkey, Spain, or Portugal. It is said to be very common among the Jews.

Bleeders, apart from their special diathesis, present no distinctive constitutional condition. There is nothing peculiar in their physique, their complexion, their pathological tendencies, or their general health. They frequently possess a very fine and transparent skin, but this feature is by no means constant. If not suffering from the effects of hæmorrhage, they may appear to be in robust health.

The actual *cause* of the disease is entirely unknown and is merely a matter of speculation.

Its *pathological anatomy* is no more precise. In many fatal cases of hæmophilia there has been no evidence of a primary disease and no abnormality has been detected in the vascular system. In other instances the heart, and especially the left ventricle, have been found hypertrophied, and the arteries to be abnormally thin, the atrophy involving especially the intima. No peculiarity has been detected in the blood.

Symptoms.—The hæmorrhages are usually due to injury. Fatal bleeding has followed scratches with pins, the removal of teeth, leech bites, the rupture of the hymen, circumcision, and the most trifling wounds, as well as lesions of greater magnitude.

It is remarkable that serious bleeding very rarely follows the wounds made in vaccination.

Continuous and even quite slight pressure may cause very extensive subcutaneous hæmorrhages. Some of these extravasations may attain great size. Some may occur spontaneously and are then most often met with on the scalp or about the genitals.

Spontaneous hæmorrhage is usually from a mucous surface, most commonly from the nose, and then in order of frequency from the gums, the alimentary canal, and the genito-urinary passages. Fatal hæmaturia is exceedingly rare. Hæmorrhage is rare from serous surfaces, although bleeding into joints is not at all uncommon. Traumatic bleeding increases the tendency to spontaneous bleeding.

If the bleeding part can be examined the blood will be found to ooze from numerous points, and not spout from one or two vessels. The blood poured out presents no peculiarity of any kind. The hæmorrhage is never furious, but it may continue for many days and even for weeks.

Bleeders will bear great losses of blood remarkably well and will recover very rapidly when the hæmorrhage has ceased.

The symptoms differ in no way from the ordinary phenomena of prolonged bleeding as observed in the otherwise healthy.

The bleeding is nearly always capillary. The quantity lost may be enormous and the resulting anæmia is marked and of long duration.

The tendency to bleed is more pronounced at some periods than at others, so that at certain times wounds in bleeders may be attended with only the normal amount of hæmorrhage. The precise nature of these periods is quite unknown.

Some patients exhibit every kind of bleeding, spontaneous and traumatic, superficial and interstitial. These are mostly men. Women seldom exhibit this excessive form of the disease. Other patients are subject only to hæmorrhages from mucous surfaces. A third series appear to be only liable to spontaneous ecchymoses.

The affection usually shows itself at the beginning of the first dentition and at puberty. It is very rare for bleeding to be noticed at birth. The manifestations decline in advancing years, so that the disease is more marked in youth than in middle life; 70 per cent. of the first outbreaks fall within the first five years of life.

Often in spontaneous bleeding there will be prodromata in the forms of flushings, a sense of heat, beating of the arteries, and restlessness.

One peculiarity remains to be noted. There is a marked association between hæmophilia and rheumatism. Bleeders are very liable to painful swelling of the joints and to muscular pains. The nature of this association remains unexplained.

The swelling of the joints is very commonly, but not always, due to extravasation of blood within them. The knee is the articulation most commonly affected. This joint complication is quite uncommon in female patients.

Mortality.—The disease, when marked, is very fatal. Most of the patients die young, and it is stated that more than 50 per cent. never reach the age of seven years. The first bleeding may prove fatal, or a great number of hæmorrhages may occur before death. On the other hand, a patient may bleed once, recover, and never bleed to excess again. A few bleeders have attained old age.

Treatment.—In the matter of treatment nothing, so far as is known, can be done to prevent bleeding in a known bleeder, apart from keeping him from risks of injury. In cases of spontaneous internal hæmorrhage the patient should be kept at rest in the recumbent posture, the bowels may be made to act vigorously if the hæmorrhage be not from the intestine; all stimulants must be avoided, and the patient treated with acetate of lead, in full doses, or with ergotin or sclerotic acid.

Transfusion has proved of little avail, and indeed, when it has been performed, the bleeding from the operation wound has sometimes added to the patient's danger.

In cases of traumatic hæmorrhage the oozing may be best checked by a tampon soaked in perchloride of iron and applied so as to exercise pressure on the part. This is certainly the most efficient local measure. The actual cautery is seldom of much avail, and acupressure and the use of ligatures are apt to add fresh risks to the case by increasing the wound. Ice may have a good effect for a while, but on its removal the hyperæmia that follows the use of cold may lead to worse bleeding than ever. In any case acetate of lead, ergot, or sclerotic acid should be administered.

Very often these drugs—and many others that have been advised—have no effect.

In the general treatment of the patient, calcium chloride has been recommended, on the supposition that it may render the blood more coagulable; and also camphor and turpentine, from their supposed power of increasing the number of the white corpuscles of the blood.

XIX. HYSTERIA IN ITS SURGICAL RELATIONS.

By FREDERICK TREVES, F.R.C.S.,

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Definition.—The term "Hysteria" is applied to a functional disorder of the nervous system, to a general neurosis that is difficult to define, but that may be described, if prominence be given to one of its primary features, as a condition of increased irritability to physical and psychical stimuli, or as a condition of general nerve instability as marked by enfeebled powers of inhibition and control. It is a disease without a pathological anatomy. From the origin of the term (Greek, ὑστέρα, the womb) it is only to be inferred that the disease is much more common in women than in men, and that it is often induced by some uterine or ovarian disorder. It is probable, however, that in at least fifty per cent. of the subjects of hysteria these organs are perfectly sound. Hypochondriasis in the male is very similar to, if not identical with, hysteria in the female.

Age.—Hysteria most commonly begins between the ages of fifteen and twenty. It may develop, however, at any time during the period of active adult life and not infrequently breaks forth at the climacteric.

The hysterical woman.—There is nothing distinctive about the appearance of the hysterical woman. She may be young or old, or plump or thin. She may be of a ruddy and cheerful countenance, or may be wan and sallow, and in a constant state of flabby woe. She may be a member of what is vaguely termed a "neurotic" family. Some of her relatives may be the subjects of epilepsy or insanity, or may be dipsomaniacs, or notoriously "nervous" individuals. She may, on the other hand, be the only individual in a family to present any nervous phenomena.

The hysterical woman is, in a definite sense, irritable. Stimuli act upon her nervous system with undue and uncertain vigour, and the effects, moreover, are apt to spread. Her emotions tend to become exuberant, to be distorted, or at least to be inconstant. Over all there is a weak mental control, a lack of attempt to limit the effects of normal stimuli; if not a constant lack, at least an occasional lack. She is morbidly sensitive, and presents an extravagant consciousness of self. She is oppressed by her surroundings, and judges of circumstances by the effects they produce upon herself. There is usually some bodily ill-health at the bottom of hysteria, possibly some uterine disturbance, more often some anæmia, and perhaps most often chronic dyspepsia. The subjects are usually weak, easily tired both in brain and body, and possessed by a frame of mind that is the reverse of self-reliant. In many cases the patient's surroundings are unfortunate, her home life is miserable, and her troubles have been numerous. In not a few of the examples of hysteria in women past middle life, there may be the elements of excessive drinking.

Symptoms.—The symptoms of hysteria may develop suddenly after some great grief or personal calamity. More often they appear slowly. Profound emotional disturbances do not so often appear to lead to hysteria as do petty forms of irritation that are constantly repeated. Worry, the continual dropping of water upon the mental stone, is a great cause of the hysterical state.

The symptoms of the affection need not be dealt with in detail and can only be briefly tabulated in this place. The features of the hysterical "fit" are familiar.

Sensory disturbances.—Hyperæsthesia, intolerance of light or of sound, intense dislike to certain odours and articles of food; neuralgia, often involving the muscles, and very commonly the breast and intercostal nerves; headache, hemicrania, "clavus hystericus" (a defined gnawing pain about the top of the head, near the sagittal suture or over one brow); abdominal pains (often in the epigastrium) which may be attended by symptoms imitating ulcer of the stomach, peritonitis, or ovarian disease. Anæsthesia, as shown by sensory paralysis of certain tracts of skin, and by hysterical blindness and deafness.

Motor disturbances as illustrated by paraplegia and hemiplegia, by palsy of the bladder leading to incontinence; of the gullet, leading to dysphagia; of the bowels, causing tympanitis and constipation, and of the vocal cords producing aphonia. Muscular tremor, muscular spasms, contracted limbs, strabismus, etc., local or general convulsions. "Globus hystericus": a sense as of a ball rising from the abdomen into the throat, where it appears to become impacted, a condition supposed to be due to peristaltic contraction of the gullet.

Vascular disturbances, as shown by tumultuous or irregular action of the heart, fainting fits, congestive headaches, exaggerated blushing, extremities that may be at one time burning and at another extremely cold; bloodlessness of certain parts of the surface, so that a pin-prick is not followed by bleeding; disordered menstruation; irregularity in the quantity of urine excreted, etc.

Neuromimesis.—The phenomena of hysteria that mostly concern surgical practice are those by which the features of certain diseases are reproduced. To these phenomena the term of Neuromimesis has been applied. This mimicry of disease may be very complete. The mimicry is no doubt unconscious, if instances of deliberate malingering be excluded, and it is usually founded upon some trifling local disturbance. Dr. Weir Mitchell reports the case of a hysterical girl in whom was reproduced a mimic cerebro-spinal meningitis, after she had been fatigued by nursing two patients with the actual disease. Probably her head ached and her back ached because she was tired out, and upon the foundation of these natural symptoms was soon built up the phantom malady.

It is to be noticed that in these mimicked diseases the patient has often a knowledge of the real disease in the person of some relative or friend.

Hysterical joint.—The symptoms of acute joint disease are mimicked; the parts most commonly involved are the hip and the knee, and the manifestations are often preceded by some trifling injury to the limb. The joint, to take the knee as an example, is very painful, is stiff, is becoming flexed, and cannot be moved. Examination will show that the symptoms complained of are quite out of proportion to the physical evidences of disease. The pain is "excruciating" or "agonising," yet there is no effusion into the joint and no swelling of the bones. The pain also varies, and is evidently in the main due to an exceedingly sensitive condition of the skin. It is a surface pain and not a deep-seated one. The painful spot is commonly between the femur and tibia, and if it be cautiously approached while the patient's attention is diverted, it may often be pressed vigorously upon without producing any outcry. There is an absence of the familiar "starting pains." Inter-articular pressure causes no inconvenience. If the patient's attention be absorbed, the joint may often be moved to a considerable extent, both actively and passively. Under chloroform all the stiffness at once disappears. In the case of the hip the position assumed by the limb is often quite incongruous, and the deformed attitude is lost when the patient is asleep or believes she is not observed. In time some swelling may appear about the joint, but it will always be in the subcutaneous tissue, and not in the articulation.

The skin will probably be cooler than normal, but fluctuations in the surface heat are often noticed, and depend upon local vaso-motor disturbances. Thus Dr. Mitchell reports a case where the surface temperature of a hysterical knee was normal in the morning, but rose in the evening to 99, 100, or 101 degrees. The temperature of the other or sound knee was always normal.

After long-continued disuse the muscles of the limb will waste, and the features of the actual disease be even more closely imitated. The appearance of real disease is often encouraged by the treatment, as by continued blistering and frequent painting with iodine.

Hysterical spine.—In this condition spinal caries may be mimicked. There may be a history of some slight injury to the spine, or the back may be painful from muscular weakness, or there may be a trifling degree of lateral curvature. The mimic disease is common at the root of the neck in the region of the vertebra prominens, and is also frequent in the lumbar spine. Intense pain is complained of, but it is quite superficial, and pinching the skin causes more distress than pressing the bone. The patient will resent forcible moving of the part, but no rigidity, no deformity, and no thickening are to be made out. A jar to the column will not cause pain. In some cases there may be paraplegia, which will present the characters of the hysterical form of that affection.

Phantom tumours are produced by an abiding spasm of a muscle or part of a muscle. By the contraction of the fibres a fairly defined swelling may be produced. This is most often met with in one of the compartments of the rectus abdominis, and may be attended by some of the abdominal symptoms that are not uncommon in hysteria. Dr. Weir Mitchell gives accounts of like tumours in the calf and in the pectoral region. These swellings can be made out to be muscular; they vary in size, they are apt to disappear, and entirely vanish under an anæsthetic and during sleep.

Abdominal troubles.—These are usually very incongruous. In not a few instances the symptoms of ulcer of the stomach are fairly imitated. In others there is a distinct dilatation of the stomach, with constant eructations. Unaccountable vomiting, flatulent distension of the intestines, obstinate constipation, a fitful diarrhœa, and more or less "violent" colic are all fairly common. (*See* page 383.)

In one case under my care the abdomen was opened because the symptoms complained of closely resembled those of stricture of the small intestine. The bowel was found to be normal, but all the symptoms vanished after the operation.

Retention or incontinence of urine in the hysterical may be recognised by the general symptoms presented by the patient, and by an absence of any of the physical conditions that would cause the complication.

Treatment.—The general treatment of hysteria concerns the physician rather than the surgeon. It will here be sufficient to say that any probable cause of the disease should be treated, uterine troubles relieved, dyspepsia treated, and debility dealt with by the usual means. Drugs, such as valerian and asafœtida, are of little use, but bromide of potash is of value when there is much restlessness, and when the patient is distressed by the nerve pains.

In obstinate cases no measures succeed so well as those adopted by Dr. Weir Mitchell:—(1) Perfect seclusion, so enforced that the patient is removed from all home influences, and from the attractive halo of indiscreetly sympathetic friends. (2) Perfect rest both of brain and body. (3) Such moral influences as may induce the patient to recognise the true nature of her ailment, and to aid treatment by rightly exercising again a long dormant or perverted will. (4) Passive exercise in the form of massage, or "muscle kneading." This shampooing of affected parts should be conducted from thirty to sixty minutes daily, and, as Dr. Mitchell expresses it, acts as an admirable "mechanical tonic." The induced current may also be used for the same ends.

In some cases of hysterical joint an immediate cure has followed a rough and sudden flexing of the stiffened limb or the application of so strong an electric current as to cause the patient to exert all her powers to prevent a threatened renewal of the treatment. In most of these mimicked joint affections massage and passive movements have good effect.

The hysterical spine is less easily managed and many patients have been bedridden for years with this affection.

The success of treatment in any of these cases will depend more upon common sense and good judgment than upon any precise and formal line of treatment.

Hysteria in the male.—Conditions are occasionally met with in the male subject which so nearly resemble hysteria in the opposite sex that—in spite of the absurdity of the term when its etymology is considered—they may be conveniently termed hysterical.

So far as the mimicry of disease is concerned, the conditions which are most frequently met with in males may be classified under two headings:—(1) Abdominal neurosis and (2) sexual neurosis.

(1) *Abdominal neurosis.*—The patient who exhibits this condition is most usually the victim of chronic dyspepsia. He becomes hypochondriacal and melancholic. He is usually constipated, and the whole interest of his life is apt to be centred in the action of his bowels. He takes aperients without stint and inspects his motions with horrible care. Sometimes he has a dreadful gnawing pain in his belly, which he is convinced is due to cancer. In other cases the stomach is dilated, eructations are continuous, vomiting may be frequent. In one or two reported examples of intestinal neurosis the vomiting was fæculent. Flatulent distension of the abdomen is common. In any case the utter misery and despondency of the man are very noticeable and contrast with his usually well-nourished condition. He is sometimes the member of a family prone to insanity. He may have been greatly overworked or greatly worried. He may have very defective teeth. He may have injured his health by excessive drinking. He speaks of his symptoms with extreme exaggeration.

Rest, dieting, sedatives, massage, and quiet will cure some cases. Electricity is useful in others. Complete change of life and scene will dispel the troubles of a third class of patient, but there are some who appear to be really hopeless. I have met with a case in which all the symptoms disappeared on the passing of a tapeworm, and another in which the securing of a long unrecognised movable kidney put an end to symptoms closely resembling stricture of the pylorus. In one or two instances of the worst kind I have carried out an exploratory incision into the abdomen. In no case was anything abnormal found and in no case did the symptoms return.

(2) *Sexual neurosis.*—The basis for the familiar symptoms of these protean troubles is commonly a slight degree of stricture, a

varicocele, or a chronic prostatitis. In some instances there is an impression in the patient's mind that he is impotent, and that he has fallen into the parlous condition so graphically described in the pamphlets of the quack. In others he declares he has a "seminal discharge," which, however, can commonly be shown not to be semen. In certain examples the neck of the bladder is the seat of the nervous phenomena, and these cases are, perhaps, the most troublesome of all. When these patients come under the surgeon's notice, it is probable that they will have already used every form of urethral injection, will have had a cystocope passed, and will have been sounded for stone. They will also have taken sea voyages. The basis of their trouble is, as already stated, often a slight degree of chronic prostatitis combined with a morbidly sensitive mind and a debilitated nervous system. The treatment of these cases must be left to common sense rather than to surgery.

XX. SYPHILIS.

By JONATHAN HUTCHINSON, Jr., F.R.C.S.,

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Syphilis is a disease confined to the human subject,? but met with in all parts of the globe, due to the entrance of a virus (the nature of which is not yet conclusively proved) into the system, either by direct inoculation or by inheritance—*i.e.* transmission from either father or mother to their child. In some points it resembles a specific or exanthem fever, but the stages are comparatively very protracted, and its remote effects far more persistent and important. There is a certain amount of evidence to point to the virus of syphilis being a very small bacillus, but we cannot at present go beyond this vague statement, and even on this point the results of various investigators have been either negative or very doubtful. (*See* page 37.)

ACQUIRED SYPHILIS.

The stages of syphilis.—We will first consider acquired syphilis, leaving till later any notice of the symptoms of the congenital or inherited disease.

After inoculation (generally by venereal contact, but also in a host of other ways) a variable *period of incubation* occurs before the primary sore or chancre appears. By the end of two, three, or four weeks the characters of the chancre are usually such as to suggest the strongest suspicion of syphilitic infection to a skilled observer: at this date the nearest lymphatic glands become enlarged. The so-called *primary stage* dates from inoculation up to the appearance of various symmetrical affections, which indicate that the poison is pervading the whole system. Chief of these are eruptions on the skin and mucous membrane of the throat, bone and joint pains, general enlargement of lymphatic glands, and thinning of the hair. With the earliest of these symptoms (usually at the end of six to eight weeks from the date of contagion) begins the *secondary stage*, which may roughly be said to last for two years. During

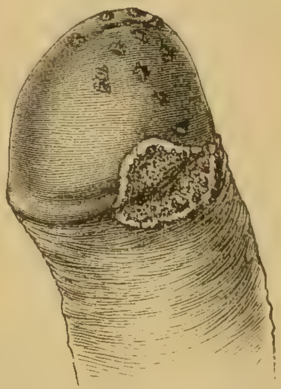

Fig. 78.—Chancre of Penis, with Herpes of Glands.

both these stages the disease may readily be transferred from the patient to others through any sore which may exist, and perhaps, under exceptional conditions, through inoculation with various normal secretions. The virus undoubtedly may retain its power for a day or two (possibly considerably longer) after it has left the subject from whom it was derived, and a single individual with syphilis may transmit it almost indefinitely, though the risk of contagion grows much less after the secondary stage has been passed through. Unfortunately the possibility of inheritance of syphilis may extend for many years after the primary disease in the parent (in some cases five, ten, or even more years); though the longer the interval the less severe will the symptoms in the child probably be. Following the secondary comes the *intermediate stage*. (*See* page 406.)

The *tertiary stage* includes the appearance of various lesions of the bones, viscera, skin, and nervous system, etc., which are much more persistent and serious than those of the secondary ones. They are generally unsymmetrical, or, at any rate, if occurring on both sides of the body, they do not present exact symmetry. Although remarkably amenable in most cases to treatment by iodides and mercury, the tendency of many of the tertiary lesions is both aggressive and destructive. The tertiary stage rarely commences before two years have elapsed from infection, but it cannot be said to have any other time-limit. It must not be supposed that every case of syphilis goes through a regular series of symptoms which can be grouped into the three stages given above. If treated efficiently and for a long period, there is often a complete exemption from any tertiary symptoms whatever, and if mercury be begun very early (of course, in cases where the diagnosis of primary syphilis can be positively made) the secondary symptoms also may be prevented or greatly modified from their usual date of occurrence and intensity.

Besides the all-important influence of treatment just mentioned, we must lay stress on the fact that the severity of the disease appears to differ considerably in different individuals, for reasons we can rarely even suggest, but which are referred to "idiosyncrasy." That syphilis is nowadays a much milder affection than in the Middle Ages is a very prevalent belief, without the slightest foundation, except in so far that the treatment of it has immensely improved.

PRIMARY SYPHILIS.

A question of some interest is from what source the contagion is derived in primary venereal chancres. It may be affirmed without hesitation that as a rule the *source of contagion* is some moist or secreting secondary lesion, of the nature of a condyloma. Primary chancres in women, although very contagious whilst they last, do not often persist more than a few weeks, if so long; whereas a condyloma of the vulva may last almost indefinitely if not treated, and its capacity for infection prove almost unlimited. It may also be noted that both primary chancres and mucous patches have been occasionally observed on the cervix uteri.

The primary lesion.—We will now consider the primary lesions in the male. For obvious reasons the site of the chancre is usually on the genitals, and here inoculation is especially favoured by the delicate nature of the cutaneous surface. The space between the glans penis and the prepuce is very apt to harbour the virus, a fact forming one out of many reasons for the practice of circumcision at an early age.

From 60 to 70 per cent. of chancres in the male are situated on the inner aspect of the prepuce, the retro-preputial fold, or on the glans penis. Often, owing to congenital phimosis, or to swelling of the prepuce, due to the irritation of the discharge, etc., it is impossible for the patient to expose the sore; but the induration to be felt through the foreskin leaves little doubt in such cases as to the nature of the concealed chancre. The meatus is by no means an uncommon site for primary sores, which nearly always occur in the form of a ring round the urinary opening. Their cicatrisation is then apt to leave a stricture, but this sequela is not constant. Second in frequency come chancres of the skin of the penis on its outer aspect, whilst they are occasionally seen on the pubes, the inner side of the thigh, the scrotum, etc. True urethral chancres—*i.e.* those occurring within the meatus—are excessively rare.

It is a mistake to suppose that the syphilitic virus always obtains access at the moment of contagion through a crack or abrasion of the skin; a perfectly unbroken surface of delicate epithelium will allow of its inoculation, and this holds true in the case of chancres of the conjunctiva as well as of the penis, etc. Where the skin is harder and thicker—as, for instance, on the fingers, cheeks, etc.—it is perhaps necessary for infection with syphilis that some breach of surface should exist. Thus, many cases have occurred through tattooing, from bites by the human subject, and through the practice of kissing when a crack exists on the lips.

Signs of infecting chancres.—After exposure to syphilitic contagion, there may sometimes be noticed an abrasion which heals, subsequently indurating and developing into a true chancre; in other cases one or more soft chancres may be present and follow the same course, but in a large proportion of cases the patient's attention is first called to the chancre at the end of three or four weeks, and on his then presenting himself to the surgeon all the characteristic features of a primary infecting sore may be present (Fig. 78). The *chancre* is well defined, raised, slightly ulcerated, and secreting a little thin discharge, rounded or oval in outline, and usually painless. When grasped between finger and thumb, a hardness, varying in degree up to that of firm cartilage, is detected; but the amount of induration will depend somewhat on the site of the lesion. It is most marked in chancres of the inner aspect of the prepuce, especially at the retro-preputial furrow; on the glans penis it may be but slight and of such little depth as to suggest parchment. It must be distinguished from mere inflammatory infiltration, and occasionally a deceptive hardness is produced in a soft chancre by cauterisation with nitrate of silver.

Within a very short time the inguinal *lymphatic glands* enlarge and form what is sometimes termed the bullety or amygdaloid bubo. All the glands in the oblique set along Poupart's ligament (especially those nearest to the penis) increase in size; the bubo is painless, or almost so, adherent neither to the skin nor to the deep tissues, and very persistent. Even if mercury be given early, it may be many weeks or months before the glands return to their normal size. The highest glands of the vertical set are also sometimes enlarged. It is a curious fact that often the chancre is on one side of the genitals, the bubo on the other, no doubt owing to crossing of the lymphatic vessels. It should be noted that in a considerable number of cases (though certainly not the majority) the dorsal lymphatic vessels can be traced up from near the chancre to the root of the penis, or even to the glands themselves, as hard cords. These indurated lymphatics are of some value in diagnosis, as they rarely or never occur except in cases of syphilitic infection. Running from a chancre of the finger I have been able to trace them up to the axilla. The glands in each groin may enlarge equally, and sometimes attain a great size—up to that of a goose's egg. It is, however, important to remember that the normal inguinal glands, if the patient be not stout, can readily be felt; and the younger the patient the more distinct are they likely to be. Again, owing to some non-venereal irritation—such as the pressure of a truss—a man may have his inguinal glands persistently enlarged.

Following extra-genital chancres we find constantly induration and swelling of the nearest lymphatic glands. Thus, with a chancre on the lips or cheek, there will be a sub-maxillary bubo; on the conjunctiva a pre-auricular one with, as a rule, swelling of the glands below the jaw; a chancre on the fingers is attended with an axillary bubo (and in the case of the sore being on the inner fingers, an epicondylar one), etc. It seems to be the fact that the bubo caused by the extra-genital chancres is more often inflamed and painful than when due to genital infecting sores, but indolent painless enlargement is not uncommon in the former case.

Is every infecting chancre attended with gland enlargement? Cases where the latter is absent, or where it cannot be detected, do undoubtedly occur, but they are so few that they may practically be disregarded.

Sometimes the chancre itself is very insignificant—merely a small indurated papule—at others it is a large deep ulcer, with foul-smelling discharge; occasionally we see a complete collar-like ring of induration around the base of the glans penis.

In women the primary lesion is usually on one or other of the labia; it has been detected on the os uteri, but curiously the true vaginal wall seems to be almost exempt. Amongst the less frequent sites of chancres we have to note the scrotum, perineum in both sexes, the anus, and the inner side of the thigh. When derived from accidental non-venereal infection, it would be almost impossible to mention any part of the body on which they have not been noted. I have seen one infecting sore on a boy's heel (no doubt from contamination due to dressing an abrasion from boot pressure), another on the vertex of a man's head. In wet-nurses the nipple and breast are favourite sites of inoculation; about the mouth chancres have been observed on the tongue, the tonsil, the pharyngeal wall, etc. It may be stated as being almost without exception, that all extra-genital chancres are syphilitic or infecting ones.

Indurated chancres are, as a rule, *single*, but by no means always so, for as many as ten or fifteen have been observed on the same patient, and two or three are not very uncommon. They are usually non-inoculable on the same patient, but this is, again, only partially true. Soft ones, however, can readily be inoculated, and nothing is more frequent than to see examples of this having occurred; as, for instance, from prepuce to glans or scrotum, etc. We have already noted that indurated sores are slow to disappear; in women, however, they would seem to have a shorter term of existence than in men, at any rate when situated on the genitals. It is important to remember that they often completely disappear, leaving no trace behind; and hence, when examining a patient with a doubtful history of syphilis, no great reliance must be placed on the presence or absence of a scar on the genitals. If a well-marked cicatrix be found, it is more likely to be the remains of an ulcerating soft sore than an infecting hard one.

Soft or non-infecting chancres.—A large proportion of sores on the genitals contracted from venereal contagion do not lead to syphilis, and (although this has been keenly debated) in all probability they have nothing whatever to do with syphilis. Considering the many varieties of micro-organisms which frequent the male and female external genitals, it is no wonder that suppurative lesions should be produced by sexual intercourse, apart from syphilis and gonorrhœa. The soft chancre (*chancroid* of the French writers) has the following characters:—(1) It has a very short period of incubation, a pustule forming within two to five days of contagion, and very rapidly developing into an open sore or ulcer; (2) it is nearly always multiple, thus there may be a ring of ten to thirty small ulcers round the coronal furrow or the preputial border; (3) it is invariably an open sore discharging pus, sometimes a very considerable amount; (4) it may cause no gland enlargement, or, on the other hand, one or more of the inguinal glands may inflame or suppurate as a result of it. The "bullety bubo" (5) is never seen apart from syphilis; if treated by strict cleanliness and an antiseptic powder, or if cauterised, it nearly always heals quickly; under other conditions it may persist almost indefinitely.

Around a soft chancre there may be a good deal of inflammatory thickening, but no true induration; but it must be noted that a soft sore may appear soon after inoculation, and after two or three weeks become indurated, and be followed by secondary syphilis. In this case, no doubt, two poisons have been introduced at the same time: the micrococcus of suppuration and the germ of syphilis (whatever that may be). The surgeon must exercise caution, therefore, in giving an opinion as to the nature of any venereal chancre until three or four weeks have elapsed from the date of contagion, though in most cases he can be fairly sure before this.

Phagedænic chancres.—When a chancre rapidly destroys the tissues, and spreads deeply into them, the term phagedæna is applied. Sharp bleeding is apt to occur from these sores, owing to the arteries being opened up; and the hæmorrhage, unless arrested by treatment, may prove very formidable, and occasionally even fatal. Phagedæna is especially liable to attack a concealed chancre far back under a tight foreskin, and may destroy the glans completely, or cause sloughing of the whole prepuce. The very foul-smelling, blood-tinged discharge from such a sore should excite suspicion in the surgeon's mind, and lead him to adopt the first essential step to its treatment—thorough exposure by slitting up the prepuce.

The slough should be cut away, and the actual cautery may require to be applied, in order to stop the hæmorrhage. In many cases, continuous immersion in a warm bath containing a very weak solution of bichloride of mercury, or some other good antiseptic, will stop the phagedænic process; but if this is not successful or practicable, the ulcer should be lightly cauterised with the acid nitrate of mercury. Particular care should be taken if the ulceration verges on the urethra. Iodoform should be freely applied in the intervals of the immersion (which to be effective should be prolonged to many hours a day). Acute phagedæna is rarely attended with any gland enlargement to speak of, and a considerable proportion of the cases do not afterwards develop secondary symptoms; others, however, suffer severely from syphilis. These latter cases are generally instances of comparatively slow phagedæna. In fact it is probable that the rapid destruction of the tissues is an accidental complication due to some virus other than that of syphilis, and having more alliance with that of noma, or of the gangrenous ulceration induced experimentally in rabbits, etc., by the inoculation of certain decomposing substances. However this may be, it is a clinical fact that acute phagedæna is usually accompanied by no bubo, and may be followed by no secondary symptoms. The term is sometimes loosely applied to any sore which tends to ulcerate deeply, but should be reserved for those in which the destruction is very rapid. (*See* page 413.)

Phagedæna may attack tertiary ulcers—*e.g.* of the buttocks and legs—but occasionally we meet with a similar complication in neglected non-syphilitic ulcers.

The diagnosis of primary or infecting chancres.—It may seem almost unnecessary to point out that the skin of the male and female genital organs is liable to various diseases of the general cutaneous surface. Psoriasis papules, inflamed sebaceous glands, boils, simple inflammatory œdema, eczema, lupus, etc., are amongst the non-venereal affections which are sometimes seen on the penis, scrotum, or vulva, and which may lead the unwary to an erroneous diagnosis. Epithelioma of the penis has often been mistaken for a syphilitic chancre. Cases of gummatous ulceration of the genital surface are frequent enough to deserve note, and probably closely allied to these are the cases of so-called relapsing induration. In this curious affection what happens is this: A man who has had secondary syphilis years before presents a slightly-raised induration on the penis, at or close to the site of his former chancre. If he has been lately exposed to venereal risk, nothing is more natural than for the surgeon to conclude it is a case of second infection. But there is no bubo, and no secondaries follow. Further, the induration may occur in those who have not been exposed to the slightest risk of a fresh attack of syphilis. That the lesion is of the nature of gummatous deposit is made almost certain by its disappearance under iodide of potassium. Although rare, the "relapsing false indurated chancre," which was first described by Hutchinson and Fournier, is well worth bearing in mind, as it may lead to curious perplexity in practice. The absence of gland enlargement, and the occurrence of the induration at the site of the old chancre, are the two features which help most in the diagnosis.

The treatment of chancres and the buboes which attend them.—Now and then it is worth while to attempt by excision, or by thorough cauterisation of a chancre, to prevent syphilitic infection. It must, however, be admitted that very few cases indeed are suitable ones for this method of treatment. If the sore be seen within a few days of known contagion, if it be situated on a part which can be easily removed—*e.g.* the end of the foreskin—and if there be as yet no gland enlargement, excision may be tried. It is, however, quite useless when there is already a bubo, or if more than a few days have elapsed from the date of contagion. Cauterisation with pure carbolic acid is to be recommended for most soft chancres, or doubtful ones, and when the sore is of unhealthy aspect and tending to spread, the surface should be cleansed, and either carbolic acid or some stronger caustic—such as nitric acid, or the acid nitrate of mercury—applied. The pain may be diminished somewhat by previously applying a strong solution of cocaine. Cleanliness should be enjoined in all cases of venereal ulcers. The sore should be bathed several times daily, and an antiseptic powder applied in the intervals. One of the best is a mixture of iodoform (one part) with boracic acid (three parts), finely powdered, and thoroughly mixed. Sometimes, even in this weak preparation, iodoform causes erythema and irritation, or its smell may be strongly objected to. Then calomel and zinc oxide powder is useful, or the sore may be kept moist with a strip of linen soaked in lotio nigra (calomel and lime water). As a rule, ointments do not suit chancres, which show little tendency to heal under their use.

Many syphilitic chancres are of the papular form, or occur as indurated plaques without any breach of surface, and then hardly require local treatment. If the induration be extensive, the quickest method of obtaining its resolution is to get the patient under the influence of mercury.

The treatment of phagedænic sores has already been described.

The immediate sequelæ of chancres.—The importance of a thorough examination of the inguinal lymphatic glands in any case of venereal infection cannot be over-estimated. It may at once be noted that true gonorrhœa is but very rarely attended by the least enlargement of these glands. It is common for patients with gonorrhœa to complain of pain in this region when the epididymis and vas are not affected, and such pain is no doubt of a "referred" nature. Occasionally one or two glands may be slightly swollen and tender, but a marked inflammatory bubo, and still more, a suppurating one, in a case of gonorrhœa is almost invariably due to some small chancre which has complicated it. And here we would lay stress on the frequent occurrence of venereal sores in the little pouches of delicate skin on either side of the frænum, sores which are readily overlooked. Of soft chancres it often is true that the smaller the sore the worse the bubo. Formidable inguinal abscesses may owe their origin to some trifling abrasion or chancre, whilst a huge phagedænic sore on the penis may from first to last never excite marked enlargement of the glands. To explain the latter curious fact we can only suggest that the rapidity of the destructive action prevents absorption by the lymphatics, and we have a parallel in the case of noma of either the face or genitals. It must not be supposed that every case of soft or non-infecting * chancre is attended with an inflamed bubo. A large percentage escape, but the risk is so great that every means should be taken to destroy the poison and to heal the sore as soon as possible. A proper cauterisation of each soft sore is the most important means to this end, and pure carbolic acid is, as a rule, the most convenient form of caustic. It causes little pain compared with nitric acid, Ricord's paste, and other caustics formerly in vogue. The exact limits of its action are readily gauged by the white colour it produces, and a single application, followed by the use of cleanliness and a powder (one part of iodoform to three of boracic acid), will usually bring about healing in a few days, unless the sore be large. In the latter circumstances it may be necessary to apply the carbolic acid repeatedly at short intervals, or to employ a stronger caustic, such as one of those just mentioned, or the acid nitrate of mercury.

The characters of a bubo following a soft chancre are well known; the gland enlargement is painful, ill-defined, the skin over it becomes early adherent, inflamed, and if left to itself as a rule the abscess bursts. It is generally undesirable to wait for true fluctuation, and an early incision will secure exit for the pus, and prevent serious undermining of the skin. Not infrequently peri-adenitis is present, and one or more glands lie in a bag of pus; this form is particularly troublesome to treat, and may require a long incision and complete evacuation of the necrosing glands by Volkmann's scoop, etc. Otherwise there is no necessity for more than a short cut into the centre of the bubo, the pus being washed out with carbolic solution, or evacuated by moderate pressure; antiseptic fomentations should then be applied and frequently changed. All incisions into inguinal buboes should be made parallel with Poupart's ligament. The patient with an inflamed bubo should, of course, rest as much as possible, and it may be at once admitted that, if the recumbent position can be secured, and the source of the contagion—the chancre—be efficiently treated, a considerable number of inflamed inguinal buboes will subside without incision. It is impossible here to lay down an absolute rule; the surgeon's instinct must be the guide in deciding whether the use of the knife is called for or not.

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* In using this convenient term we allude only to infection of the whole system with syphilis.

SECONDARY SYPHILIS.

Phenomena of secondary syphilis.—This stage commences with the appearance of a cutaneous eruption and of superficial lesions on the mucous membrane of the throat, tongue, etc., with general enlargement of the lymphatic glands; and with certain other symptoms which are, as a rule, symmetrical, tend to disappear spontaneously after a variable period, and are remarkably amenable to mercurial treatment. From the date of contagion to the occurrence of the earliest secondary lesions, eight to twelve weeks usually elapse; or from the induration of the primary chancre, about five to eight weeks. If the dates can be well ascertained, it will be found that, although occasionally only six or seven weeks have intervened between infection and the first cutaneous eruption, etc., two months is the usual time. Exceptions to this rule may, however, arise in the following circumstances:—

(1) If mercury be administered as soon as the chancre and glands become characteristic of syphilis, the secondaries may be greatly deferred or wholly prevented.

(2) If the patient develop an intercurrent illness (such as typhoid fever) soon after infection, the secondary eruption may be postponed for a considerable period.

(3) In a few cases, for some unexplained reason, the secondary stage is not entered upon for three or four months, or even more.

It is difficult to give a precise time for the duration of the secondary stage, as cases differ so much in the natural severity of the disease, and still more according to the treatment employed. Some authorities assign six months, others a year, and by some two years from the date of infection is given as the limit of time during which secondary symptoms *may* appear. On the whole, the latter appears to be the most accurate and convenient, with the reservation that there is no sharp line of distinction between the secondary and tertiary stages.

Skin eruptions.—The most frequent early syphilitic eruption takes the form of—(1) **an erythema or roseola**, consisting of scattered pinkish spots on the trunk and limbs (especially the flexor aspects), which diminish or disappear on stretching the skin, and which are not unlike the eruption of measles.

Next in frequency and order of appearance comes (2) **a papular or lichenoid syphilide.** This may consist of either small or large papules, which are composed of masses of exudation cells, accompanied by congestion and alteration of the walls of the small vessels in and close to the papules (thickening of the coats and cell deposit around them). A certain amount of proliferation and inflammatory change is common in the epithelium covering the papules, and hence there may be a thin dry scale on each, forming the papulo-squamous syphilide. The large papules are of the same nature, and if desquamation occurs on them, a more or less close resemblance to common psoriasis is produced. If situated on parts of the skin habitually moist, such as the scrotum, the vulva, around the anus, and at the lip-commissures, these large papules have a peculiar appearance, becoming covered with a delicate whitish kind of false membrane, secreting a thin discharge (which is extremely liable to convey infection to others), having a broad base and a smooth surface. They are then known as *condylomata;* and very similar lesions occur on the mucous membrane of the mouth lining the lips, the tongue, tonsils, palate, etc. Although these are frequently referred to as *mucous patches*, there is no essential difference (other than their site) between them and condylomata (Fig. 79). Condylomata are occasionally met with in such situations as the umbilicus, the

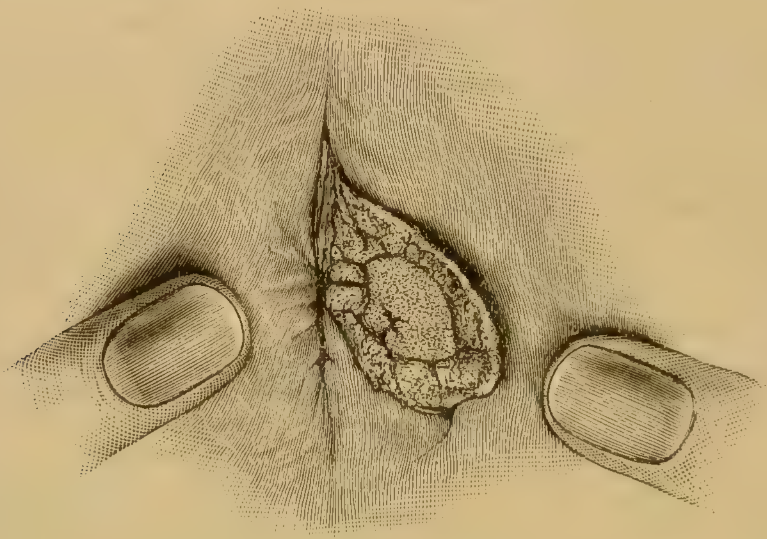

Fig. 79.—Condyloma at the Margin of the Anus, from a patient with secondary syphilis. The surface of the condyloma is seen to be intersected by fissures, and to have a somewhat granular appearance.

axillary folds, the lower border of the breasts, especially in women who perspire freely. The external meatus of the ear is another rare but interesting position, for here a condyloma may block the passage and cause temporary deafness. Between the toes they may develop, and are then prone to fissure or ulcerate deeply, giving rise to the most intense fœtor. On the tongue, lips, and at the anus, condylomata are also prone to ulcerate or fissure from various irritative causes; and hence a good deal of pain may be experienced in them. The term *rhagades* is applied to such fissured condylomata.

(3) If the infiltration of the skin be very extensive, the so-called **nodular** or **tubercular syphilide** is produced, tending to ulcerate and then heal in the centre, spreading at the edge, and closely resembling lupus. This may be met with almost anywhere, but the favourite sites are round the anterior nares, the forehead, scalp, the region over the lumbar and sacral vertebræ, and the buttocks.

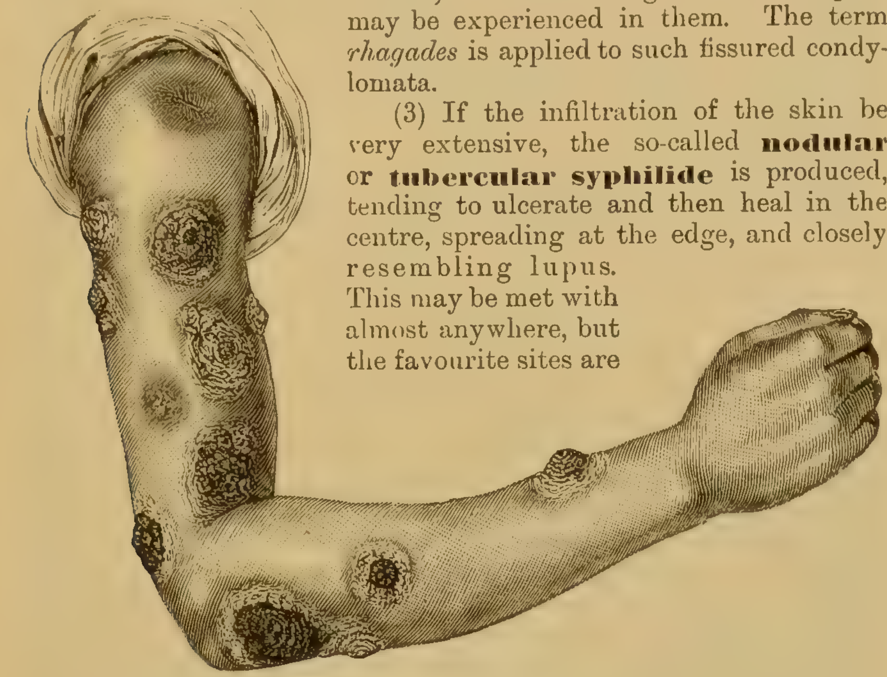

Fig. 80.—Syphilitic Rupia, showing the "Limpet-shell" dry Crusts and the dark Patches left by the healed Ulcers.

(4) A **pustular eruption** is not uncommon, and may resemble acne, varicella or variola; the individual pustules may break and form very large crusts (when the term ecthyma is applied). Occasionally a true vesicular eruption occurs, the contents of the vesicles being clear serum, but this is so rare a form of syphilide that it hardly deserves to be placed in a separate class.

(5) By **pemphigus** is understood an eruption composed of large vesicles. These are apt to suppurate and burst; a certain amount of scab is left, which is pushed up by subsequent extension of the process, until ultimately a conical mass of dried (generally dark-coloured) scab is produced, which is aptly compared to a limpet-shell. If the scab be detached, a circular superficial ulcer is exposed, the centre of which may have almost, or quite healed. To this form of syphilitic eruption the term *rupia* is applied (Fig. 80).

Both pemphigus and rupia are rare syphilides, occur late in the secondary stage, and generally denote a very severe attack or a low state of health in the patient. Pemphigus is more commonly met with in infants suffering severely from inherited syphilis, than in adults with the acquired form ; on the other hand, rupia is, perhaps, confined to the latter.

These five classes of eruptions occurring on skin and mucous membranes—namely (1) erythematous or roseolar; (2) papular or lichenoid (including the papulo-squamous or squamous syphilide); (3) tubercular or lupoid; (4) pustular and vesicular; (5) bullous or pemphigus and rupia—include all the chief varieties met with during the secondary stage. Rare forms—such as a purpuric or hæmorrhagic syphilide and the so-called pigmentary eruption—need not here be described.

Characters of the skin eruptions.—It must be clearly understood that the skin lesions of syphilis rarely keep to one type, and that, for instance, the same patient may present papules interspersed with pustules, and here and there a lupus-like patch. This *polymorphism* is a marked characteristic of syphilitic eruptions; another is their *rough symmetry* (the earlier the eruption the more decided will the symmetry be); a third feature is their tendency to *disappear spontaneously* after existing a certain time, and much more quickly under the internal use of mercury ; a fourth is the *absence of pain or itching* about the eruption; a fifth, the *copper or lean ham colour* the spots may assume; finally, they occur especially on the *flexor aspects* of the limbs, the front and back of the abdomen, the forehead, and edge of the hairy scalp. Taken together, these characters are of considerable use in the diagnosis of syphilitic skin disease, but either one of them may be wanting. Thus, for instance, a secondary eruption may be uniformly roseolar or papular, it may affect the extensor surfaces largely, may cause a fair amount of irritation, etc.

Another important point to bear in mind is that there is no single form of skin lesion peculiar to syphilis. "It may imitate all, it originates none." The most characteristic undoubtedly are the condyloma and the "limpet-shell" crust of rupia. In examining for an early syphilide, particular attention should be directed to the front of the abdomen and chest and the inner aspect of the thighs, for not infrequently the eruption is almost confined to these parts. The penis and scrotum are often affected, and a very characteristic form on the latter is a crescent of reddish infiltration or confluent papules, which may be accompanied with some peeling, or develop into a condyloma. The annular or gyrate outline is met with also on other parts of the body, sometimes as perfect circles of the eruption ; at others in most irregular figures, due to the coalescence of several circles (Fig. 89).

A similar gyrate arrangement is met with in some parasitic skin eruptions—*i.e.* those due to the tinea fungus—and it may happen that only careful microscopic examination, or a search for other syphilitic symptoms, will decide the diagnosis. The term serpiginous is applied to an eruption which creeps over the surface, leaving healthy skin in the centre, whilst spreading at the edge, and many of the later syphilides show a serpiginous tendency. (*See* Fig. 87.)

The peculiar reddish-brown (copper or lean ham) colour of many syphilides has already been alluded to. It is most pronounced when the eruption has existed for some few weeks. In a few cases the amount of pigmentation is excessive, and may last for a very long time.

The early syphilides usually disappear without leaving any trace of their existence, but the later ones—such as rupia, the syphilitic ecthyma, and the lupoid syphilide—generally heal by scarring. Whilst too much importance must not be attached to the character of scars in deciding that they are the remains of syphilis, it may be stated that such scars are commonly circular, thin, supple, and white (often suggesting tissue paper), with sometimes a ring of pigment around them, and wholly free from the hypertrophic tags of skin often met with close to scars of healed strumous or tubercular ulcers.

In describing the syphilitic eruptions, no mention has yet been made of a rather peculiar form—the *papulo-squamous syphilide* of the palmar and plantar aspects of hands and feet. This somewhat resembles, and is often termed, a psoriasis, and whilst the papules or nodules which first develop in these positions do not differ from those met with on other parts of the body, the thick overlying epidermis, which becomes loosened and remains partly detached for long in large rounded flakes, gives it a peculiar appearance. The hands alone or the feet may be symmetrically attacked, but in the later stages of syphilis the disease may be confined to one hand or foot. In many cases the eruption is extremely difficult to cure, and may persist long after all other symptoms of constitutional infection have disappeared. Through the coalescence of several patches, the whole inferior aspect of foot or hand may become involved, the margin of the disease having a slightly raised and infiltrated edge.

General symptoms.—Shortly before, or at the same time as the outbreak of the early cutaneous eruption, the patient's general health suffers in a considerable number of cases. He complains of aching pains in the long bones, the joints, and the head. Sometimes there is lumbar pain, general lassitude, or distinct febrile disorder of a mild type. About every third case of secondary syphilis has distinct nocturnal elevation of temperature for a few days (Fournier), accompanied by increased pulse-rate, malaise, etc. Several observers have proved that the proportion of red corpuscles in the blood is diminished in marked cases of secondary syphilis, to be increased again after mercury has been given for a time. Decided anæmia is, however, not very common, and is usually met with in women, who also present such symptoms as palpitation of the heart and shortness of breath more often than men during the secondary stage. It is worthy of note that occasionally the "syphilitic fever" takes on an intermittent type, and is then liable to be mistaken for ague, or a continuous rise of temperature lasting for several weeks may give rise to the supposition of typhoid. As a rule, the increase of temperature is but slight, or wholly absent, and these exceptional cases are to be remembered chiefly from the errors of diagnosis and treatment that they may cause.

General gland enlargement.—This important symptom is of very frequent, though not constant, occurrence. The lymphatic glands of the sub-occipital region, those along the sterno-mastoid muscle in both triangles of the neck, the epitrochlear glands in the arms, and the inguinal ones are most easily noticed to be swollen during the early secondary stage; but there is good reason for believing that the deep lymphatic glands in the abdomen, etc., are similarly affected. The enlargement is hardly ever painful, rarely of high degree, and may occur quite independently of any cutaneous eruption in the part supplying the glands concerned. Congestion and slight swelling of the liver and spleen are probably not very infrequent, and now and then a sharp attack of jaundice is met with during the secondary stage. If the gland enlargement be very marked, the anæmia or, at any rate, increase of the white corpuscles in the blood, will also be pronounced. It is probable that congestion of the bone marrow and periosteum accounts for the dull aching pain in the legs, head, etc., which has been mentioned as of frequent occurrence.

Affections of the skin appendages.—*Thinning of the hair* of the scalp is a notorious symptom of secondary syphilis, and may be met with in all degrees up to the production of temporary baldness. Usually, it only amounts to the hair coming away more freely than normal all over the head when combed, though sometimes the alopecia is patchy in distribution. As a rule, the hair grows again freely after a time, especially under good mercurial treatment. The eyebrows and eyelashes may occasionally be shed or thinned in a similar way to the hair of the scalp.

The *nails* are less commonly affected; but in severe cases they are liable to become brittle, roughened irregularly at their free edges, or in some cases longitudinally striated. The whole nail may be shed through inflammation of the matrix, and occasionally syphilis produces a peri-onychia or troublesome ulceration around the nail-margin. In most cases, if the nails are affected at all, many of them, both on the hands and feet, show evidence of the disease, which is nearly always severe also as regards the skin-eruption and other secondary lesions.

Secondary lesions of the mouth and throat.—These are almost as frequent and important as those of the skin, and from the pain or discomfort they produce, are perhaps even more likely to attract the patient's attention. The occurrence of mucous patches on the lips, inner side of the cheeks, and the tongue has already been noticed (Fig. 81). Another very frequent symptom of syphilis is an ulcerated patch or fissure on the commissures of the lips, which encroaches on the skin surface, and from its position is often slow to heal. Superficial ulcers are also common on the tongue, especially along its margin (though often on the dorsum), and similar erosions and mucous patches are extremely apt to occur on the tonsils and adjoining part of the soft palate or pillars of the fauces. In exceptionally severe cases the ulceration may be deep, and lead to much deformity of the pharynx by contraction; but in the early stage of secondary syphilis the most frequent lesion is a greyish-white sinuous, often rounded, patch on each tonsil, accompanied by more or less swelling of the lymphatic glands at the angles of the jaw.

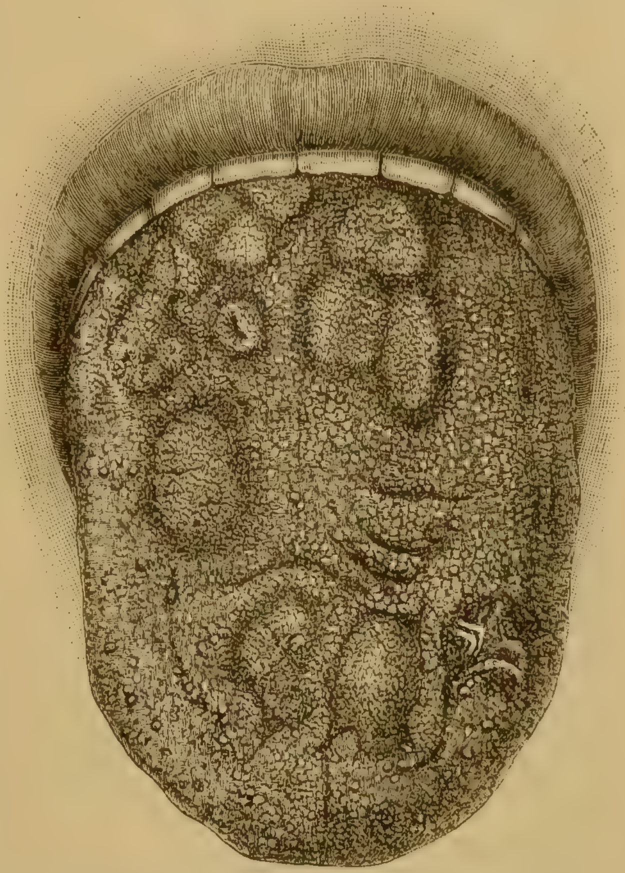

Fig. 81.—Syphilitic Eruption on the Tongue in the early Secondary Period, showing many mucous patches and some ulcers and fissures.

In other cases the tonsils are simply hypertrophied and, with the rest of the mucous membrane of the throat, congested, but not ulcerated. Bald, congested areas on the tongue are sometimes seen, the epithelial covering of the papillæ being here shed in great part, and these rounded denudation patches are very characteristic of syphilis, though they also occur in the rare disease known as "wandering rash of the tongue" (usually met with in young children). As to the severity of the secondary affections of the mouth and throat, a good deal will depend on the patient's habits and the treatment employed. If he neglect all measures of oral hygiene, continue to smoke and to drink spirits and beer, or if mercury be given injudiciously so as to cause salivation, the ulcers and mucous patches will probably be extensive, slow to heal, and very liable to relapse. Sometimes, however, grave ulceration of the mouth and throat during the secondary stage may occur apart from any want of care in treatment or on the patient's part, and may prove extremely refractory. The base of the tongue, the epiglottis, and the laryngeal orifice may be affected with mucous patches, persistent erythema, or ulcers; and there is a distinct danger of œdema of the glottis supervening in bad cases, and causing death unless a prompt tracheotomy be performed.

A common sequela of syphilitic glossitis or of mucous patches elsewhere in the mouth, provided they last for a long time, is the development of white, warty areas, which may prove very persistent.

Diagnosis and treatment.—From follicular tonsillitis—ulcers due to sharp teeth, abuse of smoking and spirit-drinking—and herpes of the mouth, the secondary syphilitic lesions require to be carefully distinguished. The "smoker's patches" may be found on the tongue, occasionally on the fauces, or as triangular areas of thickened white mucous membrane, extending backwards on either cheek from the commissures of the lips. Acute tonsillitis is much more painful, accompanied with more swelling of the soft palate, etc., and with more fever and general constitutional disturbance than the secondary syphilitic ulcers. In both the nearest lymphatic glands are liable to be enlarged, but in a more marked degree with non-specific tonsillitis than with syphilis.

The treatment consists chiefly in the administration of mercury (and in many cases of bad throat affections it is desirable to give iodides at the same time, even though the patient be in the early secondary stage), but also in various local measures. Smoking should be strictly prohibited, and if alcohol be allowed at all, it should be in the form of a glass of claret with the chief meal. Generally, it is advisable that the patient should entirely give up both alcohol and tobacco during the secondary stage, especially if the throat or tongue be at all affected. A gargle of alum (5 to 15 grains to the ounce), of chlorate of potash (10 to 20 grains), or of perchloride of mercury (¼ to 1 grain to the ounce) should be frequently used, with special care in the case of the last-named drug. Painting the mucous patches with a solution of nitrate of silver (5 to 20 grains to the ounce), or of chromic acid (in the same proportion) is often of the greatest service if repeated every few days until healing begins.

A few obstinate and severe cases of ulceration may require more energetic treatment, such as cauterisation with the acid nitrate of mercury.

Sometimes syphilitic ulcers of the tongue, cheek, etc., may be kept up by the irritation of sharp or decayed teeth, or of badly-constructed tooth-plates, and attention should be directed to these points in the treatment. When the mucous membrane overhangs a wisdom tooth, it is especially liable to give trouble if the mouth be attacked by syphilitic lesions, though it is rare for the tooth to require extraction on this account. It is almost needless to point out that very hot fluids, mustard, pepper, and other condiments, should be avoided in all cases of syphilis of the mouth. If the ulcers cause much discomfort or pain, the frequent use of marsh-mallow lozenges is very soothing and quite harmless.

Secondary syphilitic lesions of the eyes.—Of these the chief are: 1, Iritis; 2, Choroiditis; 3, Neuro-retinitis; the first-named being by far the most frequent.

1. **Syphilitic iritis** is practically confined to the secondary stage when the disease has been acquired; whilst in the inherited form it may occur within a few months of birth or many years afterwards (as a complication of interstitial keratitis). We are now dealing only with the iritis of acquired syphilis, which is commonly met with from three to eight months after infection, and forms upwards of 50 per cent. of all the cases of iritis. It is, however, when compared with syphilitic affections of the throat or skin, present in only a small proportion of cases. It varies greatly in severity, may attack both eyes, or only one; and in the former case there is often an interval of a few weeks or more between the onset of the disease on the two sides. The chief symptoms are (1) ciliary congestion with a certain amount of pain referred to the forehead or temple, photophobia, and lachrymation; (2) discoloured iris, sluggish action of the pupil, which dilates irregularly under atropine; (3) adhesions to the lens, deposit of uveal pigment, nodules of lymph in or on the iris.

The congestion, pain, and photophobia are less marked in syphilitic than in rheumatic iritis; there is little tendency to relapse in the former, and if definite lymph nodules or small gummata are present, they are conclusive as to the cause being syphilis. The *treatment* consists in the vigorous use of atropine until the pupil is widely dilated, and in pushing mercury to the verge of producing salivation or sponginess of the gums. The affected eye should be protected from light during the acute stage, and if the pain be severe, blistering the temple, or the use of opium or antipyrin internally, may be required. If only proper treatment be adopted early enough, there are few diseases so satisfactory to treat as syphilitic iritis, perfect vision being probably regained; but if neglected, complete loss of sight, owing to blocking of the pupil, secondary cataract, etc., is liable to occur.

2. **Choroiditis** may come on independently of iritis, or as a later complication of the latter, and may give rise to no subjective symptoms, especially as it is essentially peripheral, *i.e.* confined to that part of the choroid coat farthest away from the optic disc. The importance of careful ophthalmoscopic examination in any doubtful case of old syphilis, as well as search for iritic adhesions, cannot be overrated. Small patches of inflammatory cell-exudation in the capillary layer of the choroid, which atrophy, leaving white or grey areas, surrounded often by deep black pigment, the patches being scattered about the periphery, and at a deeper level than the retinal vessels —such are the characteristic features of syphilitic choroiditis. Sometimes, however, the disease occurs in large or small patches towards the central or yellow spot region, and may then produce grave defect of vision, since the retina overlying the affected choroid is nearly always much damaged.

3. **Neuro-retinitis** may occasionally develop during the secondary stage in one or both eyes, and attention is directed to it by rapid failure of sight; sometimes amounting to complete loss. The ophthalmoscope shows the optic disc and adjacent retina to be blurred and hazy, and the papilla itself to be swollen, while the retinal veins are engorged, and the arteries tortuous and obscured at various points by lymph. Small hæmorrhages may be present in the retina, radiating from the disc. Opacities are often developed in the vitreous with this form, as with some cases of choroiditis. The visual field is always markedly limited.

Treatment of both the preceding lesions of the eye to be effectual must be prompt, and in either, if mercury be administered up to producing sponginess of the gums soon after the disease is noticed, the result is often most satisfactory. Complete rest of the eyes should be enjoined for a time, and it is often advisable to give the iodides in addition to mercury. When the choroiditis is recent or progressive, treatment should be long continued, and surprising improvement sometimes results. When, however, the changes have long existed, little or no benefit can be looked for.

Variations in the secondary symptoms.—The chief symptoms of the secondary stage have now been briefly discussed, and in reviewing them, attention must be directed to the remarkable differences in severity which we observe. Some patients, fortunately only a small majority, suffer terribly from the first development of secondary symptoms. In them the ecthymatous, or rupial eruption, soon follows the roseola; the mucous membrane of throat, lips, etc., may be deeply ulcerated; very severe bone pains (with actual nodes) may wear out their strength; the glandular enlargement and general prostration may be excessive, and the secondary stage pass without break into the tertiary one. The term precocious malign syphilis has been given to these cases, and although it is most often met with in subjects broken down by drink or bad living, or with marked strumous taint, yet sometimes it is impossible to assign any such reason. In some cases neither mercury nor iodides can be tolerated, even in minute doses; in others injudicious treatment (causing salivation, diarrhœa, etc.) has been responsible for the worst features of the case. Secondary syphilis may cause death by œdema glottidis supervening on ulcers of the throat, or larynx, by early gummatous lesions of the cerebral arteries, or through the profound cachexia it sometimes produces. Its sufferers may be incapacitated by blindness from neglected iritis or retinitis, by obstinate ulcerating eruptions, or by exhausting bone pains.

On the other hand, if well treated, the majority of cases develop few and comparatively slight symptoms during the secondary stage, and (provided mercurial treatment has been continued for one or two years) it seems probable that about 90 per cent. escape any tertiary manifestations. And even if little or no treatment be adopted, many cases of syphilis are very mild during the secondary stage, and this fact partly accounts for the difficulty with which a history of the symptoms of this stage is obtained in patients presenting undoubted tertiary lesions. A slight rash on the trunk, and a few superficial sores on the throat may be the only symptoms, and needless to say, they may be readily overlooked or forgotten by the patient.

Treatment of secondary syphilis.—As soon as a positive diagnosis of syphilis can be made, the patient should be given mercury, and if possible he should continue to take it for at least a year, better for two years. Fairly often, the induration of the chancre may be so conclusive, and the bullety enlargement of the inguinal glands so typical, that we have no doubt whatever that constitutional syphilis will follow. In such cases—*and in such only*—a mercurial course should be begun at once, with the view of preventing any secondary symptoms. If the diagnosis is doubtful, it is far better to wait a few weeks until the earliest secondary lesions manifest themselves, treating the chancre locally with iodoform or black wash, etc., and explaining to the patient that it is necessary for him to remain under supervision until two to three months have elapsed from the date of exposure to risk. To submit a case to one or two years' mercurial course for what was only a simple ulcer or soft sore is neither scientific nor justifiable treatment. The earliest secondary symptoms—roseola, slight sore throat, general gland enlargement, etc.—will probably appear within eight or nine weeks if the case be one of syphilis; and if the mercurial treatment is then begun, they quickly yield.

What is a proper mercurial course? In acquired syphilis it is advisable that the patient should continue to take the drug for at least a year; better for eighteen months or two years, with short intermissions. There is no necessity to cause any salivation, or even sponginess of the gums, although some cases of severe secondaries seem to improve most rapidly when the gums are slightly affected. Of the many preparations, one should be chosen which is least irritating to the individual patient, and certainly one of the best is grey powder (mercury and chalk) in doses of one grain three or four times daily. Some patients can take two-grain doses for long without inconvenience, but in many six or eight grains daily will either purge or produce salivation within a week or two. It is customary to combine a little Dover's powder (or pure opium if there be a tendency to diarrhœa) with the grey powder. The pill form is the most convenient, but some object to this, and the liquor hydrargyri perchloridi (in 1 drachm doses) is a good alternative. Blue pill, the tannate of mercury, the green iodide, and calomel may be mentioned as in favour with different surgeons; but the first two preparations (Hyd. cum cretâ and Liquor hydrargyri) are the safest, provided the appropriate dose is determined for the individual case. The gums should be brushed night and morning, and, if necessary, an astringent mouth-wash may be ordered; the diet should be plain, and all articles of food prone to cause diarrhœa carefully avoided. Stimulants should be taken very sparingly, if at all, for there is no question that the free use of alcohol directly interferes with the action of mercury. Smoking must be forbidden if there are any sores on the throat, tongue, or lips. Should such ulcers develop, they should be painted once a day with chromic acid solution (10 to 20 grains to the ounce), or a similar solution of nitrate of silver. Gargles containing bichloride of mercury ($\frac{1}{8}$ to $\frac{1}{4}$ grain to the ounce), chlorate of potash (10 grains to the ounce), or alum, may be useful in different conditions of the throat during the secondary stage. If the eruption be copious, or accompanied by infiltration of the skin, an ointment of the oleate of mercury (5 per cent.) should be rubbed in, avoiding its use over hairy parts or where the skin is delicate, as it may there cause pustules.

Condylomata, wherever situated, are as a rule easily cured by cleanliness and the use of a powder of calomel one part, oxide of zinc three parts. Before applying this the surface of the condyloma must be thoroughly dried, and it is advisable to keep some absorbent wool between the adjacent skin folds, since moisture is an essential to the persistence and extension of this form of secondary eruption. Ulcers or fissures on the skin may be treated locally with an ointment containing one drachm of calomel or red oxide of mercury to the ounce, though not infrequently it will be found better to avoid any greasy application, and to use either weak calomel powder, iodoform, or iodol (protecting the sores from irritation by absorbent wool and a light bandage). It is needless to say that the internal administration of mercury should be carried out in every case of condylomata, etc., as well as the local treatment.

The hypodermic and inunction methods of treatment.—Especially on the Continent these are largely resorted to in preference to giving mercury by the mouth, for not very obvious reasons. At Aix-la-Chapelle, which is much visited by syphilitic patients, the chief treatment consists in systematic inunction of mercurial ointment by experienced rubbers, combined with warm baths, and, in many cases, the administration of iodide of potassium. This plan can equally well be carried out by the patient at home if he will take the trouble. Half to one drachm or more of the ointment (B.P.) should be rubbed daily into parts of the skin surface which will readily absorb it—*e.g.* the inner sides of arms or thighs, the abdomen. It is advisable to change the site of inunction frequently, for fear of causing dermatitis, and to avoid the hairy parts. A few days' vigorous inunction will render the gums spongy, proving the efficacy of the treatment; but it is a dirty and tiresome method, which often depresses the patient extremely.

All kinds of mercurial preparations have been advocated for hypodermic injection. One of the best is a simple solution of the bichloride in distilled water. From one-tenth up to one-fourth of a grain of the bichloride, dissolved in thirty minims to a drachm of water, may be injected at intervals of a few days deeply into the buttock or back, taking the utmost care as to the aseptic condition of the hypodermic syringe (a separate one should be used for each patient). It is much more difficult to graduate the dose successfully by this method than by oral administration, and sometimes very severe stomatitis and diarrhœa are set up by it. In fact, many deaths have been recorded directly due to the hypodermic administration of mercury for syphilis. Nor is there the slightest reason for thinking that, as a rule, either inunction or the subcutaneous method has any advantages over the ordinary plan followed in England and France. We do not give iodide of potassium for tertiary syphilis, or quinine for ague, by means of the hypodermic syringe or inunction, and why should either method be adopted in treating secondary syphilis by mercury?

In certain exceptional cases they may, however, be useful, and especially is this true with regard to inunction in the treatment of infants with congenital syphilis.

The question of marriage and secondary syphilis.—During or at the end of a course of treatment the surgeon is often asked how soon it is safe for the patient to marry. It is generally admitted that, even supposing a careful and prolonged mercurial course is carried out, it is advisable to wait for two years at least after any symptoms have appeared. If this rule be followed, there is but little risk to either wife or offspring; but with regard to the latter it must be admitted that no absolute guarantee can be given. Very much will depend on whether the treatment has been thoroughly persevered with for one or two years, and whether the symptoms have been slight, or have been severe and have resisted treatment.

So long as mucous patches exist on the lips, tongue, or throat, the patient should be warned as to the danger of conveying infection from them by kissing, or by the accidental transference through drinking utensils, etc. How syphilis may be conveyed in this manner is well illustrated by the following cases under my notice:—A glass-blower contracted a chancre of the lip from using the metal blowing-tube handed on to him by a fellow workman who was suffering from mucous patches of the mouth. Subsequently, the second patient infected his wife and child by kissing, both of them developing chancres of the lips.

Is syphilis curable by mercury?—To the patient the chief danger of syphilis lies in the tertiary stage, and it is, therefore, of the utmost importance to decide whether prolonged mercurial treatment will prevent the occurrence of any tertiary symptoms. According to some, who take a pessimistic view, syphilis is never really cured; according to others, out of a hundred cases of secondary syphilis treated early and for a long period (one to two years) not more than five or ten will ever have any further trouble from the disease. The latter view, if perhaps rather too favourable, seems to be the more correct, provided that the patient commences treatment early, and can tolerate mercury well. It is well known that under these conditions, and even occasionally when little or no treatment has been pursued, a man may have two distinct attacks of primary and secondary syphilis. As a rule, several years must elapse before a patient who has had syphilis can contract the disease again.

THE INTERMEDIATE STAGE OF SYPHILIS.

After all the symmetrical lesions of the secondary stage have disappeared, we see not infrequently "reminders," which take the form of peeling patches on the palms and soles of the feet, of chronic orchitis, of superficial leucomata or ulcers of the tongue or throat, and occasionally of lesions of the nervous system or eye. These are sometimes referred to as forming the intermediate stage, but it is very hard to separate them from the tertiary one, and impossible to give a time-limit for their occurrence. It must be clearly understood that the division of syphilis into stages, though very convenient, is an arbitrary one. In some severe cases there is no break between the stages, and characteristic tertiary symptoms may develop within a year or two of contagion. (*For* Treatment, *see* page 419.)

TERTIARY SYPHILIS.

Phenomena of tertiary syphilis.—The tertiary stage is escaped by a large proportion of syphilitic patients, especially those who have been thoroughly treated with mercury. In those who develop tertiary symptoms, they may supervene at any time from one or two years after contagion to thirty years or more. Apart from their late occurrence, their characteristics are :—

1. Aggressive tendency : tertiary lesions will probably spread and certainly persist for very long unless treated.
2. Resolution under treatment with iodides, if commenced early.
3. Asymmetry. This, however, is not always the case, and in inherited syphilis some very remarkable exceptions are met with.
4. In the majority of cases, the tertiary symptoms depend upon the production of so-called gummata, which we will now briefly study.

A **syphilitic gumma** commences as a collection of round cells grouped in especial profusion around the vessels of the structure in which it is placed. This is usually connective tissue, *e.g.* of skin or periosteum, or in some viscera such as the liver, testis, or brain. But in the latter cases it begins probably in the fibrous capsule or supporting tissue of the organ, and spreads to the adjoining parts; thus in the brain, gummata especially affect the dura and pia mater at first, adhering to and infiltrating the subjacent nervous structures. By the pressure of the cells upon the blood-vessels and against each other (often by a concurrent inflammation of the true vessel-wall), their nutrition is damaged and they undergo fatty or granular degeneration. This will be most marked in the centre of the cellular exudation, whereas towards the periphery organisation into fibrous tissue is the rule. Thus is produced a tough yellowish mass, firm and leathery on section, with few or no patent blood-vessels except at the margin, and capable of persisting indefinitely in this condition. If, however, a gumma is situated close to a cutaneous or mucous surface, where it is exposed to injury and to the access of micro-organisms, it will probably (unless treated) undergo softening or suppuration. The overlying skin or mucous membrane which adheres to the gumma becomes congested and inflamed, breaks at one or more points, and a yellowish-white slough is exposed. Unlike the contents of an ordinary abscess, the gummatous centre is only gradually eliminated, and the sodden wash-leather-like mass adherent to the surrounding tissues, and sometimes deeply undermining the surface, may be weeks or months before it wholly comes away. Subsequent granulation and cicatrisation lead often to puckering of the surface, and in the liver (where gummata are very frequent) deep scars in the capsule may clearly point to their previous existence and complete absorption. In the deep viscera—*i.e.* heart, liver, spleen, etc. —it is rare for a gumma to break down or to form anything resembling an abscess. Microscopically, the cells of a gumma are often hardly to be recognised individually towards the centre, as they stain very imperfectly; towards the margin they become more distinct, and the process of organisation into a low form of connective tissue may be seen; a few giant-cells may occasionally be observed.

Apart from the formation of gummata, tertiary syphilis often leads to *sclerosis* of some part of an organ. Of this nature is locomotor ataxia (where the posterior columns of the spinal cord and the sensory nerves are affected), fibrous degeneration of the liver or testis, sclerosis of the tongue or rectal wall. It may cause thickening of arteries closely allied to atheroma, which is of especial importance if it involves the cerebral vessels; more rarely it affects the veins. Syphilitic arteritis may predispose to aneurysm, though its effect in this direction has been exaggerated by some writers.

Sometimes the tertiary lesions are of the most diverse nature, and affect many different organs at the same time; but in other cases one system is chiefly or alone involved. Thus one patient will present tertiary disease of various long and flat bones; in another the skin and subcutaneous tissues are extensively diseased; in a third the viscera and lymphatic glands, etc.

Severe secondaries predispose to severe tertiary symptoms, though this is not an invariable rule. Inefficient mercurial treatment, privations, and probably alcoholic abuse may be fairly given as causes of the development of tertiary lesions in a syphilitic patient; and chronic irritation and traumatism seem to have a decided effect upon their localisation. We have already expressed a strong belief that careful and prolonged treatment during the first two years after syphilitic infection is of great importance in preventing the onset of tertiary symptoms; but at the same time, it must be admitted that in some cases it fails entirely. We will consider now the individual lesions of tertiary syphilis, roughly grouping them in the order of their frequency and importance.

1. **Tertiary syphilitic disease of the bones.**—The most

typical form is the **sclerosing osteitis,** or chronic inflammation of bone, periosteum (and perhaps the medulla), leading to the formation of dense new osseous deposit. This may be formed along the whole length of a bone, or may occur as a more localised swelling, then termed a *node.* With the thickening of the bone on its outer aspect goes a corresponding diminution of the medullary cavity, which may become entirely obliterated. This syphilitic osteo-sclerosis is, as a rule, markedly painful in its early stage, and the pain (of an aching or boring character) may persist for many weeks or months. It is usually worse at night, when the patient has nothing else to distract his mind, and when the vascular dilatation is perhaps at its greatest. But to suppose that the nocturnal character of a bone-pain is proof of its syphilitic origin is absurd, for nearly all forms of persistent pain are worse at night. That of syphilitic osteitis is remarkably amenable in most cases to iodide of potassium, though occasionally it is so severe as to call for the additional use of opium, etc. In exceptional cases, where the suffering is very great and unrelieved by iodides, mercury or other internal remedies, an incision through the periosteum or trephining the bone itself may be required.

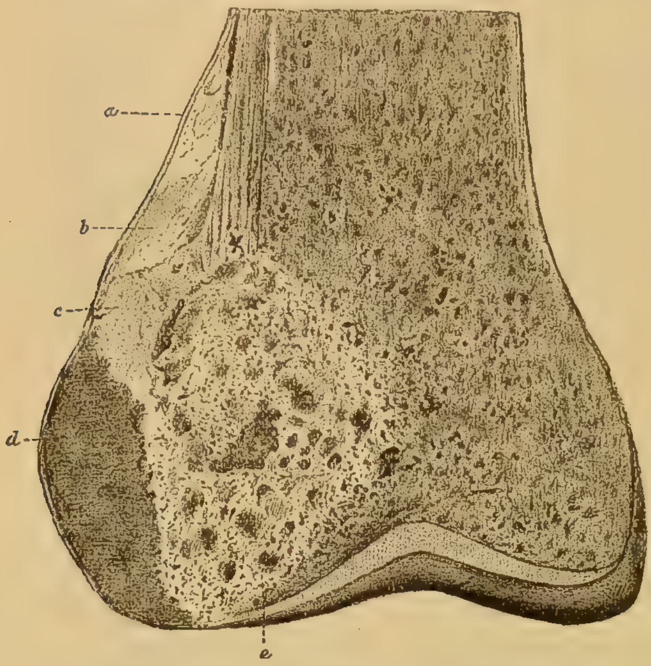

Fig. 82.—Section through Femur with Gummatous Node.

a, Raised periosteum; *b*, new fibrous deposit; *c*, edge of new bone; *d*, cavity containing thick fluid; *e*, gummatous infiltration of the cancellous tissue.

A second form of tertiary bone-disease is the **gummatous,** affecting either the periosteum or the medulla. If periosteal, it will probably soften and break down unless treated, and often produces rounded or oval excavations in the underlying bone. On the skull these smooth-edged circular depressions are remarkably characteristic of syphilis (Fig. 84). If central (starting in the medulla) it may lead to considerable expansion, and perhaps to spontaneous fracture. If in the neighbourhood of a joint the node, whether gummatous or ossifying, may set up synovitis, and is extremely apt to be mistaken for a sarcoma (Fig. 82).

Yet another form of syphilitic osteitis is that causing **necrosis** of greater or less extent, the process being usually a very slow one.

It is especially met with in the palate and nasal bones and those of the cranial vault.

The bones affected.—These various forms—(1) osteosclerosis; (2) nodes, either gummatous or ossifying, or mixed; (3) central gummata; and (4) osteitis, with necrosis or caries—may be met with in the same patient, affecting different bones. Those most frequently involved in acquired syphilis are :—

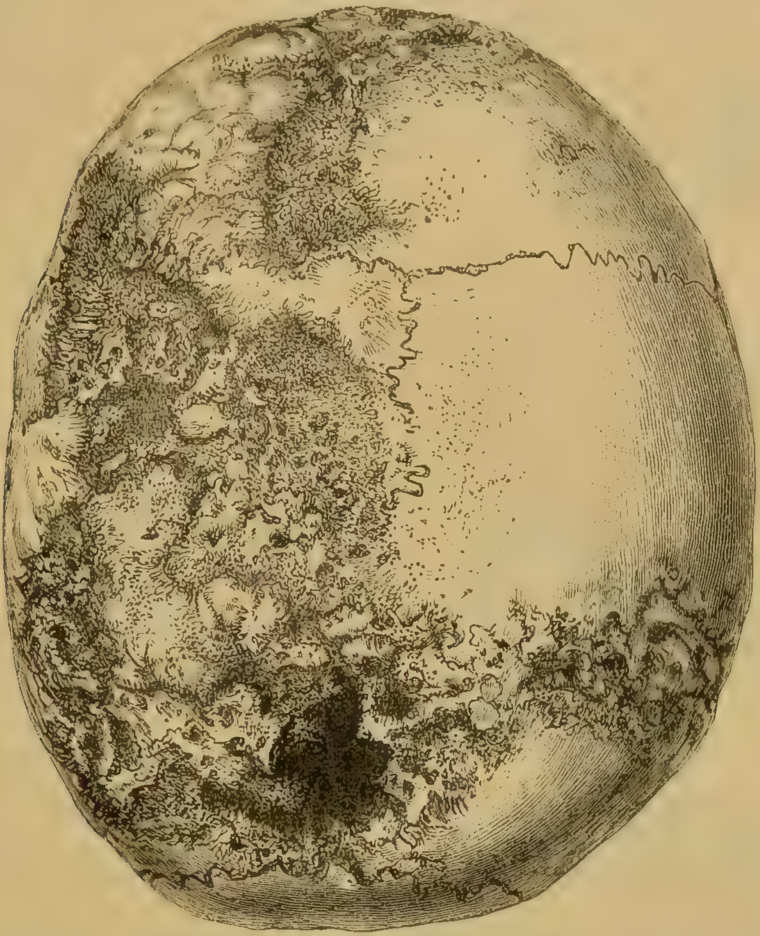

Fig. 83.—Calvarium from a Case of Tertiary Syphilis, with very extensive Caries, formation of rough Ridges of Bone, and at one point Perforation of both Tables.

(*a*) The nasal bones and the septum, leading to falling in of the bridge, perforation of the septum, and (whilst necrosis is going on) to most offensive ozæna.

(*b*) The hard palate, causing perforation and communication between the nose and mouth. This is to be remedied by the use of an obturator, a plastic operation being rarely suitable.

(*c*) The frontal and parietal bones, much more rarely the occipital (Fig. 83). Bony nodes on the frontal, gummatous erosions and pits, necrosis of the outer table, and occasionally of the whole thickness of the calvarium are the chief forms met with. When necrosis is present, there is grave risk of meningitis or cerebral abscess supervening, and as soon as the sequestrum becomes loose it should be removed, for fear of its keeping in the pus (Fig. 84).

(*d*) The clavicles, femora, tibiæ, and the bones of the fore-arm. Nodes at the joint-ends or elsewhere along the shaft, sclerosis of the greater part of one of these long bones, less commonly central gummata, are met with in the case of each of them.

(*e*) The sternum. Softening periosteal nodes, usually situated on the anterior surface, form the chief syphilitic lesion of this bone.

We may note with regard to these and other bones that syphilitic nodes are often situated at and involve the attachment of important muscles to the bone; thus the insertion of the masseter muscle to the lower jaw, the origin of the pectoralis major from clavicle, sternum, and ribs, the sterno-mastoid from the clavicle, the gastrocnemius from the femur, are all rather favourite sites for such nodes or gummata. The frequent strain of these muscles on the periosteum has very probably something to do with this fact.

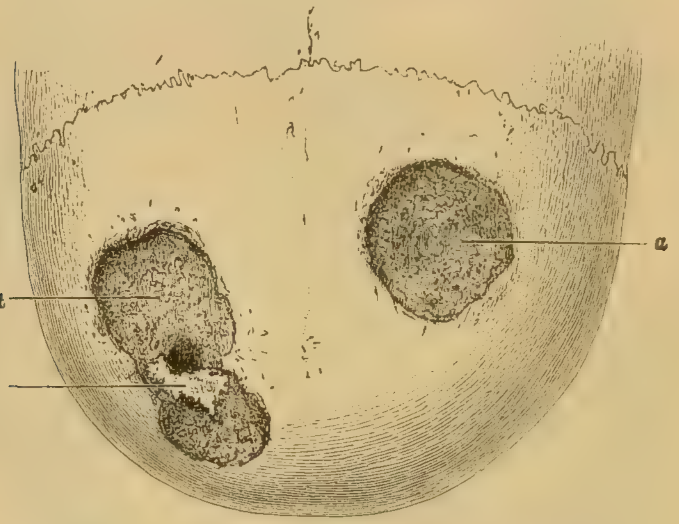

Fig. 84.—Upper Surface of the Frontal Bone of an Adult, showing a rounded, smooth faced depression, the floor of which was formed by the inner table.

a, This had been produced by a pericranial gumma, and there had been no necrosis or suppuration. A second similar excavation, *b*, contained a small sequestrum, *c*, and the suppuration round this had led to perforation of the skull and fatal meningitis.

Few, if any, of the long bones can be said to be exempt from syphilitic lesions. One interesting, but rare form of disease of the phalanges of fingers or toes, has been termed *syphilitic dactylitis.* In this there is usually gummatous deposit round the phalanx (especially those of the first row) in connection with the periosteum, and there may be central or medullary disease as well. The finger or toe becomes greatly swollen, the skin congested and ultimately gives way, broken down gummatous material being discharged (perhaps mixed with fragments of bone), until eventually cicatrisation takes place. Syphilitic dactylitis may be due to either the acquired or the inherited disease. The accompanying figure (Fig. 85) is taken from a case of the latter, and shows the dwarfing of the affected finger, due to absorption of much of the phalanx. It is of special interest from tubercle giving rise to a closely similar lesion in children, tubercular dactylitis being much more frequent than syphilitic. Curiously, tertiary syphilis has little tendency to attack the vertebræ and short bones of tarsus and carpus—favourite sites for tubercular disease. Syphilitic disease of the spinal cord, its dural sheath or its blood-vessels, is well known and quite frequent compared with the same affection of the bones forming the spinal canal; the exact reverse holds true of tubercle.

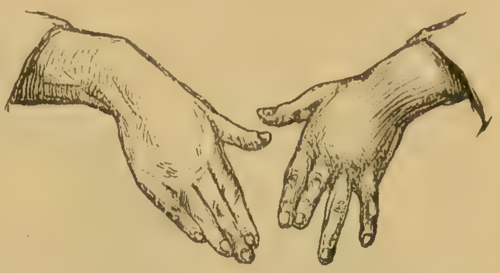

Fig. 85.—Hands of a Child, showing Dwarfing of the left middle Finger, the result of former Syphilitic Dactylitis.

The first phalanx had especially been affected, and it will be noticed that the finger is decidedly shorter than the index one.

2. **Tertiary syphilitic diseases of the skin and subcutaneous tissues.**—This important group includes the very common cases of (1) gummatous and (2) serpiginous ulceration (the so-called syphilitic lupus), and a few cases of rupia and ecthyma. With the exception of the last, to which we need not further allude, the skin lesions of the tertiary stage are characterised by their destructive and spreading tendencies, their multiplicity and asymmetry, and their amenability to treatment by iodides, etc.

1. **Gummatous ulcers.**—The stages leading to the breaking down of a subcutaneous gumma, and the formation of an ulcer over it, have been already described. (*See* page 406.) It must be noted that in some cases when the patient comes under treatment the yellowish-white slough has disappeared; in others there may be marked infiltration of the surrounding tissues, with but little slough at any stage. The ulcers may be met with on any part of the body, but most commonly on the legs, or on some part of the trunk where there is much subcutaneous tissue, such as the region of the loins, or over the sacrum. The buttocks and external genitals are other not infrequent sites.

The following characters of these ulcers are of importance in the diagnosis:—(1) History of a swelling or lump preceding the formation of the ulcer; (2) borders of ulcer sharply cut ("punched-out") and not much undermined; (3) edge rounded in outline, often infiltrated, hard, and congested; (4) base covered with whitish slough, secretion thick (unlike the discharge from a tubercular ulcer, which is thin and watery); (5) the ulcer yields (if not too long neglected) to local and general anti-syphilitic treatment, and if once soundly healed shows little tendency to recur; (6) the resulting scar is usually thin and supple (like tissue-paper), white in the centre, with often a pigmented margin. The amount of pigment which remains will vary according to the length of time the ulcer persists, and its situation. All scars, whether syphilitic or not, are most pigmented on the lower extremities, owing to the venous congestion of those parts.

Tertiary ulcers of the legs are sometimes exceedingly difficult to diagnose from ordinary inflammatory ones, and there is a form of scrofulous ulcer (known as Bazin's disease) which has most of the characters above given. In fact, there is absolutely no conclusive feature about a syphilitic ulcer. The "gummatous slough" of connective tissue may be exactly imitated in some traumatic ulcers, or in carbuncular ones. A carbuncle is, however, almost invariably very painful before the skin gives way, whilst a gumma causes but little pain as a rule, and the former is much more acute in its progress.

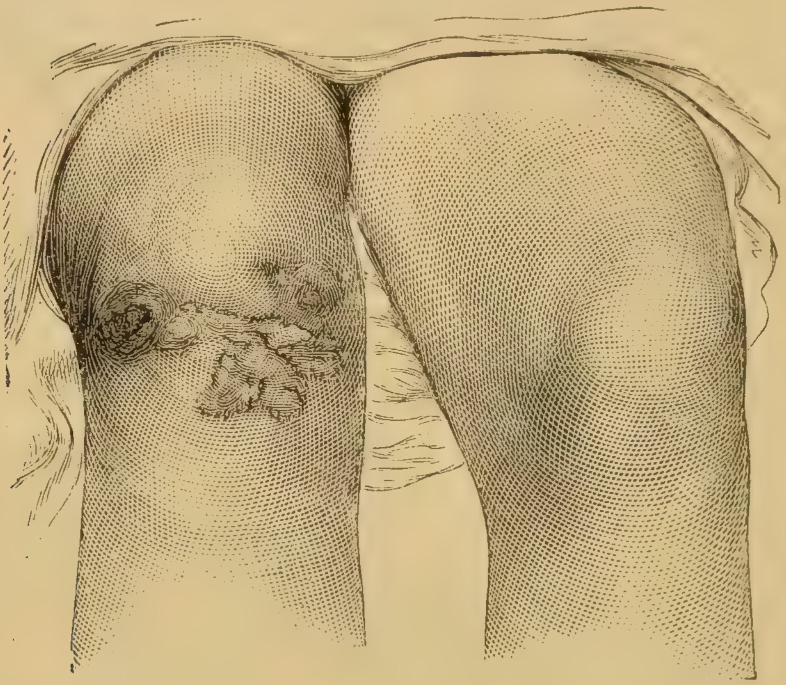

Fig. 86.—Front of Knees, showing multiple scars due to relapsing gummatous ulcers.

The ordinary chronic ulcer met with in subjects with varicose veins, and liable to long periods of standing at their work, occurs chiefly in the lower third of the leg; syphilitic ulcers may occur in the upper third, or over the calf. Gummatous ulcers are peculiarly apt to occur about the knee-joint (especially in women), sometimes over other joints, such as the elbow. These peri-synovial gummata may cause lumpy thickening of the joint-capsule, and even synovial effusion. They are decidedly prone to relapse. Fig. 86 shows the scarring due to this cause in a woman with tertiary syphilis, who had no other gummata, but who had more than one relapse of the disease round her right knee-joint, causing considerable limitation of movement with some synovitis.

Complications of syphilitic ulcers.—If a tertiary syphilitic ulcer takes on rapid destructive action, with foul sanious discharge, and angry-looking edges, the term phagedæna is applied to it. Should such an ulcer be situated near a blood-vessel, there is serious risk from hæmorrhage. Phagedænic tertiary ulcers are chiefly met with in patients who are debilitated by the severity of their syphilis or by injudicious treatment, or by alcoholism, bad feeding, etc. They may, however, occasionally occur without any such possible explanation. Tonics, such as good doses of quinine, and nutritious diet, should be employed as well as the iodides or mercury ; and the vigorous use of antiseptic fomentations or baths will probably check the destructive tendency. In some situations—*e.g.* the buttock or vulva—immersion for long periods in weak antiseptic baths may be required. Iodoform has a considerable power in checking phagedæna, and opium given internally is praised by some writers. Cauterisation (with the acid nitrate of mercury) of the base and edges is reserved for those cases which resist the milder measures given above, and will be very rarely required.

Supervention of epithelioma.—This is only to be feared in those cases of tertiary ulcers of the skin which have lasted long—many months or years—and is decidedly rare. In the case of the mucous membrane of the mouth and tongue, however, tertiary syphilitic lesions show a much greater tendency to pass into epithelioma—a most important pathological and clinical fact.

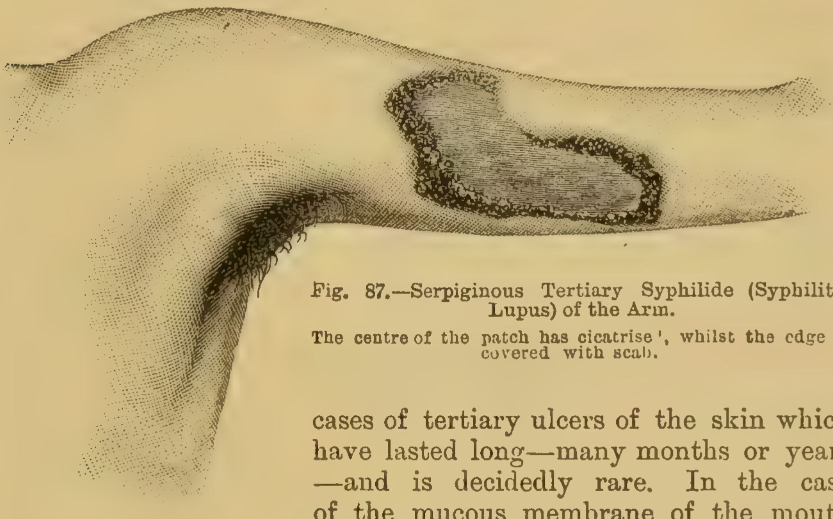

Fig. 87.—Serpiginous Tertiary Syphilide (Syphilitic Lupus) of the Arm.

The centre of the patch has cicatrised, whilst the edge is covered with scab.

2. **Serpiginous tertiary syphilides and syphilitic lupus.**—One very characteristic lesion takes the form of a raised infiltration of the skin, generally of a brownish-red colour, occurring as isolated nodules, which tend to form pustules or to scab over their

centre. These nodules often coalesce later, and a spreading line of infiltration is produced, which has a crescentic, annular, or horseshoe shape. Cicatrisation may occur spontaneously or under treatment at one part, whilst the disease spreads at its margins (hence the term serpiginous or creeping) (Fig. 87). Small characteristic gummata may be present at the same time, but generally these are absent, and the resemblance to common lupus is very close. The margin of the nasal aperture, the lips, and forehead, are particularly prone to be affected with the syphilitic form, and sometimes the arrangement is exactly like that of erythematous lupus, *i.e.* the patches involve the nose and each cheek. But no part of the body is exempt, and the back of the trunk, the buttocks, and the genital regions are very often affected.

The following chief points in diagnosis between common lupus and its syphilitic imitation may be enumerated :—

	LUPUS VULGARIS.	SYPHILITIC LUPUS.
Age of first occurrence.	Usually in childhood or under twenty years. (Many exceptions to this.)	Adult life (in the case of acquired syphilis only).
Region affected ...	Especially the cheeks and nose—those parts most exposed to the air.	May occur on any part of the body, but often affects the parts mentioned.
Multiplicity	Generally occurs in one or two patches with, perhaps, satellites around them.	More tendency to be multiple and widely scattered.
Form and colour, etc.	The lupus patch consists of a number of small nodules, pink and often semi-translucent in colour (like apple-jelly).	More extensive infiltration, often of crescentic outline, and brownish-red or coppery in hue.
Progress	Very slowly destructive; only heals as a rule after vigorous local treatment, and tends to relapse very often.	Extends much more rapidly, but may heal spontaneously in parts; and when once cured does not readily relapse.
Scars left	Perhaps thick, tight, and congested; liable to ulcerate afresh.	Supple, thin, and white.
Other symptoms, history, etc.	Often history of phthisis in family.	Other signs of syphilis, and probably history of secondaries can be obtained.

It must be admitted that in some cases the local signs are quite insufficient, and that the effect of treatment must be tried to complete the diagnosis. A few weeks' administration of combined mercury and iodides with the use of iodoform or oleate of mercury ointment locally, will usually cause syphilitic lupus to heal entirely; but now and then it is necessary to cauterise the patches, whilst in some inveterate cases all treatment fails. The serpiginous character may be extremely marked. Thus, I have known a ring of syphilitic infiltration steadily spread up from the foot to the groin, leaving behind it an extremely thin scar, or hardly altered skin.

Multiple subcutaneous gummata.—Occasionally we meet with very numerous subcutaneous gummata, in their early stage firm and resembling fibromata or the "rheumatic nodules"; later softening, and suggesting chronic pyæmic abscesses. They may also be very hard to distinguish from small lipomata or fibro-lipomata, except by the history of their short duration.

3. Tertiary syphilis of the mucous membranes.—It is a remarkable fact that whilst the mucous membrane and submucous tissue of the mouth, pharynx, tongue, and rectum are very commonly involved, the intervening stomach and intestinal tract are hardly ever affected by tertiary syphilis. The same immunity extends to the bladder, and perhaps the urethra in both sexes. The larynx is often the seat of gummatous or other tertiary disease; the trachea and bronchi only very rarely.

1. **The lips and buccal mucous membrane.**—Here we meet with either gummatous ulcers or, more commonly, very chronic white patches, often accompanied by superficial fissures or scars. The former, if they involve the lower lip, may require most careful diagnosis from epithelioma. The latter are chiefly to be mistaken for the white patches produced by constant smoking, combined, perhaps, with the abuse of spirits. Here, as in the case of the tongue and throat, it often happens that tobacco, alcohol and syphilis have combined to produce the result. The white patches referred to (the so-called *leukoplakia*) consist essentially in thickening of the epithelium (which is sometimes almost warty), with small-celled infiltration and subsequent fibrous sclerosis of the sub-epithelial layer. They occur in the secondary, as well as the tertiary, periods, and are extremely persistent. Side by side with the white patches there may be bald areas, in which the epithelium is very thin; and hence the readiness with which fissures or ulcers occur. There is a grave risk of epithelioma supervening, especially in the condition of general leukoplakia of the tongue (wrongly termed ichthyosis linguæ), in which the whole of the dorsum looks as though coated with layers of white paint. This condition, whether due mainly to syphilis or not, nearly always ends in cancer.

2. **Gummata of the tongue, palate, and pharyngeal wall.**—The ulcers produced, and the subsequent scarring, may cause deep puckering of the tongue, loss of the soft palate, adhesions of the latter to the pharynx, so as almost entirely to shut off the nose from the throat, and much narrowing of the pharyngeal aperture. Fibrous stenosis or stricture from syphilis is not often met with lower than the cricoid cartilage, though undoubtedly a few cases of œsophageal stricture are due to this cause. Gummata of the tongue are chiefly met with on the dorsum, whilst epithelioma and secondary

ulcers are perhaps most common along the margins. For other points in the diagnosis we must refer to the Article ON THE TONGUE (Art. XLV. Vol. II.). Sometimes syphilitic disease of the palate resembles ordinary lupus, but, as a rule, the destruction is much greater, and anything like a gumma does not occur in true lupus.

Gummata at the back of the pharynx may have their origin in the deep tissues immediately in front of the vertebræ, and, as they soften and bulge forwards the mucous membrane, may readily be mistaken for post-pharyngeal abscess. It is most important to diagnose them correctly, as treatment with iodides will probably effect their complete absorption, without having recourse to the scalpel.

3. **Tertiary syphilis of the larynx.**—Although attacked less than half as often as the pharynx, the larynx is not infrequently the seat of tertiary syphilitic disease, manifested in the following chief forms:—

(1) Patches of hyperplasic infiltration, occurring mainly in the epiglottis and the aryteno-epiglottidean folds, usually going on to ulceration.

(2) Gummata in the same positions, breaking down early, unless absorbed by treatment, but sometimes attaining the size of a pigeon's egg before they ulcerate.

(3) Perichondritis, which affects the epiglottis, the arytenoid cartilages, or even the cricoid and thyroid. It is a most dangerous form, as it is apt to cause necrosis of part or the whole affected cartilage, which may then cause death by becoming impacted in the rima glottidis.

(4) Stenosis or stricture of the laryngeal aperture may follow either of the preceding lesions, and may necessitate laryngotomy or tracheotomy.

(5) Paralysis of one or more laryngeal muscles occasionally results from tertiary syphilitic disease of the nerves, or the muscles themselves. It is, compared with the preceding affections, very rare.

The importance of the correct *diagnosis* of these affections cannot be exaggerated, since early treatment may not only ward off the risk of death from acute œdema glottidis (a danger which constantly threatens the subjects of ulcerative laryngitis), but may prevent the voice from being permanently damaged. The chief points in distinguishing them from *tubercular* and *epitheliomatous lesions* are:—

(*a*) The proneness of the epiglottis and arytenoid folds to be involved, and for comparatively rapid and deep ulceration to occur (in tubercular laryngitis the ulcers are generally superficial and numerous, and often affect the vocal cords, the surrounding mucous membrane being pale and thickened).

(*b*) The absence of nodules of growth round the ulcer, as in epithelioma, which, moreover, often starts on the back of the arytenoid cartilage, and hence causes early dysphagia and pain.

(*c*) The patient attacked with tertiary laryngitis is often in the prime of life, and shows no sign of disease of the lungs, which so very frequently accompanies, and often precedes, tubercular laryngitis.

It must be noted that these elements in the diagnosis are sometimes deceptive, and that nothing but a careful examination of the previous history, and the results of anti-syphilitic treatment, may decide the matter.

Mention has been made of the fact that mucous or erythematous patches are met with in the larynx during the secondary stage, but they are of trifling importance compared with the dangers of tertiary ulcers and gummata. Ulcers and stenosis of the trachea, and gummata in the lungs and bronchial glands, have been met with occasionally, but they are extremely rare, in comparison with tubercular disease of the same organs. Gummata of the lungs affect rather the lower parts—perhaps round the main bronchi—than the apices. Gummatous disease of the bronchial and mediastinal lymphatic glands has more than once caused death by pressure on, and obliteration of, the superior vena cava, etc.

4. **Tertiary disease of the rectum.**—The frequency of this is much disputed, especially with regard to the cases of fibrous stricture, which, as is well known, is more common in women than in men; and in the former the history of syphilis may be very difficult to obtain, and the result of giving iodides (when once a tough stricture has been formed) practically *nil.* Nevertheless, we believe that tertiary syphilis is the most important cause of non-malignant stricture of the rectum, and the lesions which may ultimately produce it are well recognised. In the body of a girl dying in the Lock Hospital (with no rectal symptoms) I have seen a huge gumma almost filling the pelvis and completely surrounding the rectum. The result of such a gumma, had the patient lived, would certainly have been considerable narrowing of the rectum. More commonly the rectal wall (muscular and submucous coats) is infiltrated, with more or less ulceration of the mucous coat. When this infiltration commences outside the anus, and spreads within it, the term *ano-rectal syphiloma* is given to the condition, which may come on during the late secondary or the tertiary stage. In any case syphilitic stricture of the rectum is practically always low down, within easy reach of the finger. It is often complicated with hæmorrhoids, sometimes with ischio-rectal abscess and fistulæ. In its treatment, besides the use of "specifics" internally, the frequent application of some mercurial ointment, or of iodoform locally whilst ulceration exists, is of value; and when fibrous narrowing has occurred, the daily passage of a well-oiled flexible rectal bougie may obviate the necessity of an operation. Should the latter, however, be required, linear proctotomy (a thorough division in the posterior direction—*i.e.* towards the coccyx) is worthy of trial. Inguinal or lumbar colotomy is the final resort of the surgeon in very bad cases.

5. **Tertiary disease of the external genitals.**—In the male sex the penis and adjoining parts may be attacked with gummatous ulceration, which is especially liable to start in the glans penis, and to destroy this and the prepuce. In the female, the labia and

even the vagina may suffer severely; and in both sexes, the tertiary ulcers may take on phagedænic action. The mistake is readily made of diagnosing these ulcers as primary venereal sores, though the presence of a typical gummatous slough may prevent this error.

4. **Tertiary disease of the nervous system.**—Some mention must here be made of some of the most common lesions, although they form so large and important a chapter in the history of tertiary syphilis, that the reader must be referred to works on medicine for their full consideration.

Locomotor ataxia is one of the most frequent diseases of the nervous system due directly or indirectly to syphilis; in fact, according to Fournier and others, a history of previous syphilis can be obtained in from 50 to 70 per cent. of its subjects. Usually many years elapse between the secondary stage and the onset of tabes dorsalis, which may develop by various symptoms, such as lightning pains, inco-ordination of movement in walking, oculomotor paralysis, bladder or rectal trouble, Charcot's disease of the joints, etc. Atrophy of the optic nerve may precede the more usual symptoms of tabes. A steady progress from bad to worse is the rule, even though there be a clear history of syphilis and careful mercurial and iodide treatment be carried out; but now and then very good results follow a prompt resort to these remedies.

Chronic spinal meningitis, myelitis, or gummata pressing on the cord are other lesions due sometimes to syphilitic disease, and more or less complete paraplegia below the level affected is usually the result. Recovery, complete and permanent, has been observed in many cases under thorough anti-syphilitic treatment, though in most the prognosis must be very guarded.

Within the cranium we have to notice the following:—

1. Gummata pressing on some part of the brain, starting from its bony case, or from the meninges (pia or dura mater), and causing symptoms which vary with the exact site and the amount of cortical substance pressed upon and infiltrated.

2. Chronic meningitis, perhaps affecting the sheaths of the cerebral nerves, or occurring in a patch upon the surface of the brain, most commonly at the base.

3. Disease of the cerebral arteries, leading to their narrowing or thrombosis, or to aneurysm (sometimes multiple).

4. Neuritis, especially of the optic and ocular nerves (third, fourth, and sixth), which may be secondary to the lesions mentioned under the headings 1 and 2, or may occur independently of them. Of all the cranial nerves, those passing through the wall of the cavernous sinus are most often attacked, the seventh and succeeding nerves the least.

Clinically, tertiary syphilis is frequently responsible for hemiplegia, for epilepsy or epileptiform convulsions, for paralysis of special nerves; whilst its share in a form of general paralysis and of insanity is not thoroughly established, but very probable.

Congenital syphilis undoubtedly leads to a considerable number of cases of idiocy.

5. Tertiary disease of various viscera.—There is no viscus which may not be the site of gummatous disease, the *liver* and *testicle* being, perhaps, the most often involved. Space will not allow here of a due consideration of this very important subject; and as regards the testicle, the reader must be referred to the section on its diseases. In the liver, gummata may attain such a size as to be perceptible as rounded masses through the abdominal wall, and may be mistaken for malignant growths, etc.

They very rarely break down, unlike gummata in more superficial structures, and under treatment may be completely absorbed, leaving large puckered cicatrices in the viscus. Great enlargement of both liver and spleen is not infrequently met with in tertiary syphilis, and may be attended with ascites. Gummata in the *heart wall* have occasionally led to a fatal issue, and they may occur in the *lungs*, simulating tubercular phthisis. Tertiary disease of *lymphatic glands* is not very rare, and is singularly apt to lead to an erroneous diagnosis; it is of particular importance when affecting the mediastinal region, where by pressure on the large vessels it may cause grave symptoms.

The pancreas, kidneys, and supra-renal capsules are of all the solid viscera the least often affected by tertiary syphilis. Lardaceous or amyloid disease of liver, spleen, kidneys, etc., may be indirectly due to tertiary syphilis.

The treatment of tertiary syphilis.—The most important drug in the treatment of the various symptoms due to tertiary syphilis is unquestionably iodide of potassium. It is most effective in cases of gummata or gummatous ulceration, least so, perhaps, in those of arterial disease or of long-standing bone trouble. It must be given in increasing doses, freely diluted, and it is best tolerated if combined with carbonate of ammonia (*e.g.* in the form of aromatic spirits of ammonia). The most striking differences are met with in the tolerance of iodides; some patients suffer from iodism (marked depression, coryza, pustular eruptions on the skin, etc.), with such small amounts as ten grains in the day, others can take three, six, or even twelve drachms daily with benefit. The iodides of sodium and ammonium may be given with, or to a large extent instead of, the potassium salt; whilst less liable to depress they are apparently not quite so effective as the latter.

For symptoms of the "intermediate" stage, and for many of those of the definitely gummatous type, it is advisable to give mercury as well as the iodide, and this may be done in the form of the liquor hydrargyri perchloridi, of mercury and chalk pills, or by inunction. The green iodide is not to be recommended, as it is extremely apt to cause diarrhœa, gastric pain, etc. It is best to persevere with the treatment for some few weeks or even months after the symptoms have cleared off, and some authorities recommend that a mercurial course should alternate with or follow the iodide one.

The patient whilst under treatment should be strictly temperate in his mode of life, especially as regards alcohol, and whilst under iodides should clothe warmly and avoid all risk of catching cold. It is generally advisable to take the drug shortly after a meal. The local treatment of lesions of the mouth, skin, etc., has been already indicated. Iodoform, iodol, calomel in powder are all useful; whilst for the skin ulcers the red oxide of mercury ointment, the oleate, the yellow oxide or the dilute nitrate of mercury ointment may be mentioned. As regards the prognosis, very much depends on the date at which the treatment is begun and the thoroughness with which it is carried out. Syphilitic disease of the brain, especially when due to vascular lesions, is perhaps the least hopeful; but even here complete and permanent cures are not unknown. Some patients unfortunately can take neither iodides nor mercury, but these cases are quite exceptional. Tonics, country or sea-air, and careful dieting have each their place as adjuvants in the treatment of tertiary syphilis.

INHERITED SYPHILIS

Phenomena of inherited syphilis.—Syphilis when inherited from one or other parent by the offspring must be regarded as practically the same disease as when acquired. It observes the same stages, many of the symptoms are identical in the two cases, and the same parts of the body—*e.g.* the long bones, and those of the cranium, the iris, the skin—are very commonly affected in the course of inherited as in acquired syphilis. Further, it would seem that the subjects of the inherited form have a certain amount of immunity from contracting syphilis again. But some lesions due to the former—such as interstitial keratitis—are almost, if not entirely, confined to inherited or congenital syphilis; and on the whole, the tertiary symptoms of the latter are decidedly less amenable to treatment than those of the acquired disease.

The *inheritance* may be through the father or the mother, or both. If the mother have suffered severely from syphilis, there will be a strong tendency to abortion or premature birth, and this tendency may last through many years. As a rule, it gradually dies out. The effect of mercurial treatment is most marked in these cases. Often it happens that a woman contracts syphilis whilst pregnant; if mercury be promptly and continuously given, the child may go to full time, and even present no symptoms of syphilis after birth. As regards the father, it has been already stated that, provided proper mercurial treatment be carried out for eighteen months or so, it is fairly safe for a man to marry after two years have elapsed from the appearance of the last symptom of syphilis; at the same time, it is wise to wait considerably longer, if possible.

Colles's law.—A very curious fact is occasionally observed where the husband has lately had syphilis, and his child clearly inherits the disease—namely, that the mother, although presenting little or no evidence of syphilis herself, may yet suckle her infected infant with impunity. Colles, of Dublin (who, in 1837, first called attention to this immunity of the mother), wrote:—"I have never seen or heard of a single instance in which a syphilitic infant (although its mouth be ulcerated) suckled by its own mother had produced ulceration of her breast; whereas, very few instances have occurred where a syphilitic infant had not infected a strange or hired wet-nurse who had been previously in good health."

The supposed exceptions to "Colles's law" are so few and so dubious that an absolute rule of practice may be laid down that a syphilitic infant should be reared at its mother's breast, and that no danger to the mother is to be feared. The explanation probably lies in the fact that through the placental circulation the mother is really infected, it may be, with a very mild type of syphilis; for a considerable number of these cases do show ultimately some symptoms—such as bone-pains or nodes. In those who remain healthy throughout we can only assume that an immunity has been conferred without obvious infection. Supposing that there are strong reasons against the mother suckling her syphilitic infant, the latter should then be brought up by hand, and on no account should a wet-nurse be employed. The most disastrous results have followed the neglect of this precaution, as syphilitic children suffer so often from stomatitis or mucous patches of the mouth, that contagion through suckling may occur with the greatest ease.

Syphilitic disease of the placenta is a well-established and frequent occurrence, and may lead to abortion, to death of the fœtus in utero, or to premature birth. In this way congenital syphilis is responsible for an enormous mortality, as exemplified by the following statistics:—Out of 330 pregnancies (one or both parents showing signs of recent syphilis) 191, or 58 per cent., resulted in abortion, still-birth, or death of the child within six months. Examination of these cases generally reveals extensive disease of the viscera, bones, etc. Diffuse infiltration of the liver, lungs, or spleen with small cells, and the formation of lowly organised fibrous tissue, which may go on to produce cirrhosis of the organ, are the chief lesions met with. In fœtuses born dead of syphilitic parents, the liver and spleen are often enlarged and almost diffluent. In the long bones the epiphyses may be detached by inflammation about the epiphysial discs, or there may be marked signs of periostitis in these bones or those of the skull-vault. A bullous eruption is occasionally present on the skin; but, as a rule, the latter is only affected if the child lives, and after an interval of from one to three months from birth. It seems that some infants escape any symptoms during the first year, and yet in after-life develop interstitial keratitis or other undoubted evidence of inherited syphilis, though in most of these cases probably the early lesions have been overlooked. At any rate, it is an important rule that the syphilitic child, during the first few months, nearly always shows signs of the taint, the most common being the following:—

1. **Eruptions on the skin.**—A *roseolar* or a *papular eruption* (especially on the buttock, genitals, and lower limbs), sometimes an

ecthymatous or bullous syphilide. The syphilitic erythematous and lichenoid eruption just mentioned—the most frequent early sign of congenital syphilis—especially affects those parts which are so commonly the site of eczema intertrigo and of lichen-eczema in infants; hence much difficulty in diagnosis. The more copper-coloured the spots, and the more marked the infiltration of the skin, the greater are the chances of the eruption being due to syphilis. Large smooth-topped shiny papules of a reddish-brown colour on the buttocks, scrotum, etc., are very characteristic of the disease. Round the anus, at the inguinal folds, round the mouth, and elsewhere, if the papules are kept moist, *condylomata* are very apt to develop (Fig. 88). We may see the mouth completely encircled by well-defined condylomata or mucous patches, and the healing of these, or of syphilitic fissures, may leave a permanent witness to syphilis in the form of fine scars, which radiate from the mouth across the lips, and especially from the commissures. A word of warning must be given against taking these radiating scars in later years as conclusive, for severe eczema with fissures in unhealthy infants may occasionally

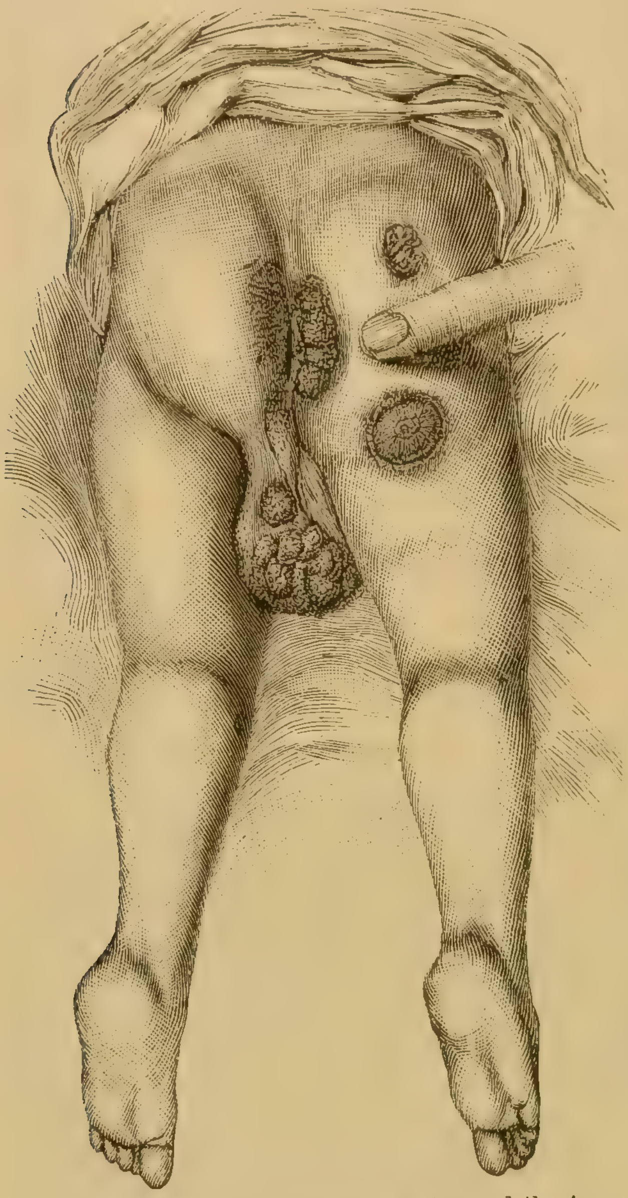

Fig. 88.—Condylomata on the Scrotum and around the Anus, with superficial Ulceration of the Buttock. (From a case of congenital syphilis, showing the regions very frequently affected during the first few months after birth.)

produce a similar appearance. Excoriations or mucous patches round the anterior nares and the eyes are also very common in congenital syphilis (Fig. 89). With regard to the bullous eruption (so-called *pemphigus*), it may be taken as a sign of severe constitutional depression, and although under mercurial treatment these children not infrequently recover, they more commonly die in a marasmic condition. Occasionally a child is born with a bullous or other syphilitic eruption on its skin, but this is rare, and nearly always the erythema or lichen first develops a few weeks after birth. Pemphigus in infants is not always syphilitic. It is said, however, that if it affects the palms and soles it is always so.

Amongst the rarer forms of skin eruption we may note an ecthyma or large pustular sores, purpura, and tubercular or gummatous nodules starting in the subcutaneous layer, involving the skin, and perhaps ulcerating.

Sometimes with the eruption goes *falling of the hair*, though this is, of course, less obvious than in adults, and the whole skin may be withered, sallow or earthy-coloured, and wrinkled, so that an "old-man expression" may be produced. It is remarkable in these cases to see the effect of judicious mercurial treatment, the infant putting on fat and the skin becoming of a healthy tint and consistence. At the same time, it is most important to bear in mind that a syphilitic infant may be plump and healthy-looking during the first few months after birth, for this fact partly accounts for the unfortunate cases of transference of syphilis by vaccination.

2. **Affections of the mucous membranes.**—These have a special importance and interest in inherited syphilis. *Mucous patches* are common on the lips, tongue, etc., but symmetrical ulcers on the tonsils are rarely seen, compared with the corresponding stage of acquired syphilis. On the other hand, a general diffused *stomatitis* and inflammation of the nasal mucous membrane (neither of which is at all frequent in secondary acquired syphilis, apart from the over-use of mercury, which produces quite a different form of stomatitis) are often met with. This stomatitis is often considered to be "thrush" by the mother, and it may have a most injurious effect upon the *developing teeth*. The temporary set may fail to develop, or decay and fall out soon after they are cut, owing to the injury to their enamel, and those permanent teeth which are nearest the surface during the first few months—namely, the upper central incisors and the first molars—may be so affected in their growth as to show, later, features characteristic of congenital syphilis. These will be described later (Fig. 91). The *rhinitis* or muco-purulent inflammation of the nasal mucous membrane causes marked obstruction in breathing and suckling; the infant "snuffles" at every deep inspiration, and the growth of the nasal bones is seriously interfered with. If long persisting, the inflammation may lead to necrosis of the delicate bones forming the nasal walls and to ozæna, though these symptoms are usually met with at a later period. In any case, whether actual necrosis occurs or not, the nasal bridge fails to

develop, and one of the commonest signs of congenital syphilis in later life is a flattened or sunken nose. From a similar cause the nasal septum fails to develop normally and the palate is consequently often high and arched (though this condition is by no means conclusive of syphilis).

A peculiar form of *superficial glossitis* must be alluded to here; it consists of bald patches with well-defined circinate margins, the area involved changing from day to day, so that the term "wandering rash" is applied to it. This curious lesion (it can hardly be called a disease) was claimed by M. Parrot as occurring only in congenital syphilitic infants and children, but this is certainly untrue, and it may be doubted whether it has any direct connection with syphilis.

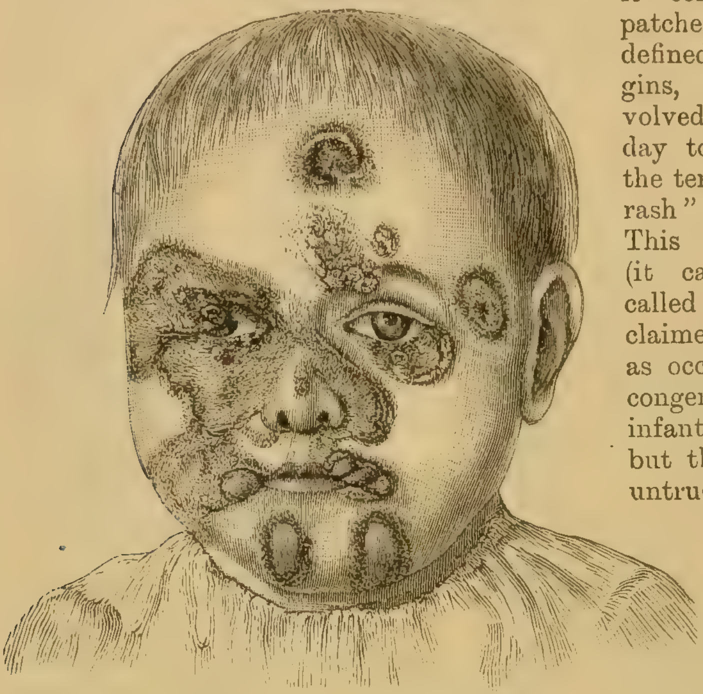

Fig. 89.—Severe Eruption on the Face of an Infant with Congenital Syphilis, having an annular or gyrate outline.

Note its occurrence at the angles of the mouth, where there were several fissures. The individual patches were soft, raised, reddish-brown in colour, and some were covered with scab.

3. **Affections of the bones.**—The effect of the syphilitic poison upon the nutrition of the comparatively soft developing bones of young children leads to changes in the secondary stage—*i.e.* during the first two years—which have no parallel in the acquired disease of adult life.

Craniotabes.—By this term is understood a thinning of the flat bones of the cranial vault, particularly the parietals and occipital, which occurs in somewhat circular patches, which may coalesce. No necrosis ever occurs, but the place of the bone may be wholly taken by fibrous tissue, and the affection is obviously a defect of nutrition of the bone, aided by pressure of the child's head as it lies in bed or on the nurse's arm, since craniotabes is almost confined to the posterior regions of the skull-vault. It may be detected by the surgeon pressing his finger over this part whilst holding the head steady, a characteristic sensation of denting will be felt as the soft patch is depressed, which is compared to that of a thin tin box.

Craniotabes is often met with in infants the subject of inherited syphilis, but not in them alone; the mal-nutrition associated with rickets is quite as important a factor in its production. (*See* page 367.)

Periostitis and epiphysitis.—It is particularly at the growing ends of the long bones that inherited syphilis is prone to cause inflammatory lesions. The disk of cartilage between the epiphysis and shaft, and still more the delicate spongy bone immediately above this, become inflamed, and in a few cases actual suppuration may ensue. Commonly, the affection is recognised only by thickening and tenderness about the joint end of the bone involved, though the epiphysis may become loosened. If, for instance, the upper end of the humerus be affected, the arm will appear to be powerless, and the term pseudo-paralysis is applied to this condition; careful examination will, perhaps, prove that the epiphysis is partially detached, becoming fixed again under mercurial treatment. If several long bones are thus affected, the prognosis is undoubtedly grave, though recovery is the rule. Complete arrest of growth is very rare. Deposit of new bone around the adjacent part of the shaft goes with the epiphysitis, and true bony nodes may form away from the joint ends. In the case of the skull, the new deposit will be formed at the farthest part of the bone from its central nucleus; thus are produced eminences around the anterior fontanelle on the parietal and frontal bones (the so-called Parrot's nodes).

The chief bones affected are the humerus, tibia, radius, and ulna, and the flat bones of the skull. The scapulæ, ribs, and iliac bones are rarely involved, the femur not uncommonly. Those of the hand and foot and the vertebræ seem curiously exempt. It will be noted that considerable resemblance exists between the bone lesions due to congenital syphilis and those caused by rickets; the latter, however, occur usually at a later period (generally during the second year or towards the end of the first), and are less truly inflammatory in their nature (suppuration and caries being never due to rickets alone). The characteristic beading of the ribs and the night-sweats of rickets are not seen in congenitally syphilitic children, unless the two diatheses are combined.

4. **Affections of the viscera.**—In a large proportion of syphilitic infants born dead, or dying in the first year or so, the liver, spleen, and sometimes the lungs, are found to be extensively diseased. A diffuse infiltration with small cells is the usual lesion present, though occasionally isolated gummata occur. Both liver and spleen can often be felt to be enlarged in the living subject, and here again there is a strong resemblance to rickets. Amyloid degeneration may be found on post-mortem examination. The lymphatic glands are more rarely affected, but a careful search may demonstrate their enlargement.

5. **Diseases of the eyes, nervous system, etc.**—The most characteristic inherited syphilitic lesion of the eye—*interstitial keratitis*—does not develop in infancy, but usually in late childhood or about puberty. The earliest age at which it has been met with is

two years, and it is very unusual under five. *Iritis*, however, may occur much earlier, even a few months after birth, corresponding to its onset during the secondary period of acquired syphilis. It is decidedly rare, but its occasional occurrence should be borne in mind, as most serious results to vision may follow its being overlooked.

Meningitis and disease of the cerebral vessels have been met with in syphilitic infants, and probably some cases of infantile hemiplegia are due to the latter cause.

Treatment of congenital syphilis in infants.—As already stated, it is important, if practicable, that the patient should be suckled by its mother, and if this cannot be done, the greatest care should be taken with the artificial feeding. So long as any symptoms present themselves, mercury should be administered, either by the mouth or by inunction. One-grain doses of mercury and chalk thrice daily may be given to an infant a few months old with less fear of stomatitis and diarrhœa than in the case of an adult. It is, however, most important to avoid mercurial stomatitis, since, if this occurs, the enamel of the permanent teeth is liable to suffer severely, and partly for this reason many authorities recommend that the mercurial course for the infant should be much shorter than for the adult, and practically limited by the disappearance of all secondary symptoms. If, however, the infant is thriving well during the mercurial course, is steadily gaining weight, and presents no sign of stomatitis, it is probably best to continue the drug for three to six months after all symptoms have ceased. For anæmic and wasted syphilitic children it is often necessary to administer cod-liver oil (which, it may be remembered, is capable of being absorbed by the skin to a certain extent, and may be thus given in cases where it causes sickness if taken by the mouth), or to give iron and the phosphates. Congenital syphilis is so often a disease in which general mal-nutrition is the most prominent feature, that too much importance cannot be attached to the feeding and general hygiene of the child. Some of the subjects of syphilitic marasmus improve remarkably if pounded raw meat be added to or substituted for their milk diet.

Mercurial inunction is very useful, either the ung. hydrargyri (B.P.) or the oleate of mercury being employed. A common method is to apply the first-mentioned preparation in drachm doses on a flannel binder worn round the abdomen, the skin of which is washed daily. The oleate (5 per cent. solution) is more readily absorbed, but is rather liable to irritate the skin. On the whole, the internal administration of grey powder is a preferable method to inunction.

The later symptoms of inherited syphilis.—After the first few months from birth the liability to skin eruptions ceases, with two or three exceptions. The chief of these is a spreading infiltration (the so-called *syphilitic lupus*), which tends to ulcerate and cicatrise, in many cases causing grave disfigurement. It may occur in inherited syphilitic subjects at almost any age, and resembles closely the form already described in dealing with acquired syphilis.

Gummatous ulcers may also develop on any part of the body and are especially common on the legs, in association with osteitis of the tibia and fibula. Deep ulcers are also met with on the mucous membranes, *e.g.* the palate and throat, and are apt to take on rapidly destructive (phagedænic) action.

With the exceptions mentioned, the subjects of inherited syphilis will almost certainly remain free from skin trouble after the first year, and remembrance of this fact may save the practitioner from error in treating cutaneous eruptions in a child, such as common psoriasis, where the patient's father admits to having had syphilis.

1. **Diseases of the skin.**—It is best to consider the syphilitic lupus and the gummatous ulceration together, for they merge into each other, although some cases present one or the other distinct lesion. By far their most common sites are the face (and here particularly the region of the nose) and the legs, though no part of the body is exempt. An infiltration into the corium, often in the form of a crescentic patch, of a brownish or deep red colour, tending to spread at its margins (serpiginous) and to invade and destroy the deeper structures —such are the characters of the tubercular or lupoid syphilide. Ulceration generally occurs and may present ashy or yellowish sloughs, or be attended with more or less crust-formation. The margin of the nasal aperture may become narrowed or obliterated, the septum is often perforated or in great part destroyed, the lips greatly thickened, etc.

It must be clearly understood that the terms "tubercular" and "lupoid" imply no real alliance with true lupus or tubercular disease of the skin, and no dependence upon the tubercle bacilli; but they deserve to be retained, since they emphasise the close resemblance clinically. Only too often is the mistake made in practice, and irretrievable damage to the patient allowed to occur for want of vigorous anti-syphilitic treatment.

For the diagnostic signs we must refer to the section on acquired syphilis (page 414), and would emphasise the more rapid destruction of the syphilitic form. I have indeed seen a case in which a young man had the face so eaten away by syphilitic lupus (due to the inherited disease) that the back of the pharyngeal vault was exposed, as from the ravages of rodent ulcer, for which it had been mistaken. Under large doses of iodide of potassium cicatrisation rapidly occurred. It is all-important to recognise the disease early, and to increase the dose until healing is secured, whether iodides alone or combined with mercury are employed. Iodoform, the oleate, or the red oxide of mercury ointment should be used locally; and sometimes, if rapid extension takes place in spite of this treatment, it is necessary to cauterise with the acid nitrate of mercury.

2. **Diseases of the eye.**—The chief of these is a very chronic inflammation of the cornea, *interstitial keratitis.* As the name implies, the substance of the cornea rather than its surface is affected, and it becomes infiltrated with cells, new blood-vessels running between the lamellæ. Sometimes the former predominates,

producing a "ground glass" opacity; in other cases, the vascularity is the chief feature, when the so-called "salmon patch" is produced, a deep pink colour due to the minute blood-vessels.

Leucoma, or a white haze, often persists for long after the latter has disappeared (though traces of the vessels can generally be detected as fine black lines by direct magnified ophthalmoscopic examination). Ulcers of the corneal surface, though not unknown, are an extremely rare complication of interstitial keratitis.

The onset of the disease usually occurs between the ages of six and nineteen, though it has been met with as early as two and as late as forty years. Both eyes are commonly affected—often with an interval between the attacks—and the duration of the attack varies from a few months to two years. At its height the patient may be for the time nearly blind; but almost complete recovery is the rule, though faint opacities, or a certain amount of myopia (short sight), are frequent sequelæ. During the attack there is always more or less conjunctival congestion, with a varying amount of photophobia; slight increase of eyeball-tension is not uncommon. Interstitial keratitis is of great importance, not only from the serious loss of vision which it produces for a long period, but also for the valuable aid it gives to the surgeon in the diagnosis of inherited syphilis. Although granular lids may produce vascularity of the surface of the cornea, and the nebulæ left by multiple ulcers may simulate the remains of the syphilitic affections, yet true interstitial keratitis is always due to syphilis, and practically always to the inherited disease.

It should be mentioned that occasionally relapses occur, even with an interval of many years.

Iritis and choroiditis.—Inflammation of the iris has already been noted to develop occasionally in infants, but it is more common as a complication of interstitial keratitis; and since the latter may obscure the symptoms and diagnosis of the iritis, it is a wise rule to keep the pupils dilated by the occasional use of atropine during the attack of the keratitis. Choroiditis or retino-choroiditis usually occurs in the form of disseminated pigmentary or atrophic (white) patches, situated in the periphery, *i.e.* away from the optic disc and yellow-spot region. Vision in these cases is but little or not at all defective, though occasionally the changes simulate those of retinitis pigmentosa, and may cause narrowing of the visual field, etc. Should the yellow-spot region or the central part of the retina be involved, the sight is liable to be markedly impaired.

Optic neuritis with sometimes consecutive atrophy is a rather rare condition in inherited syphilis, and is probably due, as a rule, to an intracranial gumma or meningeal inflammation.

3. **Diseases of the bones.**—It has been noted that in infantile syphilis the long bones are not infrequently inflamed and thickened towards the joint ends. A similar condition, only more chronic in its nature, is met with at a later period, often coincident with the attack of interstitial keratitis. The lower ends of the humerus and

femur are favourite sites, and in Fig. 90 a spindle-like enlargement above and below the elbow-joint is seen, due entirely to periostitis and osteitis of the arm and fore-arm bones. It is not difficult to mistake this condition for strumous disease of the joint itself, and there is no doubt that the articulation is occasionally affected with chronic synovitis in cases of bone disease due to syphilis; but the facts that the bone-enlargement extends far from the joint, is accompanied by considerable aching pain (usually worse at night), and that the joint-movements are but little impaired, should lead to a right diagnosis apart from the existence of other signs of inherited syphilis.

Nodes may form at any part of the bony shaft, and a *general osteitis* of almost the whole length is not uncommon in the tibia, fibula, fore-arm bones, etc. *Sclerosis*, or the formation of dense rings of new osseous deposit, which encroaches on, and perhaps obliterates, the medullary canal, as well as greatly increasing the thickness of the bone, is the almost invariable form of osteitis met with in late inherited syphilis. *Caries* and *necrosis* are liable to supervene, possibly as the result of some added traumatism, or from starvation of some part of the bone by obliteration of its blood-vessels. Here again the process is an excessively slow one. The denuded sequestrum or carious patch is hard and pitted, as though "worm-eaten," often blackened by long exposure. Interference with the surgeon's gouge or chisel is of use here, in order to hasten the process of elimination. A very curious and interesting feature is the *abnormal growth in length* which occurs when this chronic syphilitic osteitis develops and exists for some years before, or even after, puberty. The affected tibia and fibula, for example, become actually longer than the femur, and remain so throughout life. The change is generally symmetrical. Further, the inflamed bone has the appearance of being bent or curved (usually forwards), and sometimes this curving is real as well as apparent.

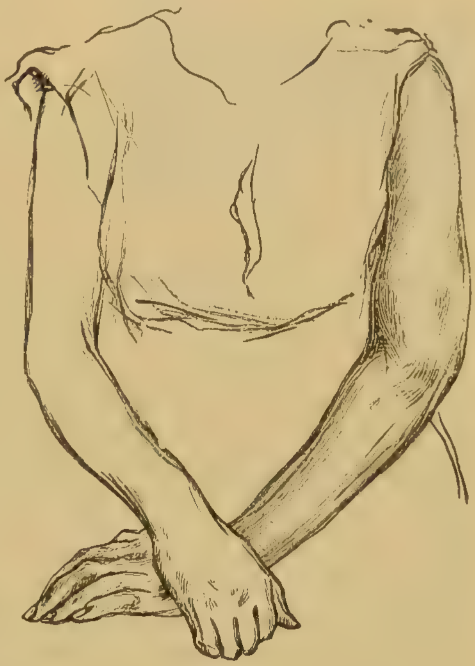

Fig. 90.—Enlargement of lower half of left Humerus, Radius, and Ulna from Osteitis and Periostitis due to inherited Syphilis. The patient was a young woman aged twenty years.

Probably from destruction of the epiphysial disk of cartilage, the bone affected with syphilitic disease may become not longer, but actually shorter, than normal. Fig. 85 shows well the dwarfing of one finger that has resulted from former osteitis in a child with other lesions of inherited syphilis.

Spontaneous fracture must also be mentioned as a rare complication, due to a gumma in the medullary portion which has thinned the bone, or to weakening of the latter from the surface by necrosis, etc.

The bones chiefly involved (as a rule, symmetrically) are (1) the tibia (by far the most frequent and one of the most easily examined); (2) the femur, humerus, radius, and ulna; (3) the clavicle, sternum; and (4) the bones of the skull. Besides isolated nodes on the vault, which are liable to soften and to be followed by limited necrosis, and the destructive inflammation of the nasal bones—*i.e.* including the vomer, turbinateds, and ethmoid—we have to notice a general sclerosing osteitis of the vault, which is occasionally due to inherited syphilis, and which, taken with symmetrical thickening of various long bones, may simulate very closely the mysterious disease known as osteitis deformans. As in acquired syphilis, it is the frontal chiefly, to a less extent the parietals, that suffer most of the cranial vault bones.

The osteitis is, as a rule, not only very chronic, but the cause of much pain, to be relieved greatly by early resort to iodides and mercury. Sometimes the former are the more useful; at others the combination answers best, and it may be advisable to insist upon rest in bed and an equable temperature, should the case prove obstinate. Mercurial inunction (preferably with the oleate) is often useful, and may be regularly employed over the affected part. If iodides are relied on, the dose may need to be steadily increased up to very considerable amounts (say, three to six drachms in the day). They should always be given with some spirits of ammonia, and freely diluted.

4. **Other symptoms of late inherited syphilis.**—One of the most interesting and most obscure is the *deafness* which not infrequently comes on about the same time as interstitial keratitis. There is reason to believe that it is due to some lesion of the labyrinth or internal ear, and it certainly has nothing to do with any obvious change in the throat or middle ear. It is usually symmetrical, progressive, and very resistant to treatment, though some cases gradually improve under anti-syphilitic treatment. Fortunately, it is comparatively rare.

5. **Diseases of the joints, glands, and nervous system.**—A peculiar form of *synovitis* with considerable effusion, almost entirely confined to the knee-joint, and, as a rule, practically painless, and subsiding after a few weeks or months, is sometimes met with in inherited syphilis. The affection is not of much importance, as complete recovery is almost invariable. Synovitis may also develop as a result of nodes on the joint ends of the long bones; and occasionally a chronic arthritis, closely resembling strumous disease, and another form in which the cartilages become eroded, may occur in this stage.

The *lymphatic glands* of the neck and elsewhere may undergo chronic enlargement. Gummata may develop in the testicle, liver, spleen, or other viscera, including the brain.

It is impossible here to review the various *cerebral disorders* that may be due to inherited syphilis. It must suffice to state that they form a large and important group; that clinically we meet with epileptiform convulsions, hemiplegia, paralysis of cranial nerves (especially the oculo-motor ones), dementia and insanity, which appear to owe their origin to this disease. Pathologically, we know that it may lead to narrowing or thrombosis of cerebral vessels, to chronic meningitis, to gummata of the cortex, etc. Possibly also locomotor ataxia may occasionally be due to inherited syphilis, though the influence of the acquired taint is of far greater importance in this respect.

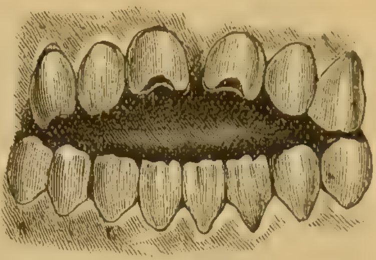

Fig. 91.—Teeth from a Case of inherited Syphilis, showing the convergence of the upper central incisors and a deep semilunar notch in each of them at their cutting edge.

The diagnosis of inherited syphilis.—We have seen how multiform are the lesions which may be produced, and how closely some of them resemble those caused by rickets, and still more those due to tuberculosis. Only of late years have many of the symptoms been rescued from the domain of both, and ascribed to their true origin. To make a correct diagnosis requires, in many cases, the most thorough examination of the patient's history and concomitant symptoms. The greatest help is frequently obtained from the condition of the *incisor teeth;* for a considerable proportion of the subjects of inherited syphilis have their upper central incisors notched and narrowed, as shown in Fig. 91. The cones of these teeth are directed somewhat towards the middle line, and their cutting edge is marked by a semilunar notch. Similar changes, on a smaller scale, are sometimes observed in the upper lateral incisors or those of the lower jaw. They are absolutely conclusive as proofs of congenital syphilis, but it must be clearly understood that only the permanent set show these changes, and that, unfortunately, only in a moderate percentage of cases.

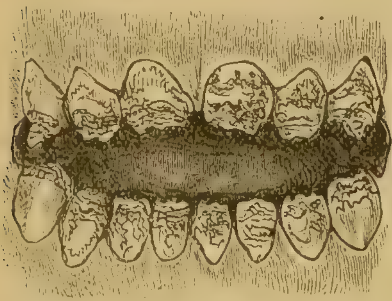

Fig. 92.—Teeth from a Case in which severe Mercurial Stomatitis had been present during Infancy. The enamel is worn off horizontally, and the cutting edges are irregular.

The horizontal wearing away of enamel, which is often due to mercurial stomatitis in infancy, is shown in Fig. 92, and must not be confounded with that previously described; naturally the same patient may present both syphilitic and mercurial changes in the same set of teeth. The incisor teeth in the subjects of inherited syphilis may fall out or require to be extracted early in life. The first molars are frequently deprived of their enamel and wear down quickly, as a result both of syphilitic and mercurial stomatitis.

Next to the teeth, the *eyes* deserve most thorough examination, which may reveal the remains of interstitial keratitis, or of iritis, or choroiditis.

The *tibiæ* should always be inspected in a doubtful case, and nodes may be found on the skull and other bones.

The *depressed bridge of the nose*, scars about the palate or fauces, and persistent *enlargement of the liver* are all suspicious symptoms. The forehead is not infrequently depressed transversely just above the eyebrows with a bony projection running across above the groove, but this change should be very marked for importance to be attached to it, and the same remark applies to the high narrow arch of the palate.

As regards the *history*, that of snuffles and cutaneous eruption, and of condylomata in infancy may help in the diagnosis, and the mother's record as to child-bearing is of great importance. Finally, it should be remembered that syphilis is no respecter of persons, and that the congenital taint is sometimes overlooked from a mistaken belief on the surgeon's part that the parents have always been too virtuous to incur the risk of transmitting the disease. It is often necessary or advisable to make the diagnosis without asking any direct questions, and it may be repeated that interstitial keratitis, notched and narrowed upper central incisors, the peculiar form of deafness, the painless synovitis of the knees, and some of the bone lesions already described, are the most trustworthy elements in the diagnosis, and that most of them can be produced by no other disease than inherited syphilis.

Treatment of late inherited syphilis.—As regards the treatment, we need only say that it is the same as that for tertiary acquired syphilis, the dose of mercury or iodide being varied according to the patient's age, etc. It is often advisable to give tonics at the same time, and the syrup of iodide of iron is very largely used for this purpose.

Prognosis of inherited syphilis.—Can the subject of inherited syphilis contract the disease on his own account? A few clear cases of this have been met with, but remarkably few. As regards the question of marriage, there is not the slightest evidence of syphilis being ever transmitted "to the third generation." Too gloomy a view of the effects of inherited syphilis should not be gathered from the account just given. It is undoubtedly responsible for an enormous infantile mortality, but, on the other hand, many of its subjects grow up to be strong and healthy in every way. In late childhood, or about puberty, there is the special liability to eye and bone diseases, etc., but after this period is gone through, no other symptoms may occur. In many instances, the only evidence lies in a single symptom, such as the peculiar teeth or the occurrence of keratitis.

XXI. GONORRHŒA.

By JONATHAN HUTCHINSON, Jr., F.R.C.S.,

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The gonococcus.—Gonorrhœa may be defined as a specific inflammation of the urethral and certain other mucous membranes, dependent upon the inoculation of a micrococcus which has the following characters:—It is an organism which occurs in pairs, the adjacent sides of which may be concave; is about the size of the ordinary micrococcus of suppuration (or slightly larger); it is cultivated with great difficulty, or not at all, on the ordinary nutrient media, but grows on human blood serum at a temperature of 37° Centigrade; stains well with the various aniline dyes, but is readily decolorised by Gram's iodine method, and is found within the pus and epithelial cells of the discharge as well as outside them. These features, and especially the last two, help to distinguish the gonococcus from the more common micrococci of suppuration. (*See* page 29.)

The accompanying figure (Fig. 93) shows the gonococcus, the streptococci present in epithelial *débris* from the normal urethra and found in all cases of inflammation of the urethra, and also certain micro-organisms which simulate very closely in appearances and reaction the gonococcus, but which (according to Lustgarten and others) are fairly common in the healthy urethral epithelium. It is evident from this that caution is necessary in expressing a positive opinion on the nature of urethral discharge from the characters of the organisms found therein.

In the male subject its habitat is the urethra, and especially the bulbous part and the fossa navicularis; in the female it rarely affects the urinary canal, but breeds in the vagina and cervix uteri, sometimes travelling up to the Fallopian tubes, and proving a fertile cause of salpingitis and its complications.

One attack of gonorrhœa, unlike syphilis, confers no sort of immunity against a second one.

The course of gonorrhœa.—We may divide the course of gonorrhœa into—(1) A stage of incubation, usually from three to five days, but occasionally as long as ten to fourteen; (2) a stage of acute inflammation, lasting from a week to a fortnight, or more if untreated; and (3) a stage of gradual subsidence, not infrequently passing into what is known as gleet, where a thin whitish discharge may persist almost indefinitely. It must not be supposed that, if untreated, gonorrhœa shows a strong tendency to come to an end in some three weeks or so. I have known many cases in which a first attack of gonorrhœa in a previously healthy and perfectly temperate man has lasted as a profuse purulent discharge for six months or more. And looking to the many complications which may ensue, and especially to the danger of a resulting stricture if the gonococcus gets a firm hold of the urethral wall, it should be regarded as an enemy to be attacked with vigour from the earliest date at which the patient comes under care.

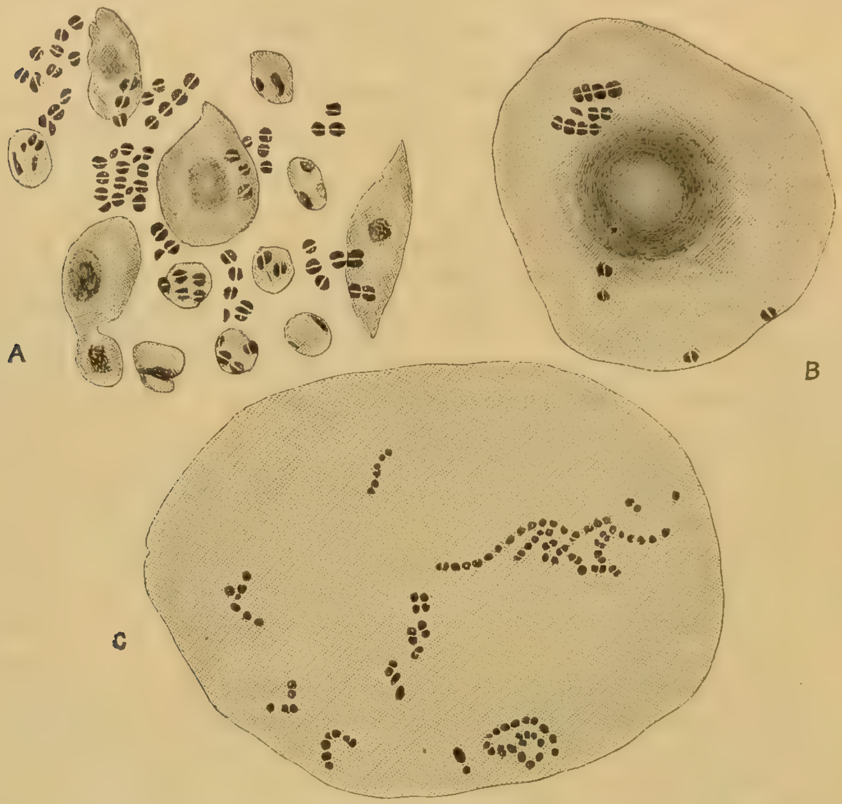

Fig. 93.—A, Epithelial and Pus Cells with Gonococci, from a Case of acute Gonorrhœa.
B, A large Epithelial Cell with several pairs of Micrococci, closely resembling the Gonococci, but obtained from a healthy Urethra.
C, Streptococci and Diplococci, from a Case of acute Gonorrhœa.
All these figures magnified 1,000 diam. (After Lustgarten.)

1. **The incubation stage.**—Three to five days generally intervene between exposure to contagion and the earliest symptom noticed by the patient—a slight adhesion or stickiness of the lips of the meatus, accompanied by a tickling or burning sensation.*

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* This duration of the incubation period is remarkably regular in the case of ophthalmia of the newly-born, in whom the conjunctiva is infected by the maternal

Those cases which present an unusually long incubation stage—a fortnight for instance—are not infrequently examples of a mild urethritis, in the production of which the gonococcus is probably not always to blame.

2. **The stage of acute inflammation.**—The slight sticky discharge which glues the margins of the meatus together rapidly becomes thick yellow pus, the glans and prepuce swell, micturition is more or less painful, heat and pain are felt along the urethra as the inflammation travels backwards, and an exceedingly common symptom is pain referred to the groins or perineum. Microscopically the discharge is found to consist chiefly of pus cells, with a considerable admixture of epithelial cells, which show by their swollen nuclei, etc., evidence of their proliferation.* Inflammatory abrasions of the glans penis are often met with, and are certainly not uncommon within the meatus. The surrounding corpus spongiosum participates in the congestion of the urethral mucous membrane, and becomes swollen and very tender. In a small percentage of cases retention of urine due to the urethral swelling, or to spasm of the muscle in its wall, may be present. Should this retention not yield to a sedative or a warm bath, and relief by instruments be required, the surgeon should use a good-sized very flexible catheter, after injecting the urethra with a 2 per cent. cocaine solution. Painful erections at night are not infrequent, and are to be relieved by bathing the part with cold water; when the penis is bent downwards during the erection by the plastic exudation into the corpus spongiosum, the term chordee is applied. To avoid these complications the patient should be put on a spare diet, and should sleep lightly clothed while bromides of potassium and ammonium (fifteen grains of each), given in a draught an hour before retiring to bed, are very useful in preventing erections at night.

3. **The stage of decline of the inflammation.**—In a fair proportion of cases, at the end of two to three weeks from contagion, the discharge tends to become thinner and more white, the painful symptoms already noticed gradually disappearing; but it is just at this stage that another complication—inflammation of the vas deferens and epididymis—is especially prone to develop. The gonococci have in these cases reached the prostatic openings of the deferential ducts, and so travelled up their canal, usually lodging in the lower part of the epididymis—the globus minor. A further account of epididymitis and the other complications of gonorrhœa will be found later.

As the discharge becomes more scanty and thinner, the symptoms

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discharges on the day of birth. Almost invariably the lids are glued together by purulent matter on the third or fourth day after delivery.

* The gonococci can be demonstrated in the pus and epithelial cells in the following manner:—A thin film of the discharge is carefully dried on a cover-glass, then stained with a freshly-prepared watery solution of methyl violet or blue, and after again drying, it is mounted in xylol balsam. A magnifying power of 700 to 1,000 diameters with Abbé's condenser is needed for the satisfactory demonstration of the gonococci.

of pain and irritation disappear, and too often the patient, on this account, neglects treatment, thus allowing his gonorrhœa to pass into a *gleet*, the duration of which is almost indefinite. The numerous glands opening into the urethra, especially in the fossa navicularis and the bulbous portion, are very prone to harbour the germs, and their secretion contributes to form the flakes and shreds which settle from the urine on standing and which are a most characteristic feature of gleet. Naturally, these are most evident when a considerable interval of time has elapsed since the urethra was washed out by the passage of urine—*i.e.* on rising in the morning. If a bougie be introduced, the presence of gleet will probably be detected by the mucus or sticky pus which clings to it on withdrawal. Apart from the chronic inflammation of the urethral glands, we have to notice the formation of a "granular patch" in the penile or bulbous urethra, a frequent complication, or rather a cause of gleet, which can be guessed at by localised tenderness on the passage of a bougie, but can only be ascertained for certain by a thorough examination with the endoscope. By means of the latter instrument, the mucous membrane at one spot may be seen to be abnormally congested, granular on its surface, and bleeding readily, whilst the thin discharge may be observed to be secreted from the patch itself. The endoscope tube is illuminated with a small electric lamp; its introduction may be rendered absolutely painless by a preliminary injection of cocaine solution (2 to 5 per cent.), which is mopped out by pledgets of cotton wool on holders. It is well to use the largest endoscope tube that will readily pass, and considerable practice is required not to mistake the normal congestion of the membranous portion for a granular or inflamed patch. Some authorities condemn the use of the endoscope as favouring needless and meddlesome topical applications, and there is a grave danger in this direction. Nevertheless, in certain cases of gleet the judicious use of this instrument renders great service, and may be the only satisfactory method of effecting a cure. Should, for instance, a well-marked granular patch be detected, nitrate of silver solution (5, 10, or 20 grains to the ounce), applied to the patch alone at intervals of a week or fortnight, aided by an astringent injection, may cure a gleet which has lasted six months or a year, within a month or two. More brilliant results than this cannot truthfully be claimed, and it may at once be admitted that some cases of gleet are still the despair of the surgeon and patient. The latter is not infrequently prone to allow the persistence of a slight mucous secretion from his urethra to prey upon his mind, or to get into a morbid habit of frequently inspecting the lips of the meatus urinarius, or of fancying "an ulcer" is present, when the only thing required is resolutely to ignore the condition of his urethral organs. In saying this, there is not the slightest intention of deprecating careful treatment of every case of true gleet, for the danger of this leading to a stricture is notorious. The gonococci seem to establish a home in the epithelial cells and sometimes in the sub-epithelial tissues; a plastic exudation follows their long-continued presence, and the fibrous organisation of this exudation will inevitably lead to narrowing of the urethral calibre.

A gleet is very liable to exacerbations under the following conditions :—Alcoholic excess, sexual intercourse, or even nocturnal emissions, and the injudicious use of injections. Great differences exist in these respects, especially as regards the use of alcohol, for whilst some patients are obliged rigidly to abstain, others find that a moderate use of stimulants has not the slightest effect upon the amount of discharge. It is important to forbid sexual intercourse so long as there is a definite discharge, for a double reason; but when the only reminder is the occasional occurrence of scanty flakes in the urine, it becomes a most difficult problem to decide. Whilst it is probably best (for fear of contagion) that such a case should abstain, it is certain that many men in this condition marry and that their wives do not always suffer on this account.

The treatment of gonorrhœa.—This consists mainly in giving internally certain drugs (sandal wood oil, copaiba, cubebs, etc.), which are excreted by the kidneys, and have a decided power in checking the urethral discharge; and in the local use of antiseptic and astringent injections. Various opinions are held as to the relative value of these two measures, and whilst it is generally advisable to employ both, the careful and thorough use of suitable injections is probably the most important. In treating gonorrhœal ophthalmia we rely entirely on local measures, and when the same poison attacks the urethral mucous membrane, they are equally called for. If the case is seen within a day or two of the onset, the following abortive treatment may be employed * :—The urethra is rendered insensitive by cocaine after the patient has first passed his water. The endoscope is then introduced, and the posterior limit of the inflammation made out so far as is possible. A solution of ten or twenty grains of nitrate of silver is then swabbed over the whole of the affected area. The patient should rest for twenty-four hours, and then use a weak boracic acid and sulphate of zinc injection for a few days, unless the return of the discharge should require a second application of the nitrate, or the use of a stronger injection.

It is equally efficient, and certainly less risky, for the patient to begin at once by injecting a solution of sulphocarbolate of zinc (two to five grains to the ounce) three or four times a day, or oftener, and by taking fifteen or twenty minims of sandal wood oil thrice daily. The bowels must be kept well open by aperients, all stimulants must be abstained from, diluents (barley water, soda-water, etc.) freely drunk, and the patient should keep as quiet as his circumstances will allow (it is rarely possible or necessary that he should remain in bed for several days). The injections should always be made after the patient has to some extent cleansed the urethra by micturition. The nozzle of the syringe (glass or rubber) should be inserted well within the meatus, from one to four drachms slowly injected, so as to distend the urethra, and held in for a few minutes. In many cases treated by this plan all trace of discharge disappears within a week or ten days; but the treatment should be continued for at least a fortnight after this has occurred, diminishing the strength of the injection. The patient must be warned against dancing, bicycle-riding, or other severe exercise for a few weeks, and sexual intercourse and alcohol must also be forbidden.

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* This method we owe to the late Mr. Cotes. Whilst undoubtedly effective in some early cases, it is one requiring considerable trouble and expenditure of time on the part of the surgeon, and is very painful, unless cocaine has been thoroughly used.

Other useful injections are:—(1) chloride of zinc (one to two grains to the ounce); (2) sulphate of zinc (two to five grains); (3) nitrate of silver (half to one grain) (this stains the linen, is more painful than most other injections, and on this account is not to be recommended as a routine measure); (4) sulphate of alum (five grains to the ounce); (5) bichloride of mercury (1 in 5,000, increasing in strength). Sometimes extract of belladonna is added to the zinc solutions (ten grains with half a drachm of mucilage), but the sulphocarbolate at least causes so little discomfort that this is rarely necessary. In a few cases the swelling of the mucous membrane about the meatus is so great that the mechanical act of injecting cannot be borne, and then the old plan of waiting until the inflammatory stage has subsided, whilst giving bicarbonate of potash and tincture of hyoscyamus with buchu, may well be employed.

In the stage of gleet it is often necessary to vary the injection from time to time. Amongst those not already mentioned, which may prove useful, are—the four sulphates (one grain each of sulphate of alum, zinc, copper, and iron to the ounce), solutions of tannic acid, acetate of zinc, or resorcin.

The oil of yellow sandal wood is preferred to copaiba and cubebs for internal administration, because (1) it rarely disagrees with the stomach, if taken within two hours of food; (2) it is free from the risk of causing a general erythematous eruption. The dose of the oil is fifteen or twenty minims, given in gelatine capsules, or in a mixture with mucilage, syrup, and cinnamon water. It is apt to be adulterated, so that care should be taken to procure the oil from a a trustworthy source. For a description of the copaiba rash *see* Article XXVIII.

Complications of gonorrhœa.—The following, amongst the complications of gonorrhœa, have already been alluded to:—(1) **Retention of urine** during the acute stage (it may also occur from alcoholic excess at a later period); (2) **chordee**; (3) **epididymitis**; and (4) **stricture.**

The last two are treated elsewhere in this work. (*See* Vol. II., Art. LI., on INJURIES AND DISEASES OF THE URINARY ORGANS, and Art. LII., on INJURIES AND DISEASES OF THE TESTIS, SCROTUM, AND PENIS.) It may here be noted that only a small proportion of cases of gonorrhœa ever develop a stricture, and that a man may have many attacks and yet escape this formidable complication. Nevertheless, a persistent gleet, particularly if with it the patient notices a little trouble in micturition or lumbar aching, should always make the surgeon suspect the development of a stricture. In such a case a full-sized bougie (metal or flexible) should be passed with the utmost gentleness and care, for nothing is easier than for the surgeon, when he encounters resistance due to muscular spasm, to mistake this for an organic stricture. A preliminary injection of cocaine is here often of use. Should a commencing stricture be found, the bougie should be passed at regular intervals of a few days for a considerable time, a measure to be afterwards continued by the patient. Some recommend the frequent passage of a large bougie in every case of gleet—a plan open to abuse and often harmful alike to the patient's purse and urethra.

A considerable number of complications of gonorrhœa remain to be noticed.

4. **Balanitis, etc.**—The delicate skin lining the inner surface of the prepuce and covering the glans penis is apt to become inflamed; and if the preputial orifice is narrow, phimosis is very likely to occur, owing to œdematous swelling of the prepuce. The œdema may further involve the skin of the penis up to its root, and sometimes it is of the nature of a lymphangitis. In such cases rest in bed, if possible, the continuous application of evaporating lead and spirit lotion, or of ice compresses, with the very frequent injection under the foreskin of cleansing or weak astringent lotions, are to be employed. There is considerable risk of producing paraphimosis, if the inflamed foreskin be drawn back over the glans, the latter then becoming engorged with blood, owing to the constricting ring of prepuce. Actual gangrene of the glans is very rare from *paraphimosis*, since ulceration of the constricting part will generally relieve the strangulation; but to relieve the pain it is nearly always advisable to divide the constriction (in the middle line of the dorsum), and if the patient will consent to a circumcision as well, this measure will often hasten complete recovery.

Phimosis with free purulent discharge may be due to gonorrhœa alone, but the presence of a concealed chancre should always be suspected, and the induration due to it can often be detected through the inflamed prepuce. Decided glandular enlargement in the groin makes it very probable that a sore is present, and not simple gonorrhœa or balanitis.

Small abrasions or superficial ulcers of the glans are often met with in gonorrhœa, and heal readily under treatment by cleanliness and the use of a drying powder (*e.g.* of zinc oxide and calomel or boracic acid and iodoform). Relapsing herpes is another occasional result of gonorrhœa and balanitis, requiring similar measures and sometimes circumcision.

5. **Warts.**—In both sexes the growth of soft warts, sometimes to an extraordinary extent, is liable to result from the long-continued irritation of purulent discharge from the genitals, though there is no reason to think that they depend directly upon the gonococcus.

The glans penis is their usual site in males, the labia in females—it is rare for them to invade the urethra. If they do not subside under the use of astringent powders (*e.g.* tannic or salicylic acid diluted with twice the bulk of zinc oxide, etc.) and strict cleanliness, glacial acetic acid should be applied to each wart daily until they shrivel. In cases of exuberant warts it saves time to anæsthetise the patient, to excise the warts with scissors, and, if necessary, to apply the cautery to their bases, which bleed very freely. Warts in the urethra require the use of a sharp curette.

6. **Bubo.**—It is only in a very small proportion of cases of gonorrhœa that there is marked enlargement of the inguinal glands, but occasionally we see a glandular abscess due apparently to this cause alone (*i.e.* not complicated with soft chancre). Not infrequently the glands are tender and slightly swollen during the first few days of the attack.

7. **Peri-urethral abscess.**—In connection with one of the glands opening into the fossa navicularis or the bulbous portion, with Cowper's gland in the male and Bartholin's gland in the female, etc., a small painful swelling may form during an attack of gonorrhœa. The farther back it is situated the more severe are the symptoms attending this peri-urethral inflammation, which forms a lump generally on the under surface of the male organ. If left alone, it may subside or may suppurate and open into the urethra; if it causes much pain and tension, etc., it is advisable to open the abscess by a small incision into its most prominent point. Unless situated behind a stricture, it rarely leads to extravasation of urine.

8. **Gonorrhœal ophthalmia.**—If gonorrhœal pus be conveyed by the patient's finger, handkerchief, etc., to the conjunctiva, a most dangerous form of ophthalmia is liable to occur. An itching and burning sensation will be followed in a day or two by conjunctival congestion and swelling of the lids, and by the third or fourth day the secretion will be freely purulent. The palpebral conjunctiva becomes soft and velvety and bleeds readily, that covering the globe may be raised into thick œdematous folds (chemosis), and when the lids are separated, a stream of yellow pus exudes or spurts out. Corneal ulcers (which tend to perforate into the anterior chamber), or even sloughing of the whole cornea may occur, and the danger of complete loss of vision is very great. Spasm of the lids is almost constant, and may so hinder treatment that division of the outer canthus with scissors may be necessary.

The symptoms and the danger to vision are practically the same in cases in which adults have inoculated their eyes from the urethral pus, as in those cases of infants infected from the vagina during delivery, though, perhaps, the gravity increases slightly with the age of the patient.

Treatment must be prompt and energetic. If one eye alone be infected, the other should be protected by a Buller's shield (a watch-glass secured between two circles of strapping) worn night and day, or by an antiseptic pad and bandage frequently changed. The lids of the affected eye should be separated and the whole conjunctival surface very frequently washed out with weak bichloride of mercury solution (1 in 20,000) or iced boracic lotion. This is best done by means of an irrigator. Nitrate of silver solution (10 grains to the ounce) must be applied to the conjunctiva once or twice daily whilst the discharge is purulent and free, though its use is attended with risk if the discharge is scanty and the chemosis great. It should never be used in the solid form, since a slough would be certain to be produced. Iced compresses give relief, and should be applied in the intervals between irrigation. During the acute stage the lids must be constantly prevented from adhering, and the measures recommended will require the continual services of a nurse both day and night.

9. **Gonorrhœal rheumatism.** — Owing probably to the absorption from the urethral wall of the virus of gonorrhœa, in a small proportion of cases a very obstinate form of arthritis is set up. The knee, elbow, the joints of the foot and hand, are frequent sites, though it would be difficult to mention any single articulation which may not be involved. Further, the synovial tendon-sheaths of wrist or foot, the bursæ, the fibrous sheaths of nerves (especially the great sciatic), the plantar and other fasciæ, the sclerotic coat, and the iris, may be affected with chronic inflammation secondary to gonorrhœa in both sexes. The most usual form is a hydrops of one or two joints and synovial sheaths, painful and very slow to clear off, starting as a rule three or four weeks after contagion. Very rarely suppuration follows in the joints with all the attendant dangers of pyæmia; more commonly the disease ends by stiffening the joints, and complete ankylosis is not unknown.

From acute rheumatism the hydrops due to gonorrhœa is known by the small number of joints affected, the absence of marked fever and sweating, and the chronicity of the disease.

Cardiac complications are very rare, but not unknown; salicylates are practically useless in the treatment of gonorrhœal rheumatism. This mainly consists in curing all traces of urethral discharge, in support and rest of the affected joints during the early stage, and massage with hot douches and the use of Martin's bandages in the later stage. Blistering may relieve the pain at first.

Internally, sandal wood oil, full doses of quinine or iodide of potassium, sulphocarbolate of soda (ten- to fifteen-grain doses) are each recommended by different authors, though unfortunately there is no remedy yet known which can be relied upon.

10. **Cystitis.**—Occasionally the gonorrhœal inflammation spreads back to the neck of the bladder, and both acute and chronic cystitis may thus be produced. For the symptoms and treatment we must refer to Art. LI., INJURIES AND DISEASES OF THE URINARY ORGANS, Vol. II. It should be noted that inflammation of the prostatic urethra may give rise to aching perineal pain, frequent and difficult micturition, or slight hæmaturia, and this posterior urethritis has been often wrongly termed cystitis. Acute inflammation of the

prostate gland and even abscess in its substance have been known to complicate gonorrhœa, but are very rare.

Gonorrhœa in the female.—Some allusion has already been made to this, and space will only allow of a very brief further notice. The vulva and the vaginal wall are first affected, the canal of the cervix uteri is often involved, and spreading up from this the lining membrane of the uterus, the Fallopian tubes, and the ovaries are liable to become inflamed. The urethra participates in only about 20 per cent. of the cases, and stricture is an almost unknown result in women. Long-persisting leucorrhœa, sterility, and pelvic peritonitis (occasionally suppurative) are amongst the complications to be feared.

The *treatment* of gonorrhœa in women is almost entirely local, and consists in warm hip baths, frequent vaginal douches, and irrigation through an indiarubber tube, introduced well into the vagina. The various astringents and antiseptics already mentioned are useful, and when the disease has become chronic, glycerine of tannic acid and powdered alum or tannin (applied on pledgets of cotton-wool introduced to the top of the vagina through a speculum) should be tried.

XXII. TUMOURS.

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THE significance of the term tumours has in recent years been very greatly curtailed, partly on account of increased knowledge in regard to their histology and the relation of micro-organisms to some forms of morbid growths ("tumour diseases" of J. Müller), and partly on account of a more accurate knowledge of embryology. The morbid conditions usually classed among tumours will be, in this Article, further curtailed by the exclusion of those diseases termed by Virchow "infective granulomata," which include tubercle, glanders, actinomycosis, and the gummatous lesions of syphilis. This list will continue to expand as our knowledge of micro-organisms increases. In spite of this restricted meaning the term tumour still remains indefinable.

Classification.—Tumours may be readily arranged in four groups:—

I. CONNECTIVE TISSUE TUMOURS.
II. EPITHELIAL TUMOURS.
III. DERMOIDS.
IV. CYSTS.

Each group contains a number of genera, and each genus comprises one or more species. A list of the genera will precede the description of each group.

Innocency and malignancy.—Before proceeding to the systematic description of tumours it is necessary to take into consideration certain peculiarities relating to them. For instance, surgeons recognise in the connective tissue and in the epithelial tumours two very important clinical features—*innocency* and *malignancy*.

Malignant tumours exhibit the following characters:—(1) infiltration, (2) infection of lymph glands, (3) recurrence after removal, (4) dissemination, (5) inevitable destruction of life.

Innocent tumours, on the other hand, are (1) usually encapsuled, and when diffuse do not infiltrate. (2) They do not infect the glands. (3) They do not recur after removal; or (4) disseminate, but tend continually to increase in size independently of the growth of the individual; and (5) only imperil life from mechanical causes, and especially when they grow in the vicinity of vital organs. The connective tissue group contains one genus of malignant tumour—the sarcomata; and the epithelial group contains two genera—epitheliomata and carcinomata. The remaining groups—dermoids and cysts—contain only innocent tumours.

As the terms *innocency* and *malignancy* are in frequent use, it will be necessary to illustrate them by some concrete examples. It is necessary to mention at the outset that innocent tumours sometimes destroy life as certainly and as quickly as malignant tumours. The essential difference between them may be expressed thus: *The baneful results of simple tumours entirely depend on their environment; but malignant tumours destroy life, whatever their situation.*

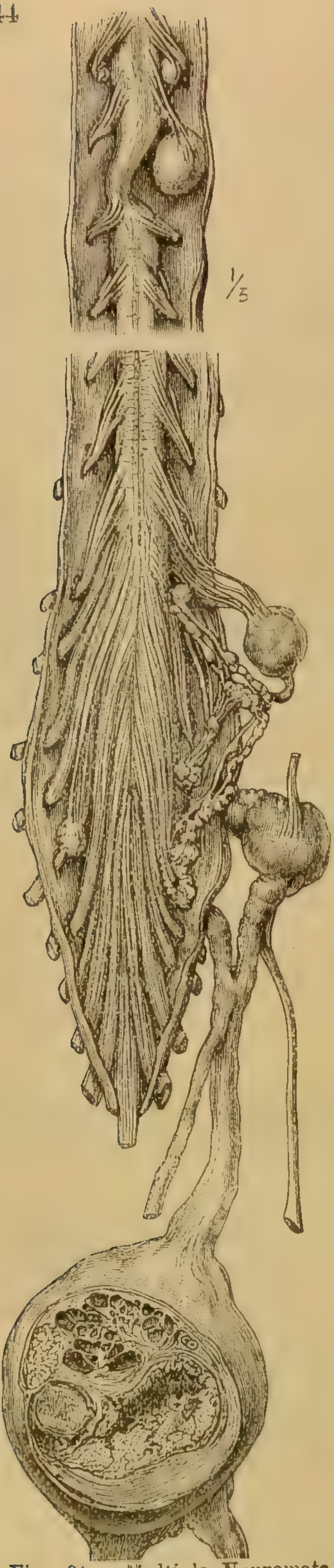

Fig. 94.—Multiple Neuromata.

Of all the simple tumours few can claim to be more innocent than the fibrous tumours of nerve-sheaths termed neuromata. When they grow on the peripheral nerves, even the largest trunks, they may be said never to cause death, although they are very often the source of intolerable pain and anguish. When a neuroma springs from the roots of the upper spinal nerves, it will cause no trouble, so long as it can be accommodated between the walls of the spinal canal and the cord; but as soon as it begins to encroach upon and compress the soft cord against the resisting bony walls of the spinal canal symptoms of paraplegia supervene, and eventually death. This is well demonstrated in the specimen (Fig. 94), taken from a man 45 years of age. The anterior crural nerve is occupied by a neuroma the size of an orange. Its existence was accidentally discovered after death. Many of the roots of the lumbar nerve contain small neuromata. Some are so closely congregated as to resemble the annulated root of ipecacuanha, but the fatal result was brought about by a tumour the size of a nut springing from the root of the fifth cervical nerve, which had squeezed and destroyed the cord in this important region.

The preceding example illustrates the effect of *environment* in regard to the destructive results of a simple tumour, but it must also be borne in mind that environment influences very largely the baneful influence of malignant tumours, and although it is as yet impossible to state why a periosteal sarcoma of the femur should be one of the most deadly tumours known, yet very good reasons can be adduced to explain the rapidly fatal course pursued by cancer of the uterus and epithelioma of the tongue.

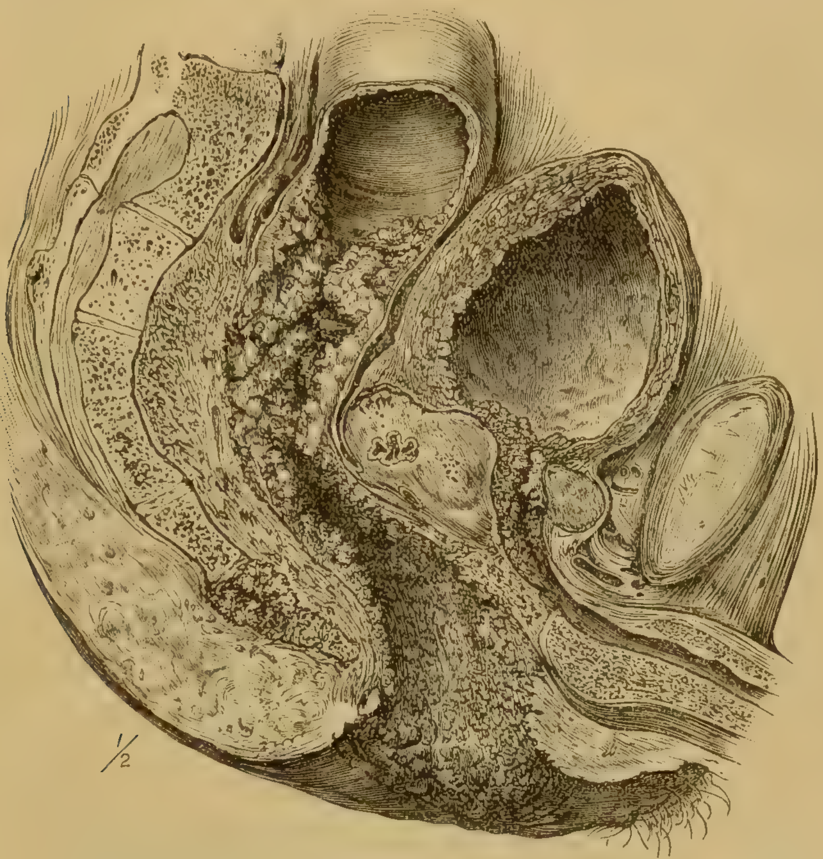

Fig. 95.—Side View of the Male Pelvis in an advanced Case of Rectal Cancer; to show infiltrating tendency.

The *infiltrating tendency* of many malignant tumours explains, in some cases, the rapidity with which they destroy life. Take as an example the specimen shown in Fig. 95. It was removed from a man 33 years of age, who died from cancer of the rectum; no disease was suspected to exist in the patient six months before his death. In this case the disease had not confined itself to the rectum, but had crept down the bowel and infiltrated the skin around the anus; it had also made its way through the muscular coat of the bowel, and had involved the sacrum and coccyx, causing necrosis of the first coccygeal vertebra. The exuberant growth in the rectum had so obstructed the lumen of the bowel, that colotomy was urgently needed six weeks before the man died. In addition, the disease had infiltrated the prostate, so that button-like knobs of the tumour obstructed the urethra and caused retention of urine, necessitating the regular use of a catheter.

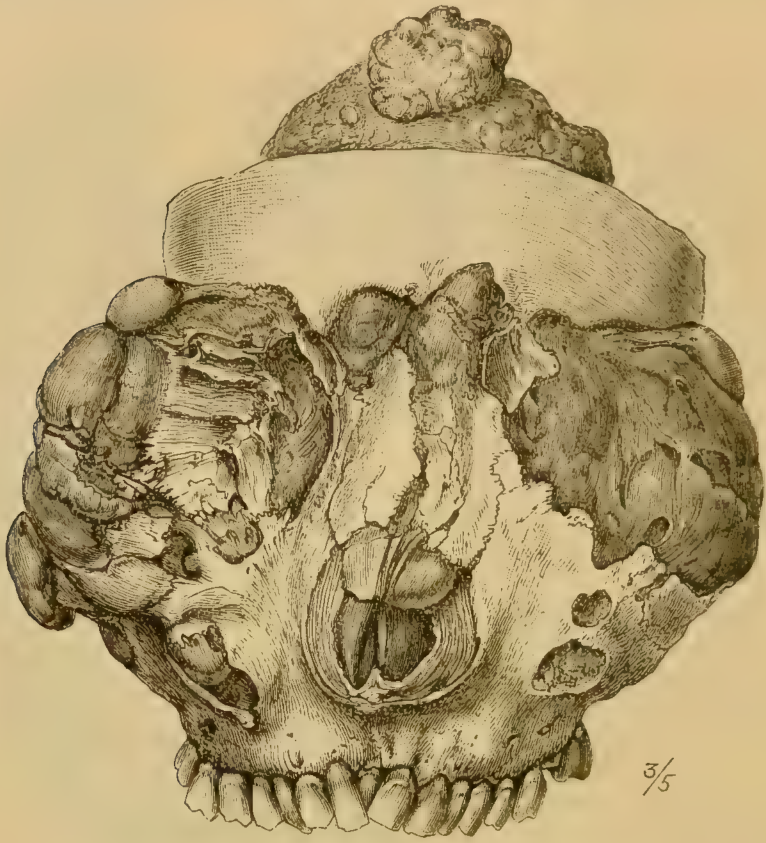

Fig. 96.—Chondroma of the Mesethmoid (Perpendicular Plate) invading the Cranial Fossa and Distorting the Bones. (Museum, St. George's Hospital.)

There were no secondary deposits; death in this case was largely due to the interference with vital organs, in consequence of the infiltrating properties of the tumour. The immediate cause of death was uræmia, and each renal pelvis was dilated and filled with purulent material. The rapid course of events in this case was due not only to the essential malignancy of the tumour, but also to the implication of the bladder and ureters, and the consequent renal trouble. The progress was assisted by progressive anæmia, caused by small losses of blood frequently repeated, and also by the persistent drainage of albuminous fluid from the tumour.

It might appear from the facts of this case that the destructive effects of malignant tumours may in a large measure depend on environment, but opportunities will be taken in the course of this Article to show that malignancy depends entirely on the character of the tumour; its destructive effects may be enhanced by its situation, but not always, for a lympho-sarcoma of the mediastinum or testis may be more rapidly fatal than a sarcoma of the cerebrum.

The baneful effects of *structure* as compared with even dangerous environment is illustrated by the skull (Fig. 96), shown in anterior view, minus its vault and mandible. A large tumour composed of hyaline cartilage fills the nasal fossæ and occupies each orbit so as to dislocate the eyeballs outwards; it overfills each antrum and expands the nasal bones; processes of the tumour invade the spheno-maxillary fossæ, and a large mound of cartilage projects into the anterior fossa of the cranium, and, in all probability, during life, came in contact with the cranial vault. The very remarkable expanding and disruptive effects of the tumour upon the facial bones are well displayed in the drawing; the maxillæ are so pushed outwards as to cause the incisor teeth to cross each other. The patient was under the care of Cæsar Hawkins in 1848, the tumour had been noticed two years previously. In 1849 she left the hospital, and though blind, "her health was little disturbed, she was calm and composed." A portrait of her appearance in 1850 is preserved in the Museum of St. George's Hospital. She survived her discharge from the hospital three years. "Up to the period of her death she had no loss of intellect, and it is believed no paralysis." An examination of the specimen indicates that the tumour sprang from the mesethmoid (perpendicular plate). It is perfectly encapsuled, and required six years to encompass the death of the woman, notwithstanding its dangerous environment, a period twelve times longer than was requisite in the case of rectal cancer (Fig. 95). The fatal effect in the case of the chondroma was due to the pressure of the tumour on the brain, for had the growth attacked a phalanx or a metacarpal bone it would not have shortened the patient's life.

Although the majority of malignant tumours are non-encapsuled and infiltrate adjacent tissues, a few are furnished with very distinct capsules; this is the case with some sarcomata, especially the variety known as myosarcomata occasionally met with in the kidneys of infants. Many of these have a perfect capsule; they are nevertheless very malignant and cause death usually within a year from the time they assume such a size as to become clinically recognisable. Innocent tumours nearly always possess a distinct *capsule*, which completely isolates them from surrounding structures, and the few species which are diffuse differ very materially from non-encapsuled malignant tumours in being non-infiltrating.

The distinction between a *diffuse* and an *infiltrating* tumour is important and demands attention. Fatty tumours are extremely common, perfectly innocent, and in nearly all cases encapsuled; there is, however, one species of lipoma (fatty tumour) which has no capsule, and is called in consequence "diffuse lipoma" (Fig. 98, page 450). Though this tumour is diffuse and, in the example selected, the fat extends without any defined limits beneath the skin of the neck and on each aspect of the platysma muscle, it does not burrow beneath the deep cervical fascia and implicate the muscles and vessels of the neck like a cancer, epithelioma, or sarcoma. Other examples of diffuse but non-infiltrating tumours will be mentioned in the genera fibromata, gliomata, and neuromata.

The infiltrating propensities of malignant tumours explain, in part, the phenomenon of recurrence, for in the process of removal the surgeon, unable to define its limits, leaves portions of the tumour undestroyed, and as the life of these fragments is in no way influenced by the removal of the main mass, they continue to grow and even attain greater proportions than the original tumour.

A very common feature among malignant tumours is *lymph gland infection.* This is most marked among epitheliomata and carcinomata (cancers). It is fairly constant in the melano-sarcomata, which arise in the skin. When simple tumours involve the skin and ulcerate, the adjacent lymph glands may enlarge; this is apt to complicate the diagnosis. There is, however, this great difference: after the removal of a malignant tumour, the infected glands continue to grow; whereas, after the removal of a simple tumour, any enlarged glands associated with it quickly resume their natural size.

The most remarkable feature of malignant tumours is their tendency to reproduce themselves in distant organs—a phenomenon known as *dissemination.* The products of this process, termed "secondary deposits," are histologically reproductions of the primary tumour. Dissemination occurs widely in carcinomata. It is very frequent in sarcomata, but is unusual in epitheliomata. Peculiarities in the distribution of secondary nodules will be discussed in connection with the sarcomata and carcinomata.

Tumours of all kinds are prone to degenerate. Many are liable to fatty, mucoid and hyaline changes; others become infiltrated with calcareous material. These are often referred to as secondary changes. Tumours which grow rapidly are liable to ulcerate, bleed, and necrose.

Pseudo-malignancy.—When carcinoma arises in the mucous membrane of the gall-bladder, intestine, or body of the uterus, small bud-like processes of the tumour are liable to make their way through the walls of the affected viscera, and project into the peritoneal cavity. Under such conditions the growing cells are apt to be detached and scattered over the belly. Many of these cells become engrafted upon the serous membrane and grow, forming secondary knots. In some cases, especially with gall-bladder tumours, these little nodules are present in great numbers.

A similar condition is occasionally met with when papillomatous cysts of the ovary and ovarian dermoids rupture, or leak, into the peritoneal cavity. The cells grow upon the peritoneum and form warts in one case, and small dermoids in the other. Dissemination of epithelial growths in this manner may be spoken of as *epithelial infection.* In the case of carcinomata and sarcomata epithelial infection of the peritoneum soon induces death. Infection from an ovarian papilloma gives rise to hydro-peritoneum; but when the primary tumour is removed, the warts quickly atrophy. In the same way papilloma of the renal pelvis may infect the bladder; and epithelium of the skin or cornea, when accidentally buried in the subcutaneous tissue or cornea, will sometimes grow into a small tumour (implantation cyst). In order to distinguish the infection of the peritoneum by papillomata and dermoids from that due to cancer or sarcoma, it will be useful to speak of it as pseudo-malignancy.

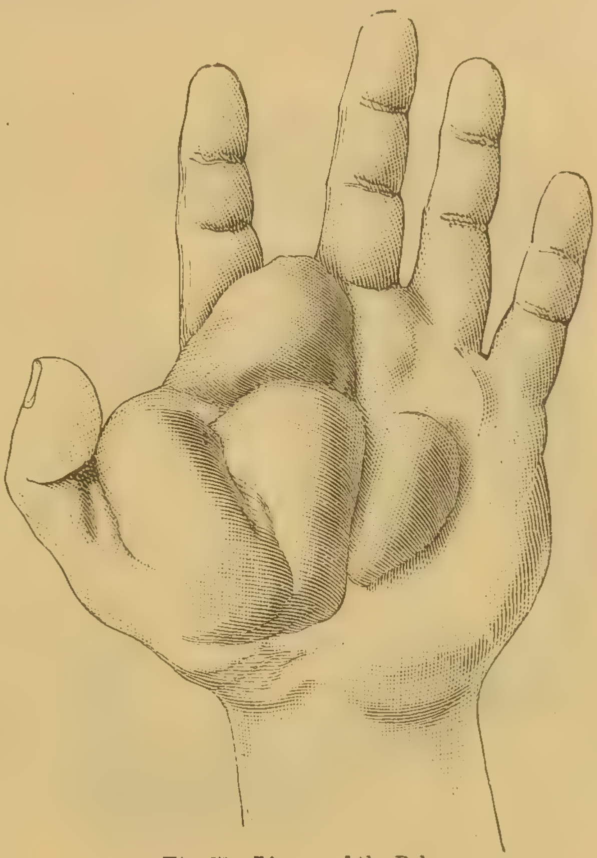

Fig. 97.—Lipoma of the Palm

GROUP I. CONNECTIVE TISSUE TUMOURS.

This group contains the following genera:—1, Lipomata; 2 chondromata; 3, osteomata; 4, odontomata; 5, fibromata; 6, myxomata; 7, gliomata; 8, neuromata; 9, angeiomata; 10, lymphangeiomata; 11, myomata; 12, sarcomata. Adrenal tumours and deciduoma are provisionally grouped with the sarcomata.

1. Lipomata (Fatty Tumours).

A lipoma is a tumour composed of fat. Lipomata are the commonest and most widely distributed of all tumours that occur in the human body. The species of this genus, determined mainly by the situations in which they occur, are:—(1) Subcutaneous,

(2) subserous, (3) subsynovial, (4) submucous, (5) intermuscular, (6) intramuscular, (7) periosteal, (8) meningeal.

1. **Subcutaneous lipomata.**—These occur as irregularly lobulated encapsuled tumours in the subcutaneous fat. Unless they have been irritated or inflamed these tumours are movable within their capsules, but cannot be completely isolated from the overlying skin, which is usually puckered. In most cases one lipoma is present, but two, ten, or more may exist concurrently. In size they vary widely; some give trouble when they do not exceed the dimensions of a ripe cherry, and others weighing ten or twelve pounds are borne by apathetic individuals without complaint. Although this species is mainly confined to the trunk and trunk end of the limbs, they may arise in the subcutaneous fat in any situation—face, neck, fingers, palms (Fig. 97), soles, or scrotum. When multiple they are often symmetrical so far as situation is concerned. Rarely a lipoma becomes pedunculated, and there is a variety known as diffuse lipoma, in which the subcutaneous fat in the neck, the axillæ, groins and pubic region, becomes excessive and forms unencapsuled masses, as in Fig. 98.

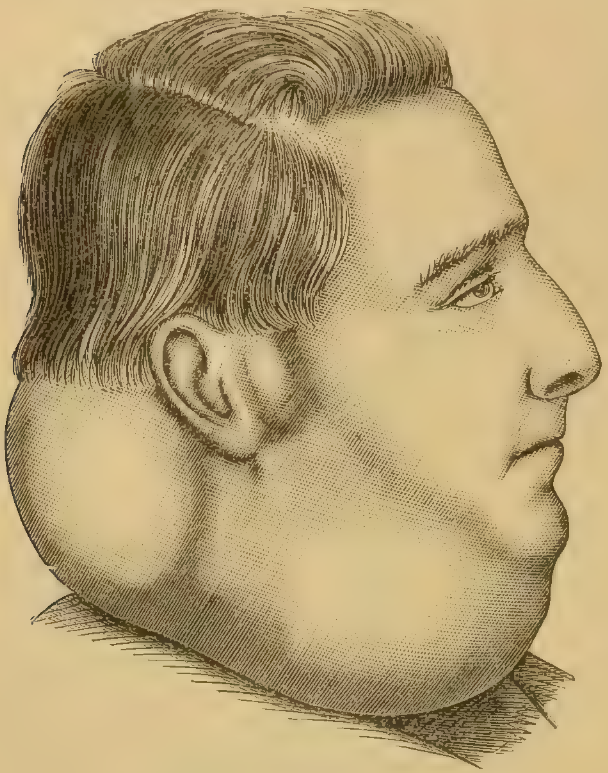

Fig. 98.—Diffuse Lipoma. (After Morrant Baker.)

2. **Subserous lipomata.**—The peritoneum, like the skin, rests upon a bed of fat. In this subserous fatty deposit lipomata are prone to occur, and, like those of the subcutaneous species, they may be sessile, pedunculated, or diffuse. When arising in the perirenal fat or in the mesentery, lipomata may attain prodigious proportions—fifty or sixty pounds. Exceptionally large fatty tumours have been encountered between the layers of the broad ligament of the uterus, simulating ovarian cysts. Sub-peritoneal fatty tumours sometimes drag upon the peritoneum in the neighbourhood of the inguinal and femoral rings, and produce finger-like diverticula of this membrane, which, when surrounded by fat,

are often mistaken for ruptures; they are known as "fatty herniæ." Occasionally a fatty hernia forms a swelling as large as a bantam's egg in the scrotum or labium, and when exposed is not unlike omentum. Subserous lipomata associated with small peritoneal pouches are not uncommon in the linea alba in the vicinity of the umbilicus. (*See* Art. XLVIII., on Hernia, Vol. II.)

Pedunculated subserous lipomata are usually associated with the colon; they are for the most part very large arborescent epiploic appendages.

3. **Subsynovial lipomata.**—Many synovial membranes have fat in the deeper layers. This fat may increase in quantity and cause the serous membrane to bulge into the joint until the mass becomes distinctly pedunculated—a subsynovial lipoma. This occurs most frequently in association with the alar ligaments of the knee-joint. The unusual variety to which J. Müller gave the name "lipoma arborescens" consists of a number of small pedunculated fatty fringes projecting into the synovial cavity. This condition is often associated with chronic joint disease. The processes have the same relation to the synovial membrane that epiploic appendages bear to the peritoneum.

4. **Submucous lipomata.**—This species rarely attains a large size. Submucous fatty tumours have been met with in the stomach, jejunum, and rectum; beneath the mucous membrane of the lips, the larynx, and the conjunctiva. Many of the cases in the larynx are pedunculated; in the intestine they may possess stalks, and have caused intussusception.

5. **Intermuscular lipomata.**—The connective tissue between muscles is often provided with fat, and is the source of lipomata, not only in the trunk but also in the limbs. The sucking-cushions in emaciated children sometimes become enlarged and simulate lipomata; they also occasionally enlarge in adults when the parotid duct is obstructed by a salivary calculus. Intermuscular lipomata in the limbs are occasionally connected with the sheaths of nerves—neuro-lipomata.

6. **Intramuscular lipomata.**—Many examples of fatty tumours occurring within muscles have been recorded. They are of interest from the trouble they cause in diagnosis. They have been observed in the deltoid, biceps (of the arm), complexus and rectus (of the abdomen); in the walls of the cardiac ventricles, and in the centre of pedunculated myomata of the uterus.

7. **Periosteal lipomata.**—This species arises from the periosteum of bone. They are usually congenital, and nearly always contain tracts of striated muscle fibre. Periosteal lipomata are commonest on the scapula and innominate bones; they have been observed on the clavicle, humerus, radius, ulna, femur, tibia, fibula, cervical vertebræ, and frontal bone. Periosteal, as well as the subcutaneous species, are sometimes so vascular that they resemble nævi; occasionally they are termed nævo-lipomata.

8. **Meningeal lipomata.**—Fatty tumours occur within as

well as on the external surface of the spinal dura mater. Lipomata within the dura mater often contain tracts of striated muscle fibre. Fatty tumours are not uncommon in the middle line of the back, especially in the lumbo-sacral regions overlying the sacs of spinæ bifidæ (Fig. 99). Intradural lipomata may be associated with the masked variety of spina bifida (spina bifida occulta).

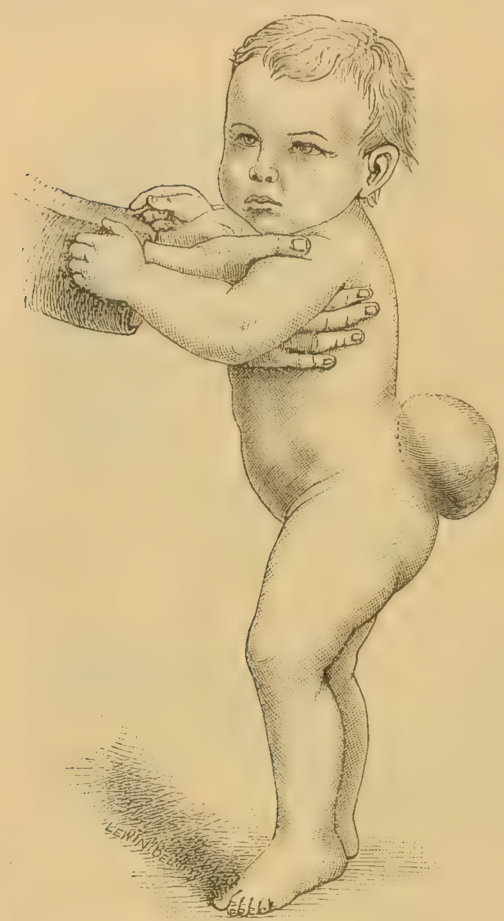

Fig. 99.—Meningeal Lipoma simulating Spina Bifida. (Témoin.)

Clinical features.—In most instances fatty tumours admit of almost certain diagnosis, especially the subcutaneous species: their lobulation, indefinite boundaries, and intimate union with the skin cause them to be readily recognised. In the groin they are apt to be confounded with herniæ, or even with abscesses. When seated on the face or scalp (rare situations) they are easily confounded with sebaceous cysts. When a lipoma grows from the periosteum of a long bone it will simulate a sarcoma: embedded in a muscle it will lead to most divergent opinions. In the middle line of the back it is very liable to be confounded with spina bifida, or a dermoid cyst, and will then require much circumspection in treatment. A lipoma within the abdomen is almost invariably a clinical puzzle.

Treatment. — Subcutaneous lipomata are easily enucleated: if pedunculated, their detachment is very simple. The diffuse variety is rarely submitted to operation. When fatty tumours are situated where they are liable to irritation from petticoat-bands, braces, and other articles of clothing, they should be excised. The removal of a subcutaneous lipoma is one of the simplest proceedings in surgery, but the extirpation of a large subperitoneal fatty tumour is invariably a difficult matter, and often attended with grave risks.

2. Chondromata (Cartilage Tumours).

Chondromata (enchondromata) are tumours composed of hyaline cartilage. This genus contains three species: (1) Chondromata; (2) ecchondroses; (3) loose cartilages in joints.

1. **Chondromata.**—Cartilage tumours in their most typical condition occur in connection with long bones, especially of the hands; and, as a rule, grow in the immediate vicinity of the epiphysial lines, hence they are most frequent in children and young adults; they are often multiple (Fig. 100), but a solitary chondroma is not a rarity. Individuals with multiple chondromata often present evidences of rickets, and it is a curious circumstance that the tissue of a chondroma resembles, histologically, the bluish translucent cartilage so characteristic of a rickety epiphysial line. These tumours are always encapsuled, and form deep hollows in the bones with which they are connected: they are painless, grow slowly, and are firm to the touch. Mucoid degeneration is common in them, and the softened parts may furnish slight fluctuation: this serves to distinguish them from osteomata with which

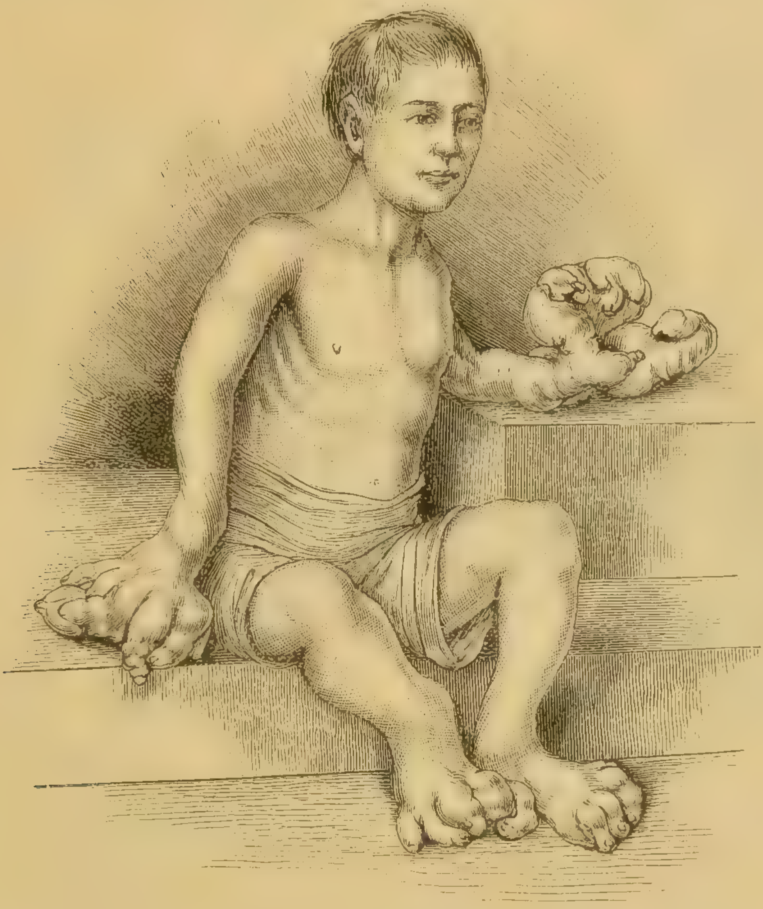

Fig. 100.—Multiple Chondromata. The lad was stunted from rickets.

they are apt to be confounded in diagnosis. Chondromata are prone to ossify.

Tumours of the testis and parotid which contain cartilage are not chondromata, but chondrifying sarcomata.

2. **Ecchondroses.**—These may be defined as small local outgrowths of cartilage: they are very common along the edges of the articular cartilage of the knee-joint; ecchondroses are not rare in relation with the triangular cartilage of the nose, and are of occasional occurrence in connection with the laryngeal cartilages.

3. **Loose cartilages.**—Bodies of various kinds are found loose in the cavities of joints, but those to be considered under the head of chondromata, in addition to detached ecchondroses, are pieces of hyaline cartilage found hanging from the synovial membrane by narrow stalks, or occupying depressions in the bone, from which they are occasionally dislodged. The origin of these loose cartilages will be dealt with in the article devoted to Diseases of Joints. (Art. XXXIII.)

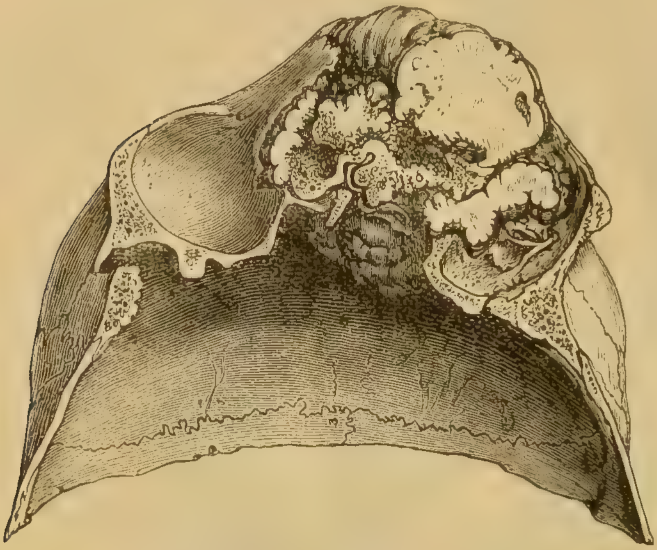

Fig. 101.—Osteoma of Frontal Sinus. (Museum, Royal College of Surgeons.)

Treatment.—The operative treatment of chondromata has been greatly simplified since surgeons have appreciated the fact that these tumours are encapsuled. Hence when it is necessary to interfere with a chondroma, even in cases where several of these tumours are present, it has become customary to incise the capsule and enucleate the tumour: this simple method is very successful. Exceptionally, cases come under observation demanding more serious measures: for instance, in such a case as is represented in Fig. 97, the hand may become so burdensome that patients have asked to have the limb amputated, and their wish has been complied with.

3. Osteomata (Osseous Tumours).

An osteoma may be defined as an ossifying chondroma. The genus contains two species: (1) Compact or ivory osteoma; (2) cancellous osteoma.

Osteomata, like chondromata, are commonly met with in the neighbourhood of epiphysial lines, and in the case of skull bones at parts where large tracts of hyaline cartilage exist in the fœtal skull. Every growing osteoma has a cap of hyaline cartilage which stands in the same relation to the growth of the tumour that an epiphysial line bears to a bone. (*See* Art. XXX.)

1. **Compact osteomata.**—These are structurally identical with the compact tissue forming the shaft of a long bone; often it is as dense as the petrosal. Although they may occur on any bone they are more frequent in the frontal sinus (Fig. 101), the external and internal auditory meatuses, and on the mastoid process than elsewhere. They are occasionally met with on the angle of the mandible; many supposed osteomata of the maxillæ are odontomes. Compact osteomata are usually sessile.

2. **Cancellous osteomata.**—These resemble in structure the cancellous tissue of bone, and are soft in comparison with the compact species. When growing from the distal end of the radius or tibia, they are often deeply channelled for the passage of flexor and extensor tendons. Cancellous osteomata may be sessile or pedunculated: frequently the summit is surmounted by a bursa.

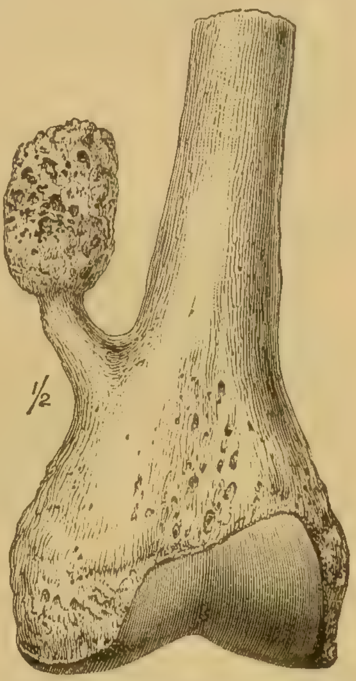

Fig. 102.—Exostosis of the Femur, produced by ossification of the tendon of the adductor magnus. (Museum, Royal College of Surgeons.)

Exostoses.—It is necessary to mention that all irregular bony outgrowths are often vaguely classed as exostoses. The conditions which should be classed under this name, and sharply excluded from osteomata, are:—1, ossification of tendons at their attachments (Fig. 102); 2, the subungual exostosis; 3, calcified inflammatory exudations.

Many macerated preparations of bones preserved as examples of multiple exostoses are from individuals affected with chondromata, some of which had partially ossified. Further, some of the skeletons supposed to be examples of multiple exostoses are really instances of the rare disease—myositis ossificans. (Art. XXXIV., on INJURIES AND DISEASES OF MUSCLES, TENDONS, FASCIÆ, AND BURSÆ, Vol. II.)

Treatment.—Osteomata, in situations where they do not involve important structures, rarely demand treatment. When they press upon important nerves, obstruct the external auditory meatus, and are clearly the source of inconvenience, deformity, or distress, they should be removed. When growing near large joints, great care must be exercised in removing them. Osteomata of the ivory species, when sessile, sometimes require very persevering efforts on the part of the surgeon, aided by the best surgical cutlery. When an osteoma grows from the roof of the orbit, it should be remembered that it often projects deeply into the cranial cavity.

4. ODONTOMATA (TOOTH TUMOURS).

Odontomes are tumours composed of dental tissues in varying proportions and different degrees of development, arising from teeth-germs, or teeth still in the process of growth.

The species of this genus, determined according to the part of the tooth-germ concerned in their formation, are :—(1) Epithelial odontome (from the enamel organ) ; (2) follicular odontome ; (3) fibrous odontome ; (4) cementome ; (5) compound follicular odontome (from the tooth follicle) ; (6) radicular odontome (from the papilla) ; (7) composite odontome (from the whole germ).

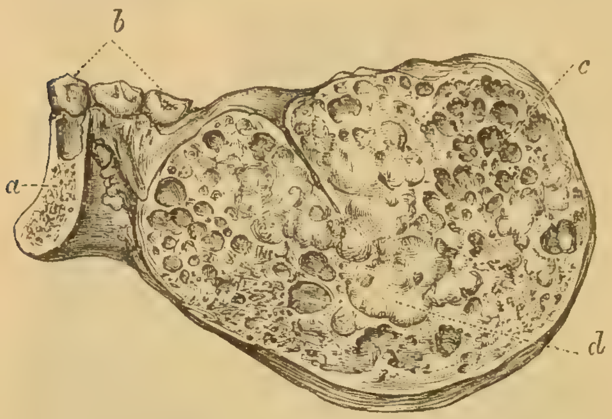

Fig. 103.—Epithelial Odontome. (After Pepper.)
a, Divided bone ; *b*, teeth ; *c*, cysts containing a glairy fluid ; *d*, lobules of the growth.

1. **Epithelial odontomes.** — These occur as encapsuled tumours in the mandible, and less frequently in the maxillæ. In section they are seen to be made up of congeries of cysts of various shapes and sizes. The loculi rarely exceed 2 cm. in diameter. The walls of individual loculi are sometimes ossified. The cavities contain mucoid fluid of a brownish colour (Fig. 103).

Histologically, an epithelial odontome consists of branching and anastomosing columns of epithelium, portions of which form alveoli. The cells occupying the alveoli vary. Thus the outer layer may be columnar, whilst the central cells degenerate, and give rise to tissue resembling the stratum intermedium of an enamel organ (Fig. 104).

2. **Follicular odontomes.**—This species includes those tumours commonly called "dentigerous cysts," a term which is so often confounded with tooth-containing dermoids that it is advisable to discard it.

Follicular odontomes are usually, but by no means invariably, associated with the permanent teeth, especially the molars, and sometimes attain very large proportions and produce great deformity. Occasionally they are multiple, especially when connected with the maxillary teeth. The tumour consists of a wall of varying thickness. Thus it may be thin and crepitant, or measure 2 cm. in thickness.

This cyst wall represents the follicle of an unerupted tooth. The cavity of the cyst is occupied by viscid fluid and the crown of an unerupted tooth. Exceptionally the tooth is loose in the cyst. It may be inverted; usually the root is truncated—incompletely developed, due to non-eruption (Fig. 105)—and rarely the tooth is absent. The cyst wall contains calcareous matter, and occasionally bone.

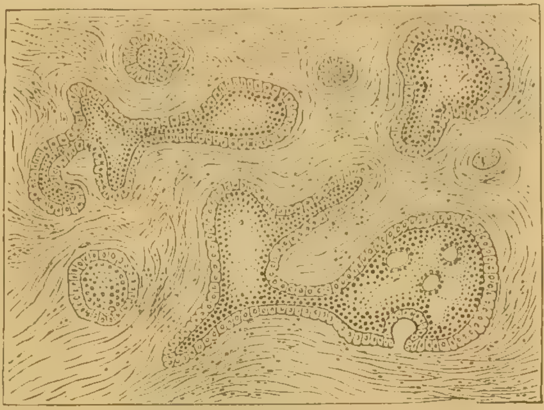

Fig. 104.—Microscopical Characters of an Epithelial Odontome.

3. **Fibrous odontomes.**— Every tooth before its eruption is enclosed in a capsule of fibrous tissue—the tooth sac. This capsule occasionally becomes thickened, and so thoroughly encysts the tooth that it is never erupted, and is sometimes completely suppressed. Microscopically these odontomes are made up of wavy laminæ of fibrous tissue, intermixed with calcareous matter. This species has in the past been confounded with myeloid sarcoma.

4. **Cementomes.**—When the tooth sac becomes enlarged, as in the preceding species, and the fibrous tissue ossifies, the tooth becomes embedded in a mass of hard tissue identical with cementum. An odontome of this character is called a cementoma. At present they are unknown in man, but are met with in horses, and in these animals grow to a very large size. Specimens are known weighing fifty, sixty, and seventy ounces.

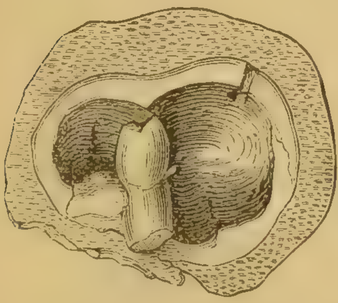

Fig 105.—Follicular Odontome.

5. **Compound follicular odontomes.**—When the thickened capsules of one or more unerupted teeth become confluent and ossify sporadically, a tumour is produced containing one or more denticles and fragments of cementum. Such tumours are known as compound follicular odontomes. Several examples have been carefully recorded. Some of these tumours have contained twenty and others forty denticles. In a few of the recorded cases some of the denticles erupted and stood above the gum. On their removal, other ill-formed teeth appear, and then a thorough search revealed the existence of a collection of denticles within a capsule. Compound follicular odontomes occur in the maxilla as well as in the mandible.

6. **Radicular odontomes.**—This species arises after the crown of the tooth has been completed, and while the roots are in process of formation. As the crown of the tooth when once formed is unalterable, it naturally follows that should the roots develop an odontome, enamel cannot enter into its composition; the tumour would consist of dentine and cementum in varying proportions.

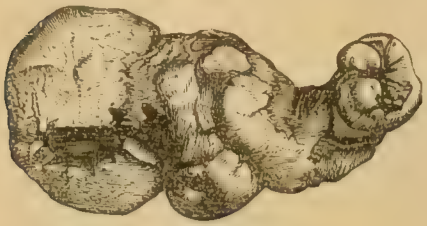

Fig. 106.—Radicular Odontome from the Maxilla. Natural size. (After John Tomes.)

Radicular odontomes are occasionally met with in man (Fig. 106); they occur equally in maxilla and mandible. In rodents they are very common and occur in multiples; four may be present in the jaws of the same animal.

7. **Composite odontomes.**—These tumours bear very little resemblance in shape to teeth (Fig. 107), but consist of a disordered conglomeration of dentine, enamel, and cementum, and may be considered as arising from abnormal growth of all the elements of a tooth-germ. They are composite in another sense, for the majority of specimens consist of two or more teeth-germs indiscriminately blended. They occur in both jaws with equal frequency, but attain far larger proportions in the maxilla than in the mandible. The clinical characters of odontomes are described in the article devoted to DISEASES OF THE JAWS (Art. XXXI.).

5. FIBROMATA (FIBROUS TUMOURS).

Fibromata are tumours composed of fibrous tissue. This genus contains three species:—(1) Simple fibromata; (2) molluscum fibrosum; (3) neuro-fibromata.

1. **Simple fibromata.**—Typical fibromata consist of wavy bundles of dense fibrous tissue; the bundles are composed of long, slender, fusiform cells closely packed together, and frequently arranged in whorls, the large blood-vessels of the tumour traverse the centre of the vortices. Fibromata occur in the ovary, intestine, and larynx, on the gum (epulis), in the uterus, and in the deeper layers of the skin. In the last-named situation they are known as "painful subcutaneous tubercles."

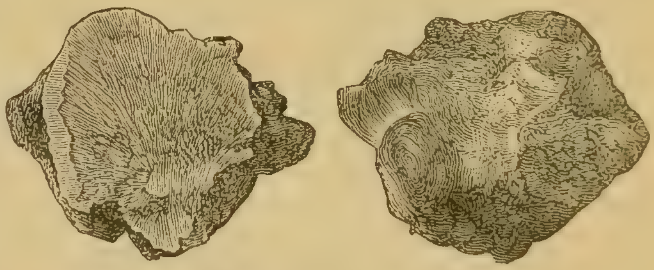

Fig. 107.—Composite Odontome from the Upper Jaw. (After Jordan Lloyd.)

2. **Molluscum fibrosum.**—This extraordinary condition of the

skin has been described under a variety of names as *fibro-cellular tumour*, *pachydermatocele* (Mott), and *dermatolysis.* It consists of an overgrowth of skin and subcutaneous tissue, which may affect a small area like the scalp, or involve a large extent of skin on the trunk and limbs, causing it to hang in pendulous folds (Fig. 108). Sometimes it assumes the form of discrete nodules scattered over the skin, the nodules varying in size, the extremes being represented by a pea and a walnut. Nodules and pendulous folds are occasionally associated in the same individual, and several cases have been reported in which molluscum nodules have been associated with a multitude of neuromata on the principal nerve-trunks.

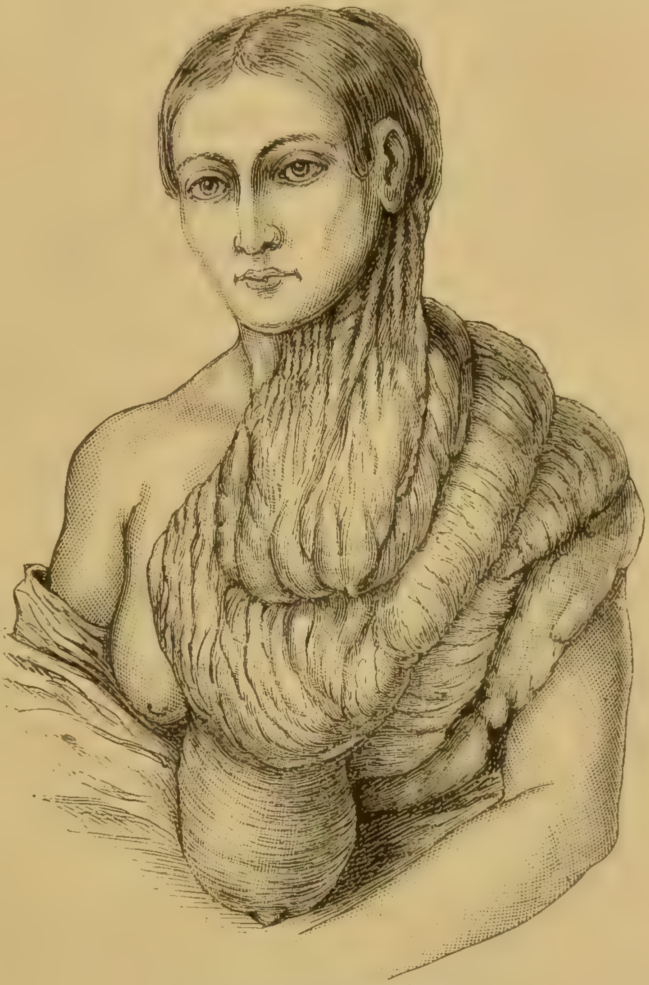

Fig. 108.—Molluscum Fibrosum.
This tumour was successfully removed by Mott.

The histology of the nodules and the pendulous flaps is similar, and appears to be represented by an overgrowth of the fibrous tissue of the skin and the subcutaneous tissues generally. Concerning the cause of this overgrowth nothing is known; it affects men and women equally, and is not confined to any race or clime.

In many cases in which the disease has been confined to a limited area the pendulous masses have been successfully excised, and the subsequent records of the patients have demonstrated that the disease shows no disposition to recur or to manifest itself in any other region of the body. It will be necessary to refer again to molluscum fibrosum in dealing with neuromata.

6. Myxomata.

Myxomata are tumours composed of a tissue identical with that which surrounds the vessels of the umbilical cord.

The genus contains three species:—(1) Nasal and aural polypi; (2) cutaneous myxomata; (3) neuro-myxomata.

Myxomatous tissue is in many instances the result of degenerative changes in fibrous, cartilaginous, and sarcomatous tissues. Its microscopical characters may be best studied in a nasal polypus. A nasal myxoma has a capsule of mucous membrane covered with columnar ciliated, cubical, or stratified epithelium; sometimes the three varieties may be demonstrated on one tumour. Immediately beneath the mucous membrane there is a firm layer of fibrous tissue, which gradually becomes continuous with a gelatinous mass composed of cells furnished with long delicate branching processes—spider cells. (*See* Art. XLII., on Diseases of the Nose and Nasal Cavities, Vol. II.)

Cutaneous myxomata may be either pedunculated or sessile. A large sessile myxoma when cut into resembles a mass of pale trembling jelly, from which a viscid fluid of a pale straw colour drains away.

Neuro-myxomata will be described with neuromata.

7. Gliomata.

Gliomata are tumours composed of the delicate connective tissue known as neuroglia. The genus consists of a single species—glioma.

Gliomata only occur in the central nervous system. Tissue very similar to neuroglia forms the sustentacular framework of the retina, and is the seat of sarcomata, which are often termed retinal gliomata.

In the brain a glioma occurs as a tumour imperfectly demarcated from the surrounding tissue. It may appear as a translucent swelling of the consistence of vitreous humour, or it may be as firm as the tissue of the pons. As a rule, a glioma is of the same firmness as the cerebral cortex. (*See* Art. XXXVII., on Diseases of the Head, Vol. II.)

Structurally, gliomata consist of cells furnished with delicate ramifying processes; the cells, which may contain one or more nuclei, are mixed with fibrous tissue. The proportion of cells to the fibrous tissue varies greatly; sometimes one set of elements preponderates, sometimes the other. These tumours are often very vascular, the vessels being irregularly dilated and occasionally sacculated. The number of blood-vessels in some specimens is so great that the tumours are described as angeiomata or angeio-sarcomata.

As a rule, gliomata are solitary and do not give rise to secondary deposits. In certain situations they rather resemble diffuse overgrowths than tumours. Virchow pointed out that when a glioma is situated near the surface of the cortex it will appear like a colossal convolution. Should it grow in the tissue of an optic thalamus, it would cause the thalamus to bulge into the third ventricle as though overgrown, and a glioma of the occipital lobe will project into the descending cornu like an additional thalamus. The best illustration of the indefiniteness so characteristic of a glioma comes out in a striking manner in the variety known as "gliomatous enlargement of the pons," which is occasionally met with in children between six and twelve years of age.

Gliomata are very rare tumours in the spinal cord. Judging from the scanty records, a glioma is twenty times more frequent in the brain than in the spinal cord.

8. NEUROMATA.

Neuromata are tumours growing from, and in structure resembling the sheath of, a nerve. The genus contains three species:—(1) Neuro-fibroma; (2) plexiform neuroma; (3) traumatic neuroma.

1. **Neuro-fibromata.** — Structurally, neuro-fibromata consist of connective tissue continuous with that of the nerves from which they grow. They are furnished with a distinct capsule, which is continuous with the neurilemma, and from which the tumour is easily enucleated.

Neuro-fibromata are very liable to myxomatous degeneration, and in large specimens this change may lead to the formation of cavities in the tumours. (*See* Fig. 94.) This accounts for the various names applied to neuromata, such as myxoma, myxo-fibroma, cystic myxoma, myxo-sarcoma, and neuro-myxoma.

2. **Plexiform neuromata.** — This species is, in comparison with the preceding, somewhat rare. Instead of forming distinct tumours as in the case of simple neuromata, it seems as if the branches of a nerve distributed to a particular area became thickened and elongated. Such nerves in section have an appearance like that presented by the umbilical cord, due to the presence of a large quantity of myxomatous tissue in the sheath of the nerve. Plexiform neuromata are, as a rule, congenital, and are apt to arise in pigmented moles. Sometimes the thickened nerves are embedded in tissue similar to that which forms the folds in molluscum fibrosum.

3. **Traumatic neuromata.** — This species comprises the "bulbs" which form on the proximal ends of divided nerves, and on nerve ends in amputation-stumps.

A fuller consideration of neuromata will be found in the article dealing with diseases of nerves (page 698); but it is necessary to point out the difficulty which surrounds the classification of neuromata. The term has been in use since the early years of this century to indicate any tumour of a nerve. The advance of histological knowledge has demonstrated that some nerve-tumours are fibrous, others are myxomatous, while a few are sarcomatous in structure. Some even consist of fat. Thus it would be better to speak of fibroma, myxoma, sarcoma, and lipoma of nerves as we speak of sarcoma or chondroma of a bone.

It is of interest to draw attention to the fact that the gelatinous tissue in the sheaths of the enlarged trunks of a plexiform neuroma bears the same relation to the nerve that a glioma bears to the brain, and this species might not inaptly be called "glioma of a nerve."

The peculiar relationship between multiple neuromata and the two varieties of molluscum fibrosum—the nodular and the pendulous—has been referred to in the section on fibromata (page 458). This much is clear, the neuromata constitute a mixed and, in a sense, a bastard genus; nevertheless, it is necessary to retain the class for a time at least.

9. ANGEIOMATA. 10. LYMPHANGEIOMATA.

Angeiomata are tumours composed of an abnormal formation of blood-vessels. This genus contains three species:—(1) Simple nævus; (2) cavernous nævus; (3) plexiform angeioma.

The angeiomata are considered in the article devoted to the DISEASES OF BLOOD-VESSELS (Art. XXIV. page 517), and lymphangeiomata in Art. XXVI., on DISEASES AND INJURIES OF LYMPHATICS (pages 663 and 679).

11. MYOMATA.

Myomata are tumours composed of unstriped muscle-fibre. The genus contains one species—myoma. They occur in the uterus: occasionally they are found in connection with the œsophagus, stomach, small intestine, bladder, and prostate; more rarely they grow in connection with the dartos tissue of the scrotum and the subcutaneous tissue of the scalp. Myomata sometimes arise in the broad ligament of the uterus, the ovary, and the ovarian ligament, and very rarely from the Fallopian tube.

Myomata occur as encapsuled tumours composed of long fusiform cells, with a rod-like nucleus. The bundles of muscle-fibres are often interwoven in such a manner that the cut surface presents a characteristic whorled appearance.

It is sometimes very difficult to distinguish between a myoma, a fibroma, and a spindle-celled sarcoma; for instance, uterine myomata were formerly called "uterine fibroids," and spindle-celled sarcomata "recurrent fibroids"; but increased accuracy in histological methods has in a very large measure served to dissipate this confusion. It is important in doubtful cases to remember that myomata only arise from pre-existing unstriped muscle-fibre. Spindle-celled sarcomata are rare in situations favourable to myomata.

Tumours composed of unstriped muscle very frequently occur in the uterus, and are the commonest tumours to which women are liable. The uterus consists almost entirely of unstriped muscle, and the larger part of the organ is covered externally by peritoneum: where this membrane is in relation with the uterus, its subserous layer (which in many parts of the abdomen consists of fat-containing connective tissue) is here replaced by a layer of unstriped muscle-fibre, which is directly continuous with the muscle-tissue of the broad ligaments. The cavity of the uterus is lined with mucous membrane, the deep layer of which is so rich in muscle-fibre that it is impossible to determine the limit between the mucous membrane and the true wall of the uterus. Thus there are three situations in the uterus in which myomata may arise: (1) in the true uterine tissue—*intramural myomata*; (2) in the submucous tissue—*submucous myomata*; (3) in the subserous layer—*subserous myomata*

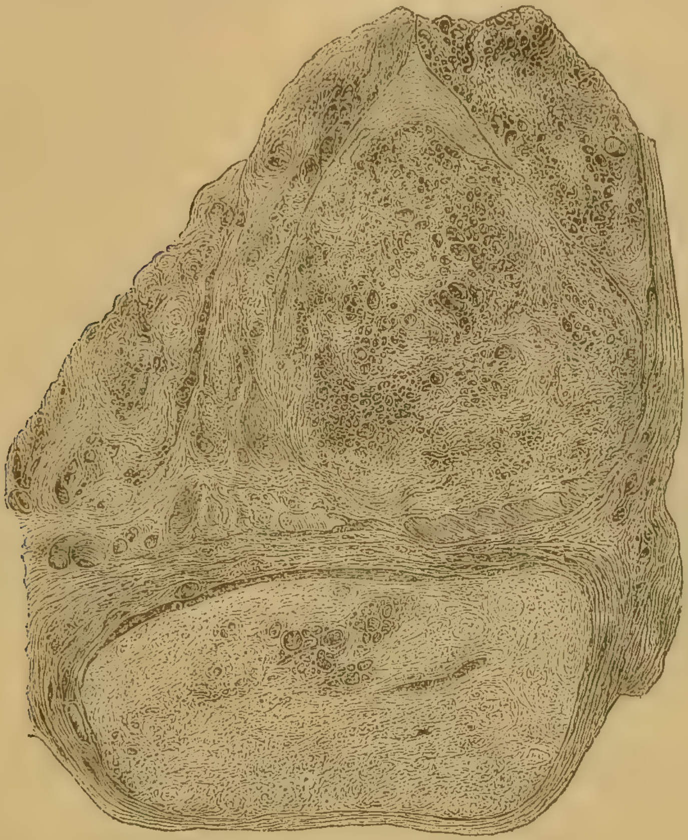

Fig. 109.—A very Vascular Myoma. (After Virchow.)

Muscle tumours may arise in, and remain confined to, any one of these layers; or they may arise in all three in the same individual. Submucous and subserous myomata rarely attain a large size, they quickly become pedunculated and project into the peritoneal cavity in the case of the subserous variety, and into the uterine cavity when originating in the submucous tissue. It must also be borne in mind that intramural myomata not infrequently project into the uterine or peritoneal cavities, acquire stalks, and simulate those which arise in the submucous and subserous layers. When intramural myomata become pedunculated and project, either on the serous or mucous aspect of the uterus, they attain larger proportions than those which arise in the subserous or submucous strata.

It not infrequently happens that when a myoma is confined to one wall of the uterus, and appears as a single tumour externally, it will be found, on section, to consist of two or more tumours growing in association, but each possessing its own capsule. This holds good of many specimens described as "general myomatous enlargement of the uterus," in which the organ is so uniformly enlarged as to resemble a gigantic pear. Myomata arise in any part of the uterus, but they are most frequent in the body and fundus. They are rare in the cervix, but in this situation very large examples are sometimes met with. The cervix is frequently involved in myomata arising in the anterior or posterior wall of the uterus.

Myomata vary greatly in their rate of growth; those which grow slowly are, as a rule, very hard, and contain a large proportion of fibrous tissue: hard tumours rarely exceed the size of a fist, and if they become pedunculated, do not commonly grow big. The softer specimens contain but little fibrous tissue, the cells are large, they grow rapidly, are very vascular, and often furnish a loud systolic murmur (Fig. 109).

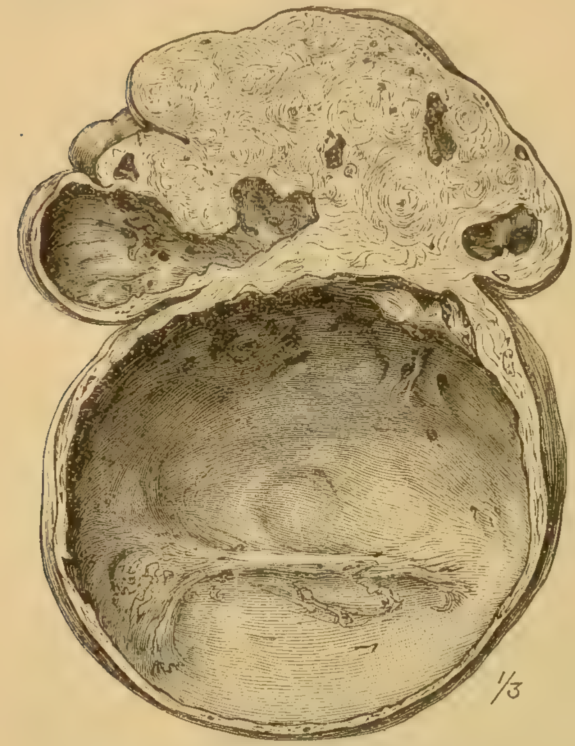

Fig. 110.—Cavities, the result of Mucoid Degeneration of a Uterine Myoma. (Museum, Middlesex Hospital.)

Myomata, when they project into the uterine cavity, cause marked hypertrophy of the walls of the uterus; when very large, and especially when projecting from the fundus, they distort the uterine cavity. In many cases the tumour gradually dilates the cervical canal and is extruded from the uterus into the vagina, and may even protrude from the vulva. Occasionally the pedicle is broken and the tumour discharged. This is known as spontaneous detachment of a uterine myoma. When a myoma occupies the cavity of the uterus, whether it arise in the mucous membrane or wall of the organ, it is in part invested by mucous membrane: so long as the tumour remain in the uterine cavity this mucous membrane retains its columnar epithelium; when the tumour protrudes into the vagina the cells covering the exposed portion become stratified, except in the glandular recesses, where they retain the columnar ciliated form. It is important to bear in mind that a myoma projecting into the uterine cavity will sometimes invert the fundus of the uterus. Intramural myomata, especially when they remain sessile, often attain great size, and, rising out of the pelvis, may even reach as high as the diaphragm and weigh sixty, seventy, or more pounds.

Uterine myomata are liable to secondary changes; they may undergo mucoid degeneration, and large tracts may become converted into a viscid fluid surrounded by a capsule, and thus resemble a cyst (Fig. 110). Thus altered, they are often spoken of as cystic myomata. Fatty metamorphosis is a rarer change; in some rare instances a collection of fat has been found in the middle of a pedunculated myoma. Calcification is a common change, the calcareous matter is not deposited irregularly, but follows the peculiar whorls of the muscle tissue (Fig. 111).

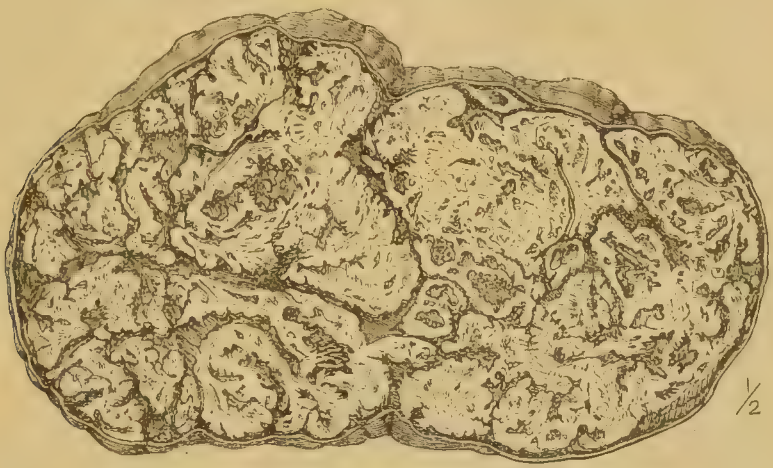

Fig. 111.—Calcified Uterine Myoma, in section. (Museum, Middlesex Hospital.)

It occasionally happens that a myoma, which has existed many years and given rise to little inconvenience, suddenly enlarges, grows rapidly, assumes formidable proportions, and gives rise to constitutional disturbance. These changes are analogous to those which occur when a submucous myoma extrudes from the cervix and becomes gangrenous, that is, they are due to septic infection.

The occurrence of these changes leads to the infiltration of the tumour with leucocytes and round cells; thus sections prepared from it resemble those from a round-celled sarcoma, and there can be little doubt that in many specimens described as "sarcomatous degeneration of uterine myomata," the changes in and the rapid growth of the tumours were the result of septic infection.

The clinical characters of uterine myomata are discussed in the section devoted to the Diseases of the Female Genital Organs (Art. LIII. Vol. II.).

12. SARCOMATA.

Sarcomata are tumours composed of tissues resembling immature connective tissue, in which cells preponderate over the intercellular substance. This genus contains six species, which are determined according to the shape and disposition of the cells: (1) Round-celled sarcoma; (2) lympho-sarcoma; (3) spindle-celled sarcoma; (4) myeloid sarcoma; (5) alveolar sarcoma; (6) melano-sarcoma.

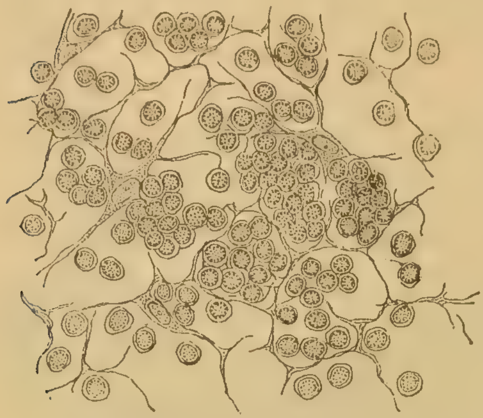

Fig. 112.—Microscopical Characters of a Lympho-sarcoma, from the mediastinum.

1. **Round-celled sarcoma.**—This species consists of round cells with very little intercellular substance. Each cell has a large round vesicular nucleus, and a small proportion of protoplasm. Blood-vessels are abundant, and often appear as mere channels between the cells; lymphatics are absent. These tumours grow very rapidly, infiltrate surrounding tissues, become disseminated, and recur after removal.

There is a variety known as the "large round-celled sarcoma;" the cells are of unequal size, many contain two or more nuclei; a few are multinuclear and resemble myeloid cells.

The round-celled sarcoma is the most generalised tumour which affects mankind; it may occur in any organ, in bone, brain, muscle, spinal cord, ovary, or testis, and even in the delicate sustentacular framework of the retina. It attacks the body at all periods of life, from the fœtus or the child just born up to the extreme limits of age. Among vertebrate animals it is ubiquitous.

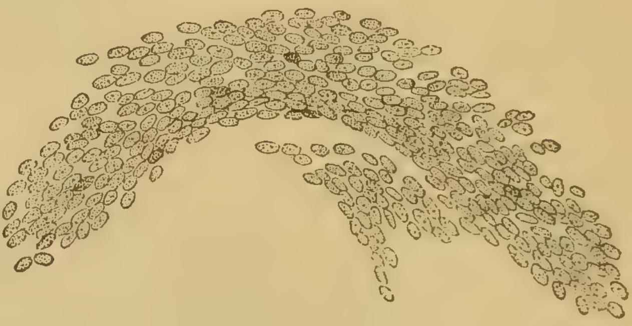

Fig. 113.—Small Spindle-celled Sarcoma from a Metacarpal Bone.

2. **Lympho-sarcoma** consists of cells identical with those of round-celled sarcomata, but the cells are contained in a delicate mesh, and resemble the tissue of lymphatic glands (Fig. 112); hence the origin of the term. These tumours must not be confounded with simple (irritative) enlargement of lymphatic glands, nor with the general overgrowth of lymphadenoid tissue associated with leukæmia or lymphadenoma (Hodgkins' disease). (*See* page 674.)

3. **Spindle-celled sarcoma.**—The cells of the species classed under this head vary considerably in size, but they agree in the circumstance that they are oat-shaped (Fig. 113) or fusiform. The cells have a tendency to run in bundles, which take different directions, so that in sections of the tumour seen under the microscope some cells will be cut in the direction of their length and others at right angles. This must be borne in mind, or an incorrect opinion will be formed as to the nature of the tumour.

The following facts will afford some idea as to the degree of variation in size of the cells of spindle-celled sarcomata. In some of the tumours the cells are so thin, slender, and contain so little protoplasm that they seem to consist only of a nucleus and cell processes. It is difficult to distinguish such cells from those of moderately-firm fibrous tissue.

In other specimens the cells are large, beautifully fusiform, and rich in protoplasm. They give rise to considerable difficulty to the morbid anatomist, and he often feels incompetent to decide between such cells and those of young unstriped muscle-fibre. The complexity of such tumours is further increased by the fact that occasionally these long spindle-cells are transversely striated like voluntary muscle-fibre. This variety is sometimes called *myo-sarcoma* (Fig. 114).

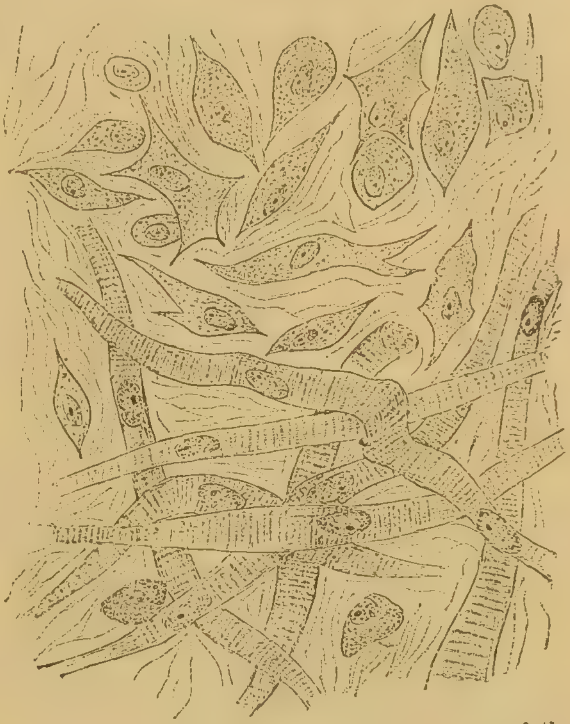

Fig. 114.—Cells from a Spindle-celled Sarcoma of the Neck of the Uterus. Some of the cells have a cross-striation. (After Pernice.)

Another peculiarity of spindle-celled sarcomata is the frequent presence of tracts of immature hyaline cartilage; in some instances this tissue constitutes so large a proportion of these tumours that they are described as chondromata; the cartilage is sometimes calcified and even ossified. When cartilage is very abundant in a sarcoma it is termed a *chondro-sarcoma*. In slow-growing examples of this species the spindle-cells become converted into fibrous tissue; such are often called *fibro-sarcomata*. Spindle-celled sarcomata often contain round cells and even large multinuclear cells.

4. **Myeloid sarcoma.**—This species is composed of tissue histologically resembling the red marrow of bone. When fresh, the cut surface of the tumour is of a deep red or maroon colour, and looks not unlike a piece of liver. The tissue consists of large numbers of multinuclear cells embedded in a matrix of spindle or round cells (Fig. 115). Many central tumours of bone contain large multinuclear cells, but it is only when these large cells are present in such quantity as to make up a large part of the tumour that they should be classed as myeloid sarcomata. Myeloid sarcomata only occur as central tumours of bones. (*See* Art. XXX.)

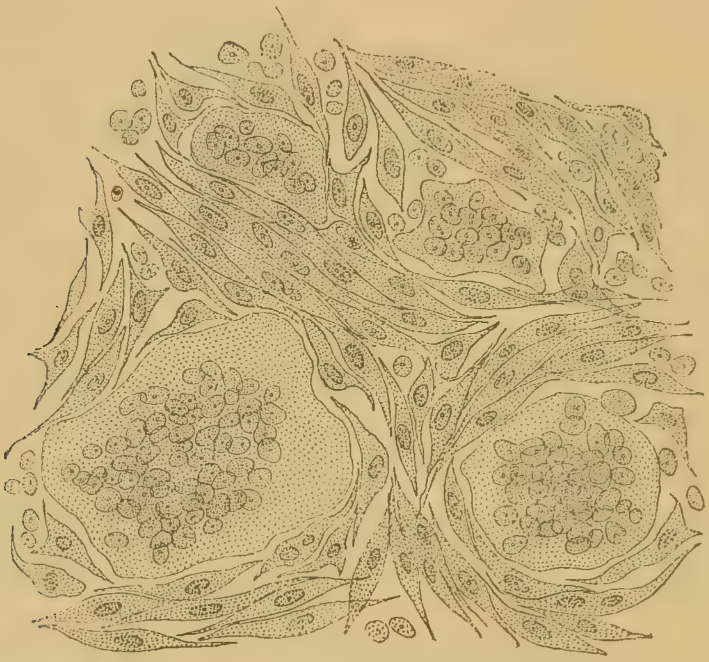

Fig. 115.—Microscopical Characters of a Myeloid Sarcoma, from the acromial end of the clavicle.

5. **Alveolar sarcoma.**—This is a peculiar species, inasmuch as the cells, contrary to the rule of sarcomata generally, assume an alveolar disposition, similar to that observed in sections of a carcinoma. Although they mimic the cancers in this respect, there is, however, one great point of difference—they do not imitate the structure of secreting glands. In carefully-prepared sections such tumours rarely cause difficulty, because the cells are usually of large size, and even when they resemble epithelium it is possible to distinguish a delicate reticulum between the individual cells, a condition never found in cancer.

Alveolar sarcomata have occasionally been described as growing in connection with bone, but their common situation is the skin, especially in relation with those congenital defects known as hairy and pigmented moles.

6. **Melano-sarcoma.**—Structurally this species may be composed of round or spindle cells. Occasionally they are arranged in alveoli. The distinguishing feature is the presence, in the cells and intercellular substance, of a variable quantity of black pigment.

It was formerly the custom to describe all varieties of melanoma as cancers. Now that the histological distinctions between sarcoma and carcinoma are more accurately defined, it is clear that the majority of pigmented tumours must rank as sarcomata.

In these tumours the amount of pigment varies greatly; in some there is only sufficient to produce a brown discoloration, others are jet black. Occasionally it happens that the primary tumour contains very little pigment, yet the secondary deposits are inky black. The pigment particles are lodged in and among the characteristic cells of the tumour, and also in its fibrous matrix and in the walls of the vessels.

Melano-sarcoma of the skin.—Two varieties occur in the skin. The most frequent arises in pigmented moles; the rarer in or near the nail-matrix of a finger or toe.

Melano-sarcomata occurring in moles differ from other melanomata in that the cells are collected in alveoli; the tissue forming the base of a mole, as a rule, presents an alveolar disposition, and this structural peculiarity comes out very strongly when a mole is the seat of a melanoma. (*See* page 718.)

A pigmented mole may remain quiescent throughout a very long life and never cause the least inconvenience; in other instances, fortunately rare, as life advances the mole ulcerates, perhaps bleeds freely, and may even become partially healed. Coincident, however, with the onset of ulceration the adjacent lymph-glands enlarge, become charged with pigment and sarcomatous tissue, spaces filled with black fluid form in them, and finally the overlying skin ulcerates. The infection may not proceed farther than this; recurrent hæmorrhage from the fungating glands or a furious bleeding, should a large vein or artery become broached by ulceration, carries off the patient. In many cases the morbid material is disseminated, and secondary knots form in the liver, lung, kidney, or brain, and death arises from interference with the function of these organs.

In other cases, instead of ulcerating, the mole becomes prominent and the lymph-glands in anatomical relationship with the part enlarge, and deposits at the same time occur in viscera, bones and skin.

It does not necessarily follow that in all cases of melanoma occurring in moles secondary deposits are formed in the viscera. In some rare cases the tumour seems to become mainly a source of pigment, large quantities of which enter the circulation, to be discharged with the urine in which it is recognised as melanin. Exceptionally the skin will assume a dusky tint.

Digital melanoma.—Melanosis in connection with the fingers and toes assumes two forms: it may occur as a deep pigmentation of the skin, usually in the immediate neighbourhood of the nail, and often involving the matrix, or even the nail itself; or as a small pigmented nodule arising in the nail-matrix or in the adjacent skin. These nodules quickly ulcerate, and dissemination follows. In some of these nodules the pigment is very scanty—indeed in some cases it is only discovered with the microscope; yet secondary nodules of an intense black colour will arise in the various organs and tissues.

The hallux is the digit most prone to be attacked by melanoma, and several examples have been fully recorded, most of the patients being women.

Intra-ocular melanoma. — Pigmented tumours arising within the eyeball belong to two genera, sarcoma and carcinoma. Of these, the melano-sarcoma is very much more frequent than the melano-carcinoma.

Melano-sarcoma may arise from any part of the uveal tract—that is, either from the posterior part of the iris, the ciliary body, or the choroid, posterior to the ciliary body. A melano-sarcoma of the iris is excessively rare, and such a tumour is ten times more common in the choroid than in the ciliary body. Intra-ocular melano-sarcomata have been observed as early as the fifteenth year, but the liability increases with age. By far the greater number occur between the fiftieth and sixtieth years: they have been met with as late as eighty-four.

Intra-ocular melano-sarcomata are very apt to recur after removal, and to become disseminated. The most frequent situation in which to find secondary deposits is the liver; but any organ may contain them, even the bones. It is surprising, considering that the eyeball is so near to, and in such close relation with the brain, by so large a nerve-trunk as the optic nerve, that it should be so rarely implicated. It is a fact that when the brain is the seat of deposit, it is excessively rarely the result of extension along the nerve. The amount of dissemination varies greatly; in some cases secondary knots occur in almost every organ; in others they will be limited to the liver. The lymph glands adjacent to the orbit are rarely infected. It is curious that in most cases death results more often from the secondary growths involving important organs than from the local effects of the primary tumour, in spite of its proximity to the brain.

Sarcomata of secreting glands.—The round and spindle-celled species of sarcomata are prone to arise in the parotid and submaxillary glands, the kidney, testis, ovary, and occasionally in the mamma. They have attracted great attention on account of the variety of tissues they sometimes contain. Thus spindle-celled sarcomata of the parotid gland almost invariably contain cartilage, myxomatous and connective tissues, entangling groups of ill-formed glandular acini. Similar tumours occur in the submaxillary gland. Of spindle-celled sarcomata growing in the testis, half contain tracts of hyaline cartilage. In the kidneys of infants sarcomata contain ill-formed glandular tubules, and tissue which in sections is indistinguishable from striated muscle. In the mammary gland these tumours contain, in addition to sarcomatous elements, hyaline cartilage and clusters of glandular acini. In the ovary clusters of ovarian follicles are found here and there embedded in the tumour. Many writers refer to these tumours as adeno-sarcomata, a term which needs to be strongly discouraged. It has already been pointed out that striated muscle-tissue occurs in spindle-celled sarcomata of other organs than glands, and it may be here mentioned that in the kidney, where myosarcomata are most frequently met with, when the striated cells are abundant, gland tubules are scanty, and often absent, and the converse of this is equally true. It has been urged that complicated sarcomata of glands should rank as a separate genus, but the convenience and justice of keeping them among sarcomata is indicated by the fact that when a myosarcoma is removed and recurs, the recurrent tumour contains only round and spindle-celled elements.

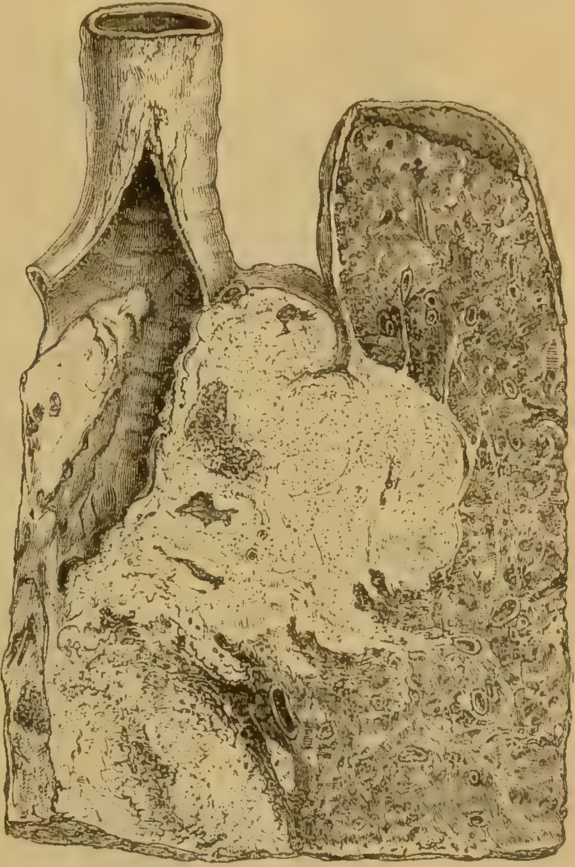

Fig. 116.—Lympho-sarcoma of the Mediastinum infiltrating the Lung.

The general characters of sarcomata.—It will now be necessary to refer to the characters of sarcomata in general.

The blood supply.—The vascularity of sarcomata varies greatly. In all the circulation is mainly capillary. In the small round-celled species the vessels may be so numerous as to cause distinct pulsation and a loud murmur or "hum." The slow-growing spindle-celled species are poor in vessels. In tumours, which grow rapidly, the walls of the blood-vessels are very thin, and rupture from the force of the circulation, converting the centre of a sarcoma into a blood cavity. Tumours thus transformed were formerly described as "malignant blood cysts."

Infiltrating properties. — In some instances sarcomata are furnished—at least, in their early stages—with a capsule, but the majority are unencapsuled, and extensively infiltrate the planes of connective tissue adjacent to the tumour. This infiltrating tendency comes out well when a sarcoma starts within the sheath of a muscle, for it will dissociate the fasciculi and extend throughout the muscle, transforming it into a hard indurated mass. Retinal sarcomata, after escaping from the eyeball, infiltrate the ocular muscles, and periosteal sarcomata infiltrate the adjacent muscles, and render it impossible to determine the real limits of the tumours, especially when seated in the limbs. Lympho-sarcomata, arising in the mediastinum, illustrate the disastrous effects of infiltration (Fig. 116).

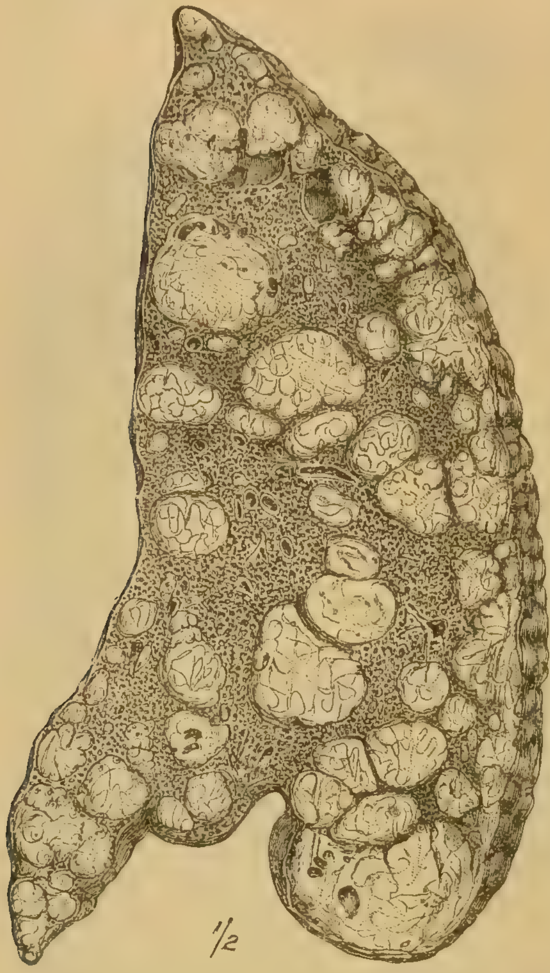

Fig. 117.—Section of Lung, with Nodules of Sarcoma secondary to spindle-celled Tumour of the Testis. (Museum, Royal College of Surgeons.)

Dissemination. — Sarcomata are liable to reproduce themselves in distant parts. The most usual organ in which to find secondary deposits is the lung (Fig. 117), unless the primary tumour is situated in the territory of the portal circulation, then they will be found in the liver. In very malignant sarcomata, especially the round-celled species, secondary deposits may form in every organ of the body. They are always identical in structure with the primary tumour.

The relation to veins.—It has long been recognised that when sarcomata disseminate, the situations of secondary nodules indicate that the distribution has been effected by means of veins. The venules issuing from a sarcoma will often be found plugged by small outrunners from the tumour, and occasionally, when a large sarcoma of the pelvic bones implicates the iliac veins, portions of the tumour will travel along the lumen of the vein, and even extend into the inferior vena cava (Fig. 118). This is not uncommon in connection with sarcoma of the kidney. Sometimes large pieces detached from these intravenous processes produce embolism of the pulmonary artery, and have been so large as to obstruct the right auriculo-ventricular orifice.

Secondary changes.—Sarcomata are very prone to degenerative changes; for instance, hæmorrhage is very apt to take place in those which grow quickly, producing spurious cysts. The tissues of the tumour are prone to liquefy, and myxomatous changes are very common. Calcification occurs in those which grow slowly, especially when connected with bone. When sarcomata grow rapidly and involve the skin, ulceration is very ready to occur and leads to profuse and oft-repeated hæmorrhages, which not only exhaust the patient, but in many cases induce death.

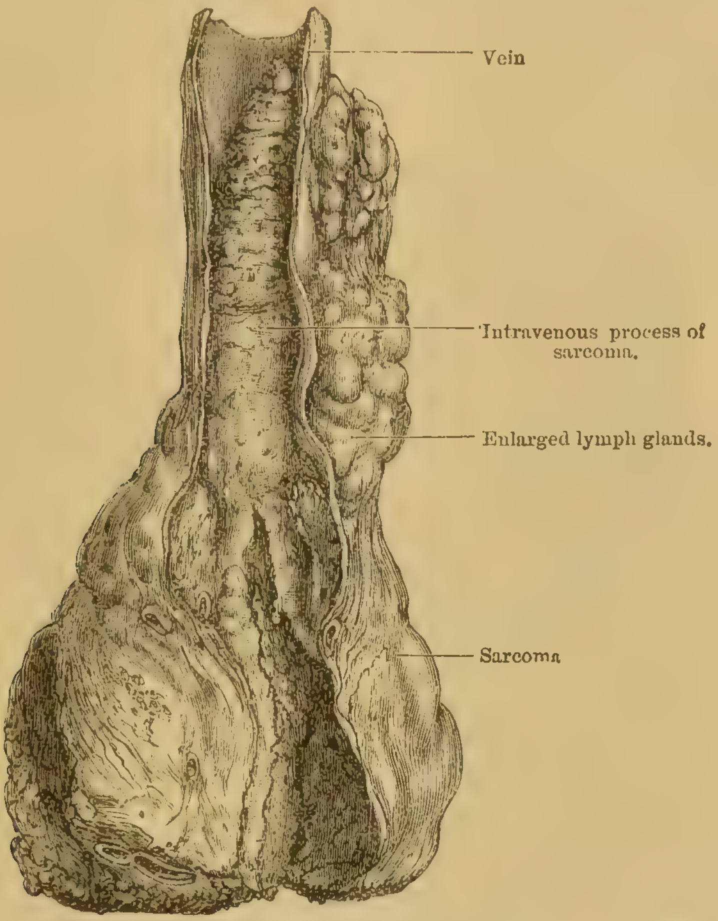

Fig. 118.—Periosteal Sarcoma of the Ilium invading the Inferior Vena Cava. (Museum, St. Bartholomew's Hospital.)

Occasionally considerable portions of a sarcoma will necrose; this is more apt to occur in very large tumours. In such cases a large spurious cyst forms in the sarcoma, and on cutting into it the fluid escapes with large, irregular pieces of the tumour, which are generally of a greyish-white colour. When necrosis occurs extensively in a large sarcoma it will sometimes check its course in a very marked manner.

The distribution of sarcomata.—As connective tissue occurs in every organ of the body, so sarcomata are ubiquitous. But they grow in some situations much more frequently than in others. Some species arise in any organ, others are restricted to certain definite structures. Sarcomata frequently arise in subcutaneous tissue and fascia, intermuscular septa, periosteum, marrow of bone, testis, ovary, and salivary glands; occasionally they occur in the brain, rarely in the spinal cord and nerves. They are very rare as primary tumours of muscles, liver, lung, spleen, alimentary canal or uterus. Sarcomata grow from the retina of children and uveal tract of adults, and in connection with congenital defects of the skin. The clinical characters of sarcomata are considered in the Articles devoted to special organs.

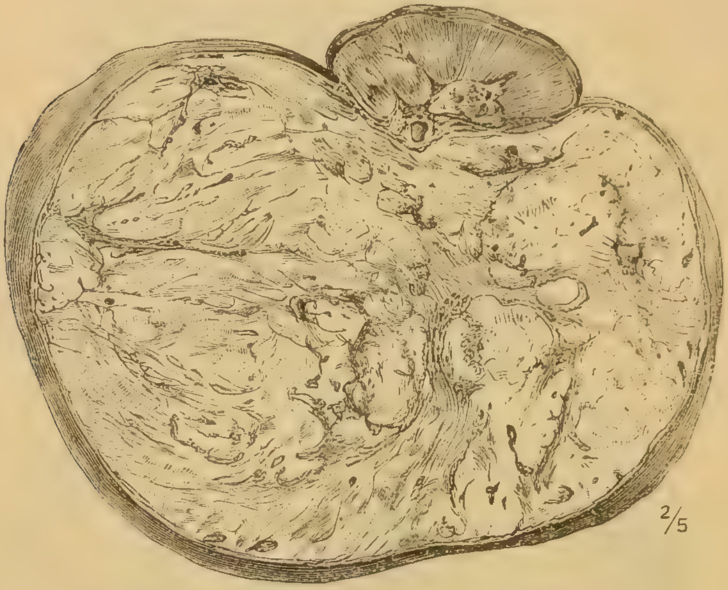

Fig. 119.—Adrenal Tumour, with the Kidney *in situ.* (Museum, Royal College of Surgeons.)

Adrenal tumours.—Tumours exhibiting the histological characters of the zona fasciculata arise in the adrenals and in accessory adrenals.

An adrenal may become transformed into a large tumour in the same way that the thyroid gland becomes a goitre, and sometimes the mass produced by this change may equal a melon in size (Fig. 119). In children both adrenals may be affected.

Accessory adrenals are occasionally found embedded in the cortex of the kidney. On superficial examination they resemble small fatty tumours, and have often been described as renal lipomata. When thin sections of these bodies are prepared for the microscope and deprived of fat, they exhibit the peculiar structure of the zona fasciculata of the adrenal.

These accessory adrenals are probably the source of certain renal tumours usually described as sarcomata, but resembling the adrenal in structure. The subject is of practical importance, inasmuch as renal tumours of this character are far less malignant than are other species of renal sarcomata.

Deciduoma.—This name is applied to an excessively malignant tumour characterised histologically by cells identical with the large decidual cells of the placenta. This tumour probably arises in fragments of placental tissue left in the uterus, as they have only been observed in women after recent delivery or abortion. The tumour grows rapidly, and is usually lobulated; on section the lobules resemble in colour and consistence the pulp of a pomegranate. Secondary deposits occur in the viscera and bones.

GROUP II. EPITHELIAL TUMOURS.

In the group of tumours now to be considered, epithelium is not only present, but is the essential and distinguishing feature. Epithelium is so disposed in the bodies of complex animals as to serve many functions. In some situations it acts as a protective—*e.g.* the epidermis, where it becomes modified into hair, nail, horn, or into the hardest of all animal tissues—enamel. In other parts, processes of epithelial cells dip into the underlying connective tissue to form secreting glands; some are very simple—*e.g.* the tubular glands of the intestine; others are very complex—*e.g.* the liver, mamma, and kidney. Whether a gland is simple or complex, the principle of construction is identical—namely, narrow channels lined with epithelium, resting upon a connective-tissue base, in which blood-vessels, lymphatics and nerves ramify.

Each epithelial recess of a gland is known as the acinus, and each acinus is in communication with a free surface, either directly by its own duct, as in the case of sebaceous and mucous glands, or indirectly by means of a number of main ducts, as in the case of the mamma; or by a common duct, as in the pancreas. To this rule there are three notable exceptions: the thyroid gland, the pituitary body, and the ovary.

Classification.—The differences in the disposition of epithelium enable epithelial tumours to be arranged in four genera: 1, Papilloma; 2, epithelioma; 3, adenoma; 4, carcinoma.

1. Papilloma (Warts).

A papilloma consists of an axis of fibrous tissue containing blood-vessels, surmounted by epithelium, projecting from an epithelial surface: this surface may be uniform, or covered with secondary processes, like a mulberry, or with long villous tufts.

There are four species of papillomata: (1) Warts; (2) villous papillomata; (3) intracystic warts; (4) psammomata.

1. **Warts.**—These are very common on the skin, but they also arise from mucous membrane covered with stratified epithelium. They occur singly or in great numbers, and are rarely painful, unless irritated. Crops of warts are common on the hands of children and on the scalp. They are often numerous on the glans penis, the anus, and labia when these parts are irritated by purulent discharges, especially that of gonorrhœa. Skin warts are overgrown papillæ, and in section the epithelium will be found to pass from papilla to papilla in an unbroken line without invading the fibrous framework.

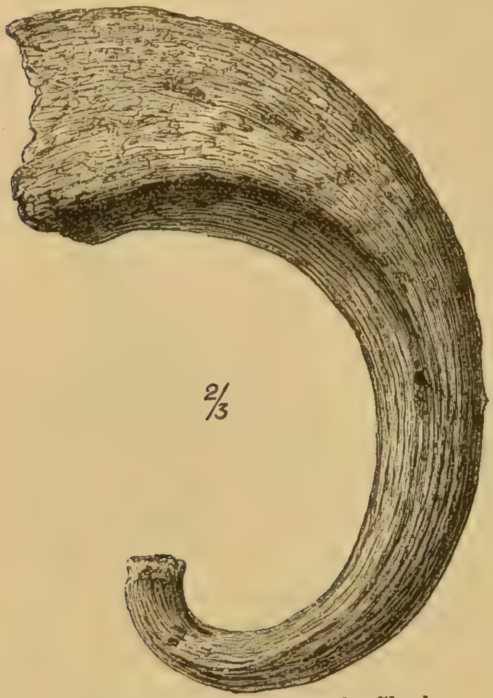

Fig. 120.—Wart-Horn from the Cheek. (Museum, Middlesex Hospital.)

A curious feature of multiple warts is that they sometimes appear in great number on the hands or scalp, and after persisting for some weeks, or even months, suddenly disappear as if by magic.

A solitary wart may grow from any skin-covered surface, and persist. Such a wart may attain the dimensions of a walnut. Large solitary warts often contain black pigment, and some become the source of melanomata. A wart occasionally grows very rapidly, and may reach the size of a bantam's egg. Its surface ulcerates, and gives rise to a foul discharge, with a very offensive odour. The cells covering cutaneous warts sometimes become transformed into horns—wart-horns (Fig. 120). (*See* Art. XXVIII., pages 708 and 710.)

Warts on mucous membranes are not so common as on the skin, but they are apt to produce serious consequences, especially when they arise in the larynx (Fig. 121). Laryngeal warts in children are often multiple; in adults they are less frequent, almost invariably solitary, and are frequently imitated by laryngeal epithelioma.

2. **Villous papillomata.**—These grow from the mucous membrane of the bladder, and occasionally from the pelvis of the kidney. They consist of long-branched feathery tufts, resembling the villi of the chorion. The bladder wart consists of a connective-tissue axis traversed by delicate vessels surmounted by columnar or

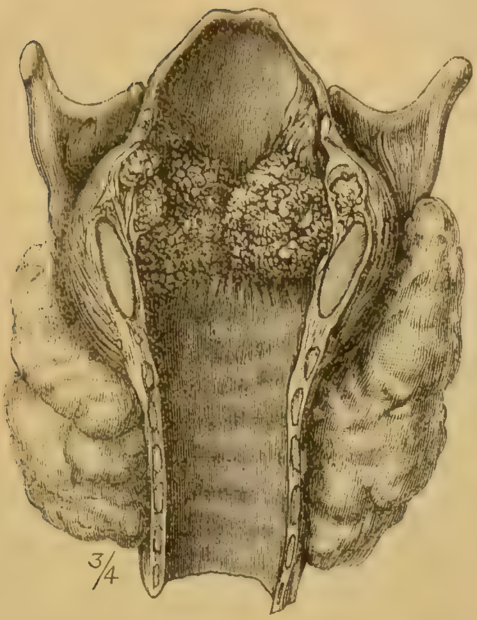

Fig. 121.—Laryngeal Warts.

cubical epithelium. Villous papillomata sometimes possess broad bases, but often the points of attachment are so narrow that the tumour may be described as pedunculated. These tumours may be single or multiple. Instances are known in which villous papillomata of the renal pelvis have been associated with similar smaller tumours of the vesical mucous membrane near the orifices of the ureters, probably due to epithelial infection. (*See* Art. LI., on INJURIES AND DISEASES OF THE URINARY ORGANS, Vol. II.)

A very interesting variety of villous papilloma arises from the choroid plexuses of the cerebral ventricles. When large, and situated in the choroid plexus of the fourth ventricle, they have produced pressure symptoms and death. These tumours rarely attain such size as to be fatal without undergoing calcification.

3. **Intracystic papillomata.**—The interior of all true cysts is lined with epithelium. Warts springing from the inner walls of cysts are not common, save in two situations — (1) cysts of the mammary gland, (2) cysts of the Paroöphoron and Gartner's duct. They have been observed in cysts arising in accessory thyroid glands.

4. **Psammomata.**—These are tumours composed of globular bodies, consisting of epithelial cells arranged in layers, usually calcified, and embedded in connective tissue (Fig. 122). They are confined exclusively to the pia mater of the brain and spinal cord

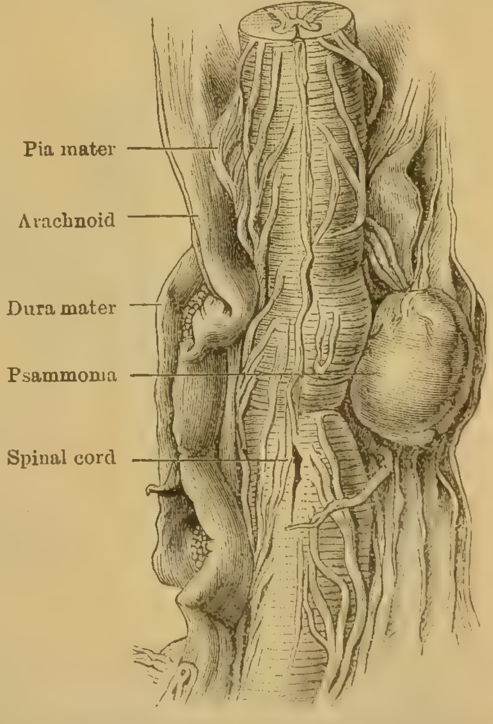

Fig. 122.—Psammoma of the Spinal Membranes. (Museum, Middlesex Hospital.)

(Fig. 123). In the case of the brain they grow most frequently from the choroid plexuses of the ventricles. The amount of calcareous matter in psammomata varies greatly. Sometimes the tumours are of stony hardness. In the brain they rarely exceed the dimensions of a shelled walnut. Favourite spots for these tumours are the tufts of villi which protrude from the lateral recesses of the fourth ventricle. The close relations of such tumours to the medulla, the trigeminal, facial, and auditory nerves lead to disastrous consequences.

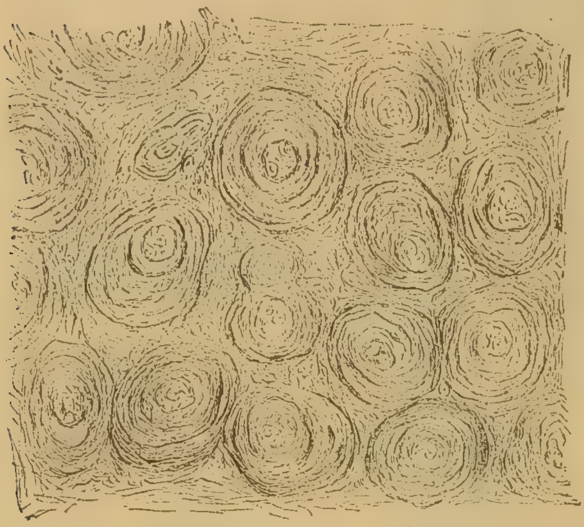
Fig. 123.—Microscopical Characters of a Psammoma.

2. Epithelioma.

An epithelioma differs from a wart in the fact that the epithelium is not limited by the basement membrane, but passes beyond it into the underlying connective tissue. This invasion is attended by peculiar cell formations known as nests. The disease often recurs after removal, and is exceptionally liable to infect adjacent lymph glands. This genus consists of a single species—epithelioma.

Site and mode of origin.—An epithelioma may arise in any part of the body where stratified epithelium exists, and is particularly prone to occur in situations where there is a transition from one kind of epithelium to another, and especially at spots where skin and mucous membrane come into relation with each other—*e.g.* the anus and lips. Injured parts are sometimes attacked by this disease—*e.g.* the edges of cicatrices (Fig. 124) and ulcers.

An epithelioma may make its appearance as a wart, as a fissure, or as a nodule on the surface of skin or mucous membrane. Perhaps the most frequent form is that in which the epithelioma appears as an ulcer with raised, rampart-like edges.

When the disease starts in a fissure, and ulceration keeps pace with the infiltration, then, instead of raised edges, the ulcer has margins as sharply defined as those of a rodent ulcer, or even undermined.

There is a third variety, in which processes project from the skin like warts, and their free surfaces are sometimes quite horny.

Structure.—Although these three clinical varieties of epithelioma look so different, they are identical in structure. When sections are cut in such a way as to include not only the edge of the ulcer but the adjoining tissue also, the surface epithelium will be found to dip into the underlying tissue in the form of long columns (Fig. 125). The parts around these cell-columns are infiltrated with adventitious cells; among and beyond these columns as well as within them, curious concentric bodies known as *nests* may be found. The cells composing these nests are arranged around two or more altered cells like the layers of an onion. The cell-columns are not enclosed by membrane, and some of the larger columns tend to branch and even fuse with adjacent columns, forming a network in the deeper tissues. It matters not whether the epithelioma grows on the lip, tongue, larynx, or edge of a scar, this peculiar disposition of cell-columns is observed, accompanied by cell-nests. The size of the columns and number of the nests vary in different cases, but the plan of invasion is the same in all.

It is important to bear in mind that the three clinical varieties of epithelioma occur in most of the situations which are liable to this disease; in addition to the lip, it has come under my notice in the tongue, anus, buccal aspect of the cheek, glans penis, vaginal surface of the uterine cervix, and at the edge of old scars. The non-recognition of these three manifestations of epithelioma has produced much confusion in surgical writings. For instance, the warty form has in many instances been described as epithelioma supervening on warts.

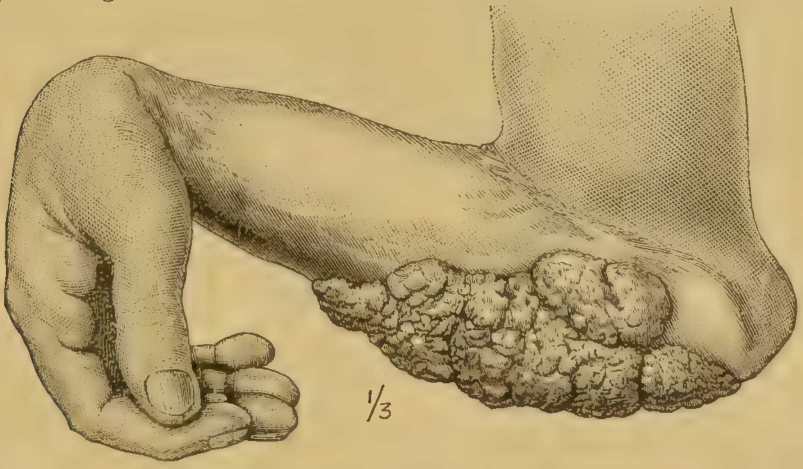

Fig. 124.—Epithelioma arising in the Cicatrix of a Burn. (Museum, Middlesex Hospital.)

Course.—The primary ulcers when left to themselves may extend and involve extensive tracts of tissue, or fungate and form huge granulating cauliflower-like growths. In either case the superficial parts are continually being cast off in a foul, fœtid discharge containing sloughs of tissue, cellular detritus, and blood. Any vascular tissue such as skin, muscle, and mucous membrane, is quickly infiltrated and destroyed; even bone is rapidly eroded and removed piecemeal. Cartilage resists invasion. This is seen in a striking way in those instances where the pinna is the seat of epithelioma; the skin and soft tissues quickly disappear, whilst the cartilaginous framework stands out prominently amidst the surrounding ruin.

Epithelioma in whatever situation it occurs destroys life rapidly. The quickness with which it ulcerates and overcomes all resistance enables it to open up large blood-vessels should any lie in the way. Hence death from hæmorrhage is frequent; when the tumour is near the air-passages septic material is inspired and initiates pneumonia.

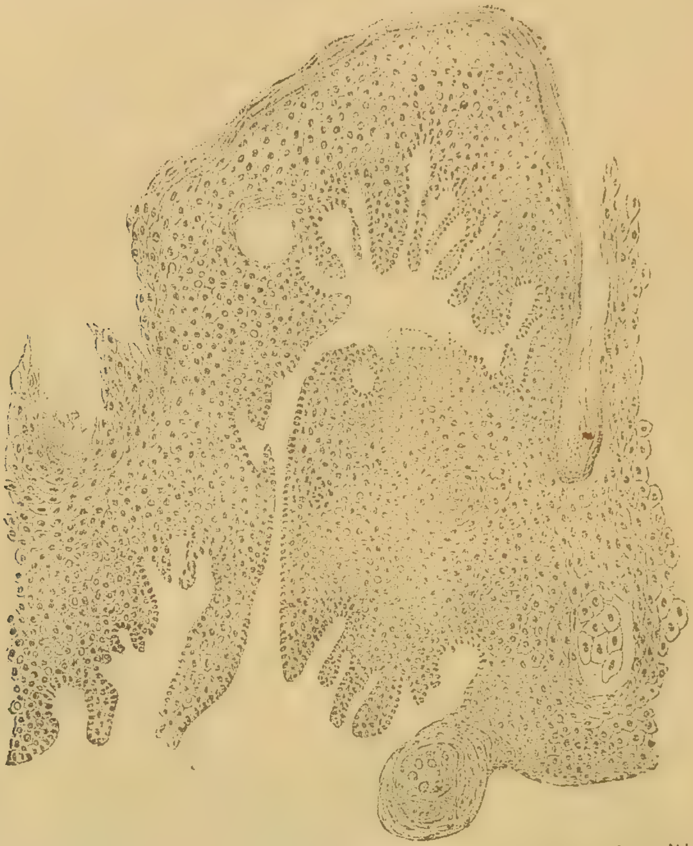

Fig. 125.—Disposition of Epithelium in Epithelioma. The stroma is omitted. (After Delépine.)

Particular modes of death occur according to the situation of the epithelioma, and it will be more convenient to consider them when dealing with the disease in the various situations in which it occurs than to attempt a summary of them here.

The three varieties exhibit different degrees of malignancy. The burrowing variety rapidly kills, whilst warty epithelioma runs much the slowest course; each manifests its malignancy in the same manner by infecting adjacent lymph glands, recurring after removal, and occasionally by dissemination.

Lymph gland infection.—The rapidity with which lymph glands are infected is the most remarkable and dangerous feature of epithelioma; the large size the affected glands attain to in many cases is often astonishing, and their enlargement stands in no relation to the size of the initial lesion, for an epithelioma 2 cm. square or less, will lead to the formation of a gland-tumour as big as a cocoa-nut. Such conditions are most frequently met with when the tongue, lip, and scrotum are the seats of this disease.

The gland complication in epithelioma is always a serious element of danger. When the cervical glands are enlarged they interfere with the trachea and œsophagus; they also become firmly adherent to the sheath of big vessels, and as the glands break down the ulceration not infrequently opens up the jugular vein or carotid artery, and, in the inguinal region, the femoral vessels. A peculiarity of glands infiltrated by epithelioma is the tendency they exhibit to break down in the centre and form spurious cysts. This should be remembered, for fluctuating glands associated with epithelioma does not necessarily signify suppuration. When the skin becomes implicated portions of the infected glands slough, and leave large horrible gaps, from which a foul fœtid discharge proceeds, whilst the edges of the chasm produced by the sloughing continue to extend and involve the neighbouring tissues.

Dissemination.—It has already been mentioned that secondary deposits are exceptional in epithelioma; it cannot be said that they are rare, but dissemination certainly happens far less frequently, and never so extensively as in cancer. It is also noteworthy that epithelioma is in some situations more liable to disseminate than in others. For example, secondary deposits are rarely met with when this disease attacks the larynx, and the mucous membrane in relation with the mandible or maxilla, and the œsophagus. The explanation sometimes offered of this peculiarity is that epithelioma in these situations usually runs a rapid course, and often destroys life so quickly that the period is too short to allow of the formation of secondary nodules. This is inadmissible, as in epithelioma of the scrotum dissemination is almost as exceptional as when the larynx is attacked.

Treatment.—The principles on which surgeons rely for the treatment of epithelioma are:—(1) Early and free removal of the primary disease whenever it is in an accessible situation; (2) when adjacent lymph glands are enlarged they should be dissected out coincidentally with the removal of the primary lesion; (3) when there is recurrence, and the condition of the part admits, and the general health of the patient is such as will permit an operation to be performed with safety, the tumour should be excised.

The early excision of epithelioma is practised for two very important reasons:—

The earlier the diseased area is removed the greater the prospect of eradicating the disease before it affects the adjacent lymph glands.

The extirpation of an epithelioma in its early stages is ofttimes a very trivial proceeding; when allowed to extend, its complete removal will demand a very extensive, difficult, and frequently a dangerous operation, and often is an impossible task.

It is difficult to formulate rules for the operative treatment of epithelioma and to decide what is, and what is not, justifiable surgery. Every surgeon must be guided by individual experience. It is exceedingly difficult to express collectively the effects of operation in eradicating this disease. The facts broadly stated stand thus :—

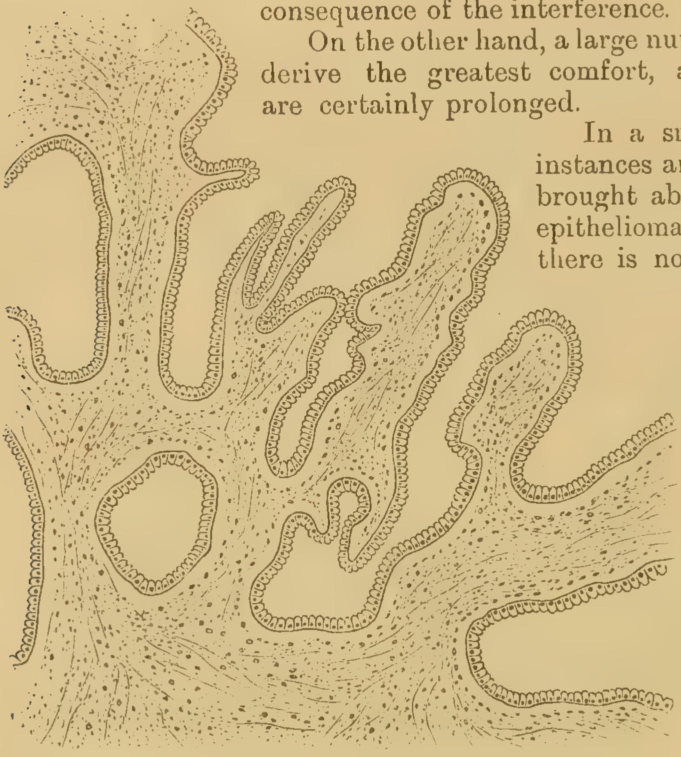

Fig. 126.—Microscopical Characters of an Ovarian Adenoma.

In a small proportion of cases the operation is of doubtful utility, and in a few instances life is sacrificed in consequence of the interference.

On the other hand, a large number of patients derive the greatest comfort, and their lives are certainly prolonged.

In a small number of instances an actual cure is brought about. When an epithelioma is removed and there is no recurrence for five years, the individual may be regarded as cured.

It will be useful to reiterate here that of the three clinical varieties of epithelioma the burrowing form is not only the most malignant, but gives the worst results after operation. The warty variety is not only the least malignant, but affords the best results when excised.

It may be regarded as an axiom that in cases where operations are performed for epithelioma, and, as far as could be judged, the incisions were carried wide of the diseased tissues, a quick recurrence of the disease, either in or near the cicatrix, or the subsequent enlargement of the lymph glands, may be taken as an indication of a high degree of malignancy, and, as a rule, the uselessness of further operative interference.

The tumours often classed as columnar epithelioma, which are so common in the intestine, especially the colon, are carcinomata, and arise in, and exhibit, the structure of intestinal glands.

3. Adenoma.

An adenoma may be defined as a tumour constructed upon the type of, and growing in connection with, a secreting gland, but differing from it in being impotent to produce the secretion peculiar to the gland it mimics (Fig. 126). Occasionally adenomata produce a perverted secretion, especially the rectal, uterine, and prostatic species.

As regards *seat,* adenomata occur as encapsuled tumours in such glands as the mamma, parotid, thyroid, and liver. In the mucous membrane of the rectum, intestine, and uterus they are pedunculated. A single adenoma may be present, but not infrequently two or more exist in the same gland. In the case of the intestine, a score or more may be present in the same individual. In *size* they vary greatly. Some are no larger than peas, whereas in certain situations—*e.g.* the mamma—an adenoma will occasionally attain to the dimensions of a man's head, and in the case of the ovary, an adenoma weighing forty pounds is no rarity; in such the acini are usually distended with fluid.

The *effect* of adenomata depends mainly upon the situations in which they grow. The following statements are true for all:—When completely removed there is no fear of recurrence; they do not infect neighbouring lymph glands, nor give rise to secondary deposits. When an adenoma causes death, it is in consequence of mechanical complications, depending on the situation and size of the tumour.

Although the distinguishing *structural peculiarity* of an adenoma is the presence of epithelium disposed as in a secreting gland, the connective tissue (stroma) entering into its composition must also be taken into account. In many adenomata the epithelial element is the most conspicuous; in others the connective tissue is out of all proportion to the epithelium, and occasionally preponderates to such a degree that the tumour from some writers receives the misleading name of "adeno-sarcoma." When the epithelium-lined spaces are distended with fluid, the tumour is spoken of as a "cystic adenoma" and sometimes as an "adenocele."

The *species* of adenomata are determined by the glands in which they arise. The chief species are:—Mammary, sebaceous, thyroid, pituitary, prostatic, parotid, hepatic, renal, ovarian, testicular, gastric, intestinal, Fallopian, uterine. The peculiar pathological and clinical features of each species of adenoma will be described in the Article devoted to the particular organ in which it arises.

Very little is known as to the *cause* of adenomata, but there is good reason to believe that many of them arise in detached and isolated portions of secreting glands, to which the name "rests" has been applied. Rests have been detected in connection with the mamma, pancreas, liver, parotid, and thyroid glands; and there can be no doubt that, with the increased attention now being devoted to this matter, many more examples will come to light. Perhaps the most curious facts in relation with "rests" is that they may remain latent for years and then, without any obvious cause, suddenly take on active growth and become formidable tumours.

Although all secreting glands are liable to adenomata, these tumours are commoner in some glands than others. For instance, adenomata are very common in the mamma, the ovary, and the thyroid gland. They are fairly frequent in the intestine and prostate, but rare in the liver and the pituitary body.

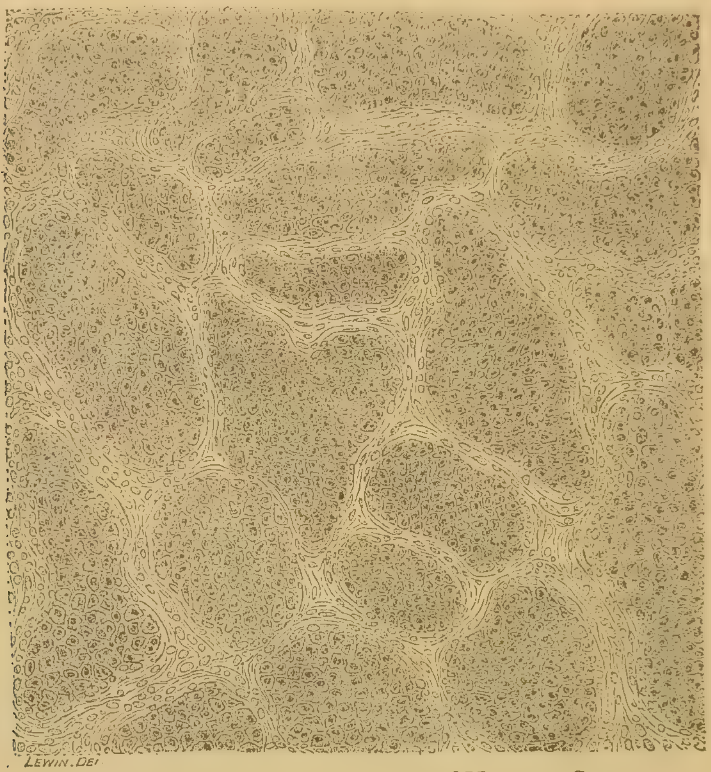

Fig. 127.—Microscopical Characters of Mammary Cancer.

These tumours are met with in youth and adult life, and the liability decreases with each decade after the thirtieth year. In some situations—such as the intestine, parotid, and prostate gland—the adenomata never attain large size, whereas in the ovary they form the biggest tumours from which mankind suffers.

In the mamma and parotid gland adenomata never destroy life, whereas an intestinal adenoma no larger than a walnut sometimes leads to intussusception, and one the size of a cherry in the prostate will so obstruct the outflow of urine from the bladder as to lead to grave and often fatal changes in the kidneys.

The huge adenomata of the ovary cause death from mechanical causes alone, and this is true of pituitary adenomata. In one case a tumour weighing fifty pounds may be accommodated in the belly with but little inconvenience; in the other a tumour of the size of a cherry leads to fatal pressure on the base of the brain.

4. Carcinoma (Cancer).

Carcinomata are tumours that always grow from pre-existing gland tissue, and mimic the parent gland; but they differ from adenomata in the fact that the structural mimicry is incomplete. The epithelium, instead of exhibiting the regular disposition so constant in adenomata, is, in the cancers, collected in the acini and ducts in irregular clusters, or fills them so completely as to give rise to the appearance of sections of epithelial columns when seen under the microscope (Fig. 127).

As in the case of adenomata, the species of carcinomata depend upon the relation of the epithelium to the stroma of the tumour.

Situation.—Carcinomata arise in every secreting gland that gives rise to an adenoma; but they are very common in some glands and exceedingly rare in others; indeed, those glands which are the most frequently affected with adenomata are the most liable to carcinoma, with the exception of the ovary. The chief species of carcinoma are: Mammary, sebaceous, thyroid, prostatic, parotid, pancreatic, hepatic, renal, gastric, intestinal, and uterine.

General characters.—Cancers are not encapsuled, but infiltrate surrounding tissues and pass beyond the glands in which they originate; they are very prone to involve the superficial tissues, ulcerate, and quickly infect the lymph glands in the near neighbourhood. A marked feature of carcinomata is their great tendency to undergo degenerate changes and necrosis. The rapidity with which the lymph glands are infected is due to the abundance of lymphatics in most species of cancer.

Dissemination.—Cancers are exceptionally prone to become disseminated; the secondary growths may make their appearance in any organ or tissue, and not infrequently in the bones. The cancer grafts which give rise to these secondary nodules are transported by lymph and blood-vessels, and when these minute emboli are lodged in suitable situations they multiply, giving rise to a growth which, in its histological features, exactly resembles the parent tumour. So faithful is this reproduction that the nature of the primary tumour can often be correctly inferred from a microscopic examination of a secondary nodule.

Indeed, few things in pathological histology are more surprising than on examining a cancerous nodule in the shaft of a long bone —secondary to rectal cancer—to find it present the tall columnar epithelium so characteristic of intestinal glands.

The amount of dissemination varies greatly. In some cases secondary deposits will be found only in the liver, whilst in another and apparently identical case, in so far as the structure of the tumour is concerned, secondary knots occur in almost every organ of the body, including the skeleton.

Secondary deposits of cancers are not always so small as merely to merit the name of knots, but form occasionally tumours of some magnitude, and they may even excel in size the primary tumour.

The characters, clinical and pathological, of particular species of carcinoma are considered in connection with the organs in which they arise.

Varieties.—It is necessary to mention here that the method of dividing cancer into three varieties — scirrhous, encephaloid (medullary), and colloid—is not only misleading, but has no structural basis. It is also important to bear in mind that many misconceptions have arisen from the circumstance that pathologists have been in the habit of interpreting the structure of cancers from plane sections, without in the least taking into consideration the relation of a given section to the entire tumour. Hence a scirrhous cancer was said to be composed of an alveolar meshwork of fibrous tissue, the alveoli enclosing epithelial cells. If, instead of drawing conclusions from one or two sections selected haphazard, a number of consecutive sections be taken, and a composite figure framed from them, it will at once become clear that the alveoli are sections of glandular ducts and acini filled with cells.

This fact is admirably illustrated in the case of rectal cancer: sections from this kind of tumour may take the form of closely-packed cylinders, or of bays lined with epithelial cells encroaching on a bed of delicate connective-tissue. When the sections are examined collectively, it will be found that the alveoli are sections of greatly-enlarged intestinal glands filled with cells and cut in various planes. What is true of the rectum holds good for cancer of other secreting glands.

Cause.—Very little positive knowledge is available in regard to the cause of cancer. To-day the prevailing opinion among those best qualified to judge is that the disease depends on a micro-parasite, which in all probability flourishes in glandular epithelium.

The micro-parasite does not belong to any of the known species of bacteria or micrococci, but recent researches tend to show that it probably belongs to those curious micro-organisms of which the common amœba is the type. At present the evidence is barely more than hypothetical.

Although the active investigations carried on in bacteriological laboratories during the past few years have done but little to remove the cause of cancer from the domain of hypothesis, they have nevertheless strengthened the view that in its earliest stages cancer is a local disease, and that prompt and complete removal whenever possible is the most satisfactory method of relief at present available.

The wisest observers differ in opinion as to the heredity of cancer. It certainly is not contagious.

Every secreting gland is liable to become the seat of cancer; nevertheless some glands are more prone to it than others. For instance, the female mamma and the glands of the cervical canal of the uterus are so liable to cancer that many thousands of women die annually from this disease in these two situations in all civilised countries. Of the liability of savages to cancer nothing is known. Cancer of the intestinal and gastric glands is common in both sexes, but cancer of the prostate, thyroid, pancreas, and the glands of the body of the uterus are exceptional.

Cancer in the restricted sense in which the term is here employed is unknown before puberty: it is very rare indeed, before the age of twenty-five, but after that period it becomes common, and increases with each decade of life. These facts are the reverse of those observed in regard to adenomata.

Termination.—The modes by which cancer kills varies with the situation of the tumour: thus mammary cancer causes death by implicating the pleura, or by secondary deposits in the lungs, brain, etc. Uterine cancer will induce death from bleeding, or from nephritis or uræmia due to implication of the ureters; or from peritonitis. Cancer of the intestine is usually fatal from intestinal obstruction or peritonitis, gastric cancer from starvation and thyroid cancer from suffocation.

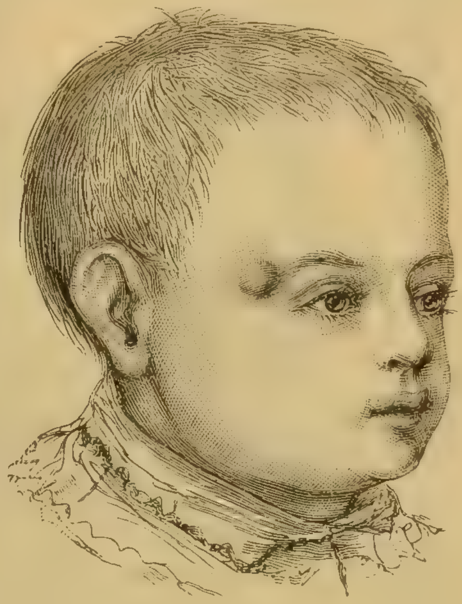
Fig. 128.—Dermoid at the Outer Angle of the Orbit.

GROUP III. DERMOIDS.

Dermoids are tumours furnished with skin or mucous membrane occurring in situations where these structures are not found under normal conditions. They only possess tissues and structures which naturally belong to skin or mucous membrane. Dermoids may be arranged in four genera: 1, Sequestration dermoids; 2, tubulo-dermoids; 3, ovarian dermoids; 4, dermoid patches.

1. Sequestration dermoids.—The species of this genus arise in detached or sequestrated portions of surface epithelium, chiefly in places where, during embryonic life, coalescence takes place between skin-covered surfaces.

The chief lines of coalescence in the trunk are situated in the mid-dorsal and mid-ventral regions, and extend from the external occipital protuberance backwards along the spine through the perineum (and scrotum in the male) forwards to the episternal notch. Dermoids occur in any part of this extensive line.

In the face dermoids occur in connection with all the facial fissures, including the mesopalatine. These temporary fissures are liable to three defects directly associated with dermoids. They may fail to close; then such faults as hare-lip, cleft-palate, macrostoma, cleft-sternum, and spina bifida are the consequences, according to the seat of failure. The clefts may close imperfectly and leave complete or incomplete fistulæ, which receive such names as post-anal or coccygeal dimples, sternal dimples, mandibular fistulæ, and the like. A fissure may to superficial appearances close perfectly, but a tract of epithelium lies buried between the opposed and coalescing surfaces; this buried skin or epithelium may grow and form a dermoid.

Sequestration dermoids usually assume a cystic form; that is, the skin forms the wall of a central cavity, which gradually enlarges as the individual grows, and becomes occupied and distended by the shed epithelium, hair, and products of the glands occupying the skin lining the cyst. Hair, sebaceous and sweat glands are usually present. Teeth are excessively rare in this genus of dermoids. Exceptionally, dermoids take the form of solid tumours, the skin clothing the exterior; still more rarely they are pedunculated.

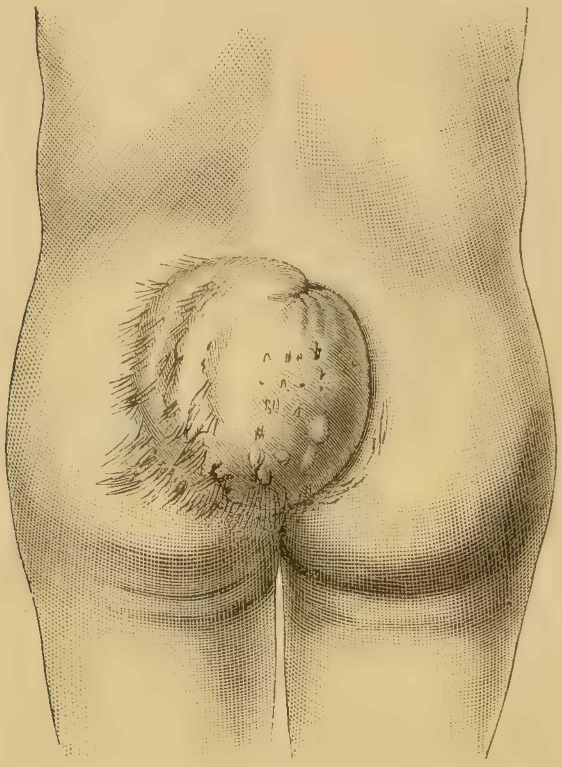

Fig. 129.—Dermoid simulating Spina Bifida in an Adult.

Dermoids arising in fissures are very common near the angles of the orbits, especially the outer angle (Fig. 128), and are not uncommon in the pinna or auricle: they are fairly common over the lumbo-sacral vertebræ, where they are apt to be mistaken for spina bifida (Fig. 129). Some have been reported in relation with the anterior and posterior aspect of the sternum (Fig. 130). They also occur in relation with the middle line of the scrotum, and are often reported as testicular dermoids.

An important species of sequestration dermoid arises in connection with the scalp. In the embryo the scalp and the dura mater are in contact; later the cranial bones interpose and separate them. Pieces of the scalp may be retained in connection with the dura mater, and, acting as tumour-germs, may grow into dermoids.

These tumours may lie in depressions in the bones and project the scalp, and be mistaken for sebaceous tumours, or even meningoceles if they grow, as they are apt to do, over a fontanelle and receive pulsation from the brain. Exceptionally, they project on the cerebral aspect of the dura mater, and lead to fatal compression of the brain. Dermoids of this kind have several times been observed in relation with the tentorium cerebelli.

Implantation cysts. — It is a recognised fact that dermoids do not occur in the limbs, but certain small tumours are occasionally observed in the hands and fingers, which have been described as dermal cysts, sebaceous cysts, or digital dermoids.

In structure these tumours present a central cavity containing epithelial *débris* and a lining of skin; indeed, when examined microscopically, they appear "as if a piece of skin had been inverted into the subcutaneous tissue" (Shattock). These tumours are known as "implantation cysts," as there is every reason to believe that they are produced by small pieces of skin accidentally transplanted by injury, especially pricks by sharp instruments, such as awls, needles, forks, etc. They also occur in cicatrices. Implantation cysts are usually of the size of cherry-stones, but they may reach the size of bantam's eggs.

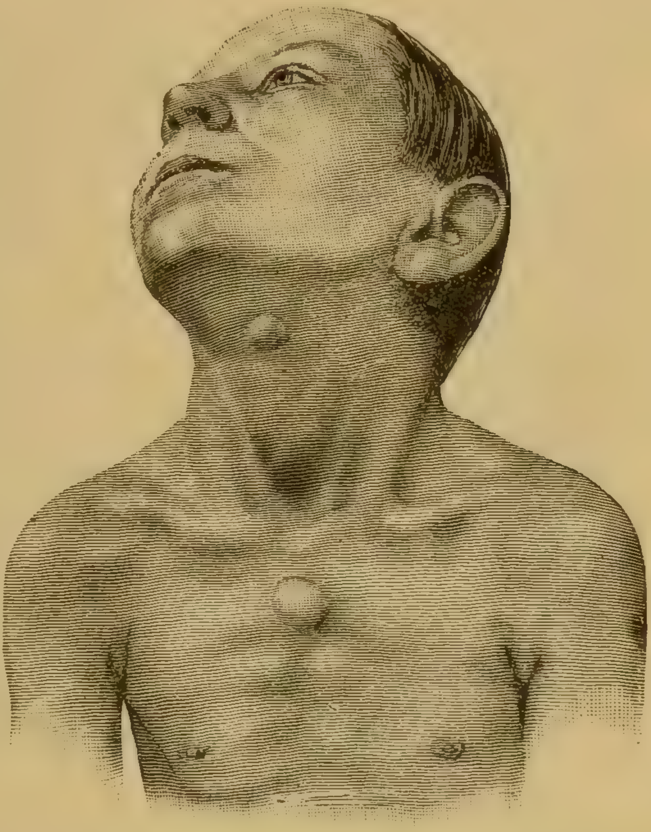

Fig. 130.—Dermoid situated over the Junction of the Manubrium and Gladiolus; there is a dermoid over the left cornu of the hyoid bone. (After Bramann.)

These tumours are not confined to the limbs; they have been observed on the trunk, scalp, and face. Very many examples have been reported in the cornea and iris, the results of punctured wounds, injuries and operations on the eyeball. Detached conjunctival epithelium and eyelashes carried in and transplanted on the vascular iris may grow into tumours.

2. **Tubulo-dermoids.**—There exist in the human embryo certain canals and passages, many of which normally disappear before birth. Three of these obsolete canals—the thyro-glossal duct, the branchial clefts, and the post-anal gut—are occasionally the source of dermoids.

Lingual dermoids, especially those situated between the genio-hyo-glossi muscles and attached to the hyoid bone, doubtless arise in persistent portions of the buccal extremity of the thyro-glossal duct. Dermoids situated deeply in the substance of the tongue have been frequently mistaken for sebaceous cysts. They sometimes attain very large proportions, and bulge from the mouth. Occasionally, dermoids arising in the lingual segment of the thyro-glossal duct are structurally identical with the thyroid gland; they are known as thyroid-dermoids.

Rectal dermoids.—It must be borne in mind that dermoids are occasionally found hanging by pedicles from the mucous membrane of the rectum; others, sometimes of large size, grow between the rectum and the anterior face of the sacrum; these are known as post-rectal dermoids. There is a third species, which occurs as a large massive tumour projecting from the sacro-coccygeal region, and in structure closely resembling thyroid gland tissue.

There are good reasons for the belief that post-rectal dermoids, and those congenital sacro-coccygeal tumours situated anterior to the coccyx and sacrum, arise in the post-anal gut of the embryo.

Branchial dermoids.—These arise in persistent portions of the branchial clefts. These tumours are always congenital and are usually situated beneath the deep cervical fascia. Like congenital (lateral) cervical fistula, they always project in front of the anterior border of the sterno-mastoid muscle. Those arising in connection with the deep parts of the second cleft may bulge in the mouth between the genio-hyoid and genio-hyo-glossi muscles. They must not be confounded with dermoids arising in persistent remnants of the glossal portion of the thyro-glossal duct.

Tubulo-dermoids, apart from their mode of origin, differ from the sequestration genus in the following points:—(*a*) They often attain very large proportions; (*b*) exhibit a more complex structure; (*c*) and not infrequently contain teeth.

3. **Ovarian dermoids.**—These tumours differ from other genera of dermoids on account of their mode of origin and the greater variety of dermal structures they contain.

It is from the epithelium lining the ovarian follicles that the mucous membrane, skin and dermal structures are derived, which constitute such an extraordinary feature in the majority of ovarian dermoids.

Ovarian dermoids not only differ from the sequestration and tubulo-dermoids in their complexity, but also in the greater development of the dermal structures they contain. Thus, in ovarian dermoids hair is longer and more abundant; and has been known to reach a length of five feet. Skin glands of all varieties are large, abundant, and often very active—even mammary glands occur. (Fig. 131). Teeth to the number of four hundred have been counted; horn, nail, and epithelial pearls are not uncommon.

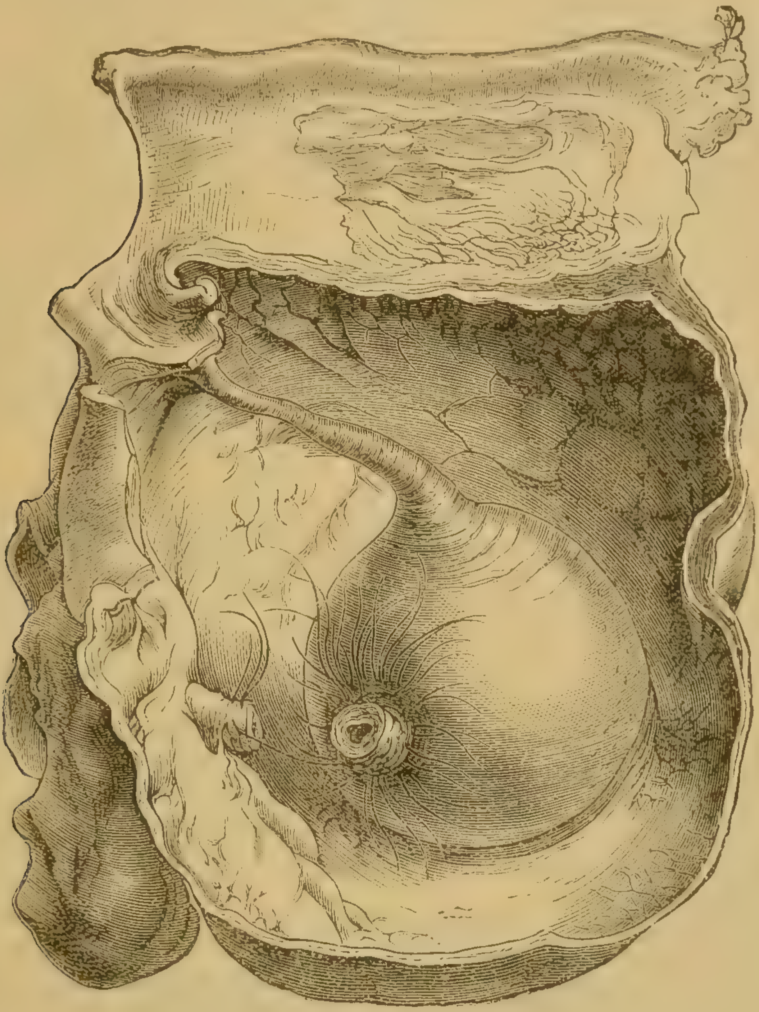

Fig. 131.—Ovarian Dermoid containing a Mamma.

It is a fact to bear in mind that all genera of dermoids except the ovarian are congenital. There is no reliable evidence of the existence of an ovarian dermoid in a child under a year.

The characters of the dermal elements are not influenced by the age at which the tumour occurs except in the matter of hair. Although the colour of the hair is independent of that on the exterior of the individual, it loses its colour and becomes shed in advanced age. Teeth develop at all ages; fully-developed ovarian teeth will be found in children four years old, and developing teeth in women at forty.

It is impossible to enter into a full consideration of the varieties exhibited in the contents of these extraordinary tumours, but some further details and illustrations will be given in the Article devoted to the ovary (Art. LIII., on INJURIES AND DISEASES OF THE FEMALE GENITAL ORGANS, Vol. II.). Neither is it necessary to discuss seriously the guesses at the nature of ovarian dermoids conveyed in such expressions as parthenogenesis and hypererchesis, or such phrases as excess of formative energy, inclusion of surface epiblast, etc.

Dermoids are occasionally found on the omentum, mesentery, peritoneum, and under surface of the liver; these are due to transplantation and detachment, and to epithelial infection from ovarian dermoids.

4. **Dermoid patches.**—Under this heading is included the pigmented hairy patches known as "moles" and especially those which are occasionally found on the conjunctiva. Hairy moles, whether situated on skin or conjunctiva, possess sebaceous and often sweat glands. They are very vascular. The most striking histological feature of a mole on the skin is that the tissue underlying it is often similar to that of an alveolar sarcoma. (*See* page 468.)

GROUP IV. CYSTS.

Cysts result from the abnormal dilatation of pre-existing tubules or cavities. In the simplest forms they consist of a wall usually composed of fibrous tissue, but not infrequently mixed with plain muscle-fibre. The cyst contents may be mucus, bile, saliva, etc., according to the nature of the organ with which the cyst is associated. Cysts may be arranged in four genera:—1, Retention cysts; 2, tubulo-cysts; 3, hydroceles; 4, gland cysts.

1. **Retention cysts.**—When the duct of a gland becomes obstructed, the secretion, hindered from escaping, accumulates in the ducts and acini and dilates them. If the obstruction be maintained or oft repeated the gland tissue becomes impaired, then atrophies; finally, the gland and its duct are converted into a fluid-containing sac or cyst.

It is generally believed that when the duct of a gland is completely obstructed the conversion of the parts into a cyst is a passive process; this, however, is not the case. When an excretory duct is so completely obstructed that no secretion escapes, then the gland rapidly atrophies. Retention cysts are due to obstruction to the free flow of secretion, or temporary arrests of the flow frequently recurring.

The purest forms of retention cysts arise in connection with hollow organs, the inner walls of which are provided with glands.

The chief species of retention cysts are hydrometra, hydrosalpinx, hydronephrosis, and hydrocholecyst.

Any of these cysts may, and often do, reach very large proportions and endanger life, not only by interfering with the due performance of the functions of the organs in which they arise, but the large size some of them attain to causes them to interfere with adjoining viscera and to encroach on the thorax by displacing the diaphragm. In addition, the contents of these sacs often consist of highly albuminous material, and should septic organisms gain access to it, either from without or in consequence of adventitious communications with hollow viscera, especially intestine, suppuration with all its attendant evils will be the consequence.

This change is expressed by slightly altering the name; thus a hydrometra becomes a pyometra, and a hydrocholecyst becomes a pyocholecyst, and so on. It must be remembered that a dilated renal pelvis, or an abnormally large gall-bladder, or a distended Fallopian tube, may have contained pus from the outset of the trouble.

2. **Tubulo-cysts.**—This genus includes cysts arising in functionless ducts. These ducts belong to structures which were useful to the embryo—*e.g.* the vitello-intestinal duct and the urachus. Others, like the parovarium and Gartner's duct, are serviceable in the male, but, so far as we know, useless in the female. Functionless ducts must not be confounded with obsolete canals. (*See* page 490.)

This genus contains six species:—Cysts of the vitello-intestinal duct, allantoic (urachus) cysts, cysts of the paroöphoron, cysts of the parovarium, cystic disease (adenoma) of the testis, cysts of Gartner's duct.

3. **Hydroceles.**—These consist of excessive accumulation of fluid in the peritoneal diverticula which accompany the descent of the testis in the male, the round ligament of the uterus in the female, hernial sacs, and the occasional ovarian pouch.

There are four species:—hydrocele of the tunica vaginalis, hydrocele of the canal of Nuck, hydrocele of hernial sacs, hydrocele of ovarian sac.

4. **Gland cysts.**—This genus includes cysts arising in connection with the salivary glands and those of similar construction, such as the lachrymal and pancreatic glands. Thus it comprises those to which the term *ranula* is often applied, and *dacryops.*

There is a strong tendency to restrict the term ranula to cystic dilatation of the ducts of the three sets of salivary glands, and to designate them as submaxillary, sublingual, and parotid ranulæ. If surgeons would use the term in this definite sense, much unnecessary discussion would be saved.

In the majority of cases ranulæ are probably retention cysts due to obstruction of a duct. They are common in connection with the submaxillary and sublingual glands. The cysts are, as a rule, thin-walled, and lie in the furrow between the gum and the tongue, and bulge upwards into the floor of the mouth. When large they cause a prominence in the submaxillary triangle. The cyst may be filled with saliva. Sometimes it contains mucus and a yellow substance resembling the yolk of an egg.

Occasionally the obstruction is caused by a calculus impacted in the orifice of the duct, but cases come under observation in which the duct is not completely obstructed, yet the fluid is retained. It is reasonable to believe that ranulæ sometimes arise independently of obstruction to the main duct, and, as in the case of pancreatic ranulæ, observation supports the view that there is a pathological cause apart from mere obstruction concerned in their production.

Parotid ranulæ are very rare.

Dacryops, cystic dilatation of the ducts of the lachrymal gland, are exceedingly rare.

Cystic dilatation of the duct of the pancreas—*pancreatic ranula*—is a common condition, and is rarely a source of trouble; but it must not be confounded with those large extravasations of pancreatic fluid and blood behind the peritoneum or in the lesser sac of the peritoneum which so frequently follow severe injuries to the belly.

PSEUDO-CYSTS

There are conditions often classed as cysts which may, with greater propriety, be arranged in a sub group under the title pseudo-cysts. This sub-group contains four genera:—1, Diverticula; 2, bursæ; 3, neural cysts; 4, parasitic cysts.

1. Diverticula.—The name diverticulum is used to denote hernia or protrusion of the lining membrane of a cavity through a defective spot in its walls. Such protrusions occur in the pharynx, œsophagus, intestines, bladder, larynx, and trachea.

When such protrusions occur in connection with joints, they are termed *synovial cysts* and, in the case of tendon sheaths, *ganglia*.

Localised projections of the meninges of the brain or spinal cord beyond their bony coverings are referred to as *meningoceles*.

2. Bursæ.—Where muscles and tendons glide over osseous surfaces, or in situations where skin lies in contact with bony prominences, membranous sacs filled with glairy fluid occur; these are bursæ. Many of them are constant structures, *e.g.* the prepatellar and olecranon bursæ. Others are due to pressure resulting from habit or particular occupations, and may arise in any situation where the skin is subjected to intermittent pressure combined with friction.

Most subcutaneous and many subtendinous bursæ arise after birth. When a subcutaneous bursa attains an abnormal size, it is invariably due to unusual pressure associated with particular occupations. For instance, too much kneeling on hard material, whether in housemaids, devout persons, or carpet-layers, produces the familiar prepatellar bursa; repeated blows on the elbow produce the miner's bursa; from carrying weights on the shoulder porters are liable to acquire a bursa over the acromial end of the clavicle; tailors from their cross-legged habit of sitting are sometimes troubled with one over the external malleolus; whilst weavers and lightermen, from prolonged sitting on hard seats, suffer from enlargement of the bursæ over their ischial tuberosities; soldiers, when sleeping too frequently on the hard floor of the guard-room, irritate those over their greater trochanters; the pressure of ill-fitting boots develops a bursa over the enlarged head of the metatarsal bone of the hallux. When associated with partial dislocation of the first phalanx it is known as a bunion, and bursæ are quite common on the ends of amputation stumps.

A bursa is often present between the body of the hyoid and the thyro-hyoid membrane; sometimes it is as large as a ripe cherry.

Bursæ are liable to inflame, a process that may lead to suppuration, or stop short of that condition and become chronic or recurrent, and lead to secondary changes in the wall of the sac, so that its cavity becomes almost obliterated. A chronically-inflamed bursa sometimes attains the size of a fist, especially the prepatellar and ischial varieties. An enlarged prepatellar bursa sometimes rapidly thickens and its cavity becomes obliterated in the syphilitic.

3. **Neural cysts.**—This genus includes those abnormal conditions of the brain known as meningoceles and encephaloceles or their combinations, and dilatations of the ventricles included in the term hydrocephalus. Also the malformations of the spinal cord and membranes comprised in the name spina bifida.

4. **Parasitic cysts.**—This genus will include echinococcus cysts and colonies, and the cysticercus stage of tænia solium occasionally met with in man.

Summary.

Cysts.

Genera.	*Species.*	*Genera.*	*Species.*
1. Retention cysts...	Hydrometra.	3. Hydroceles	... Of tunica vaginalis.
	Hydrosalpinx.		Of canal of Nuck.
	Hydronephrosis.		Of hernial sacs.
	Hydrocholecyst.		Ovarian.
2. Tubulo-cysts	... Vitello-intestinal.	4. Gland cysts	... Ranulæ.
	Allantoic (urachus).		Dacryops.
	Paroöphoronic.		Pancreatic cysts.
	Parovarian.		
	Testicular.		
	Gartnerian.		

Pseudo-cysts.

Genera.	*Species.*	*Genera.*	*Species.*
1. Diverticula	... Diverticula.	3. Neural cysts	... Meningocele.
	Synovial cysts.		Hydrocephalus.
2. Bursæ ...	... Bursa.		Spina bifida.
		4. Parasites ...	... Hydatids, etc.

XXIII. INJURIES OF BLOOD-VESSELS.

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In this Article will be considered the various points connected with the injuries of blood-vessels and their repair, and also the questions connected with the most constant and important result of these injuries—hæmorrhage.

I. INJURIES OF ARTERIES AND VEINS.

Injuries of arteries may be caused by any kind of contusion, laceration, or wound; but as the effects of the various forms of injury differ in important particulars, they must be considered separately.

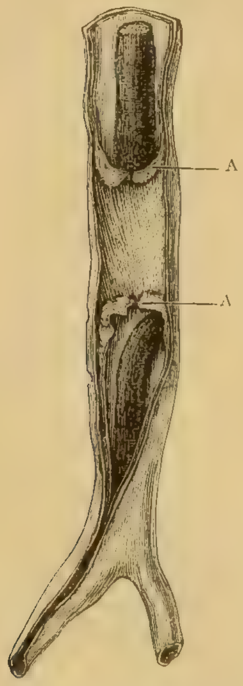

Fig. 132.—Contusion of Artery, showing rupture of inner coats and coagulum on each side of the injury.
A, Curled up ends of inner coats.

1. Contusion and subcutaneous rupture of arteries.

—Contusions are often subcutaneous injuries. They are produced by blows, severe crushes of a part, stabs with blunt weapons, and bullets travelling at a low rate of velocity. As the contusing force varies in intensity within wide limits, the effects produced upon an artery are very diverse.

In the slightest cases there is no obvious lesion of the vessel, although examination with the microscope may reveal minute tears in the muscular coat. The chief effect of this slight injury is to lead to proliferation of the endothelium. This is followed by coagulation of the blood within the vessel (thrombosis) and permanent obliteration.

A more severe contusion tears across more or less completely the two inner coats of the vessel, the tough and elastic outer coat remaining intact (Fig. 132). On the uneven surface of the outer coat and the curled-up ends of the inner coats the blood usually quickly coagulates, and the vessel is occluded. But the force of the circulation may produce two other effects: it may over-distend and rupture the thin tunica adventitia and lead to external hæmorrhage or a traumatic aneurysm, or it may strip up the divided inner coats and fold them down like a valve, or even invert them as a complete tube within the vessel. These effects are well seen in the accompanying drawings of specimens in the Middlesex Hospital Museum (Figs. 133, 134).

Still more severe contusion severs all the coats of the vessel, producing its so-called *rupture.* Owing to the curling up of the inner coats, and to the outer coat being drawn out, frayed, and falling over the end of the vessel, coagulation of the blood may rapidly occur, and even in the case of large arteries there may be little or no hæmorrhage. But if the ends of the ruptured artery are not thus closed at once, blood escapes from one or both of them; and if the rupture be subcutaneous, the blood infiltrates the cellular tissue of the part, often spreading for a great distance, and producing great tension of the parts, which interferes with the venous circulation and with the anastomosing arteries, so that gangrene quickly comes on. (*See also* page 142.) The discoloration of an ordinary bruise is due to the escape of blood from small vessels which have been ruptured by the contusion.

Contusion of an artery may have two other serious effects. It may so impair the vitality of the arterial wall, and lessen thereby its power of resisting the influences of micro-organisms, that if the surrounding tissues are infected, septic arteritis quickly ensues, and runs on to ulceration and sloughing of the vessel with great danger of secondary hæmorrhage. The contusion may also destroy the vitality of the outer coat without severing it, and the separation of the dead from the living part of the vessel is attended with the dangers of traumatic aneurysm and hæmorrhage.

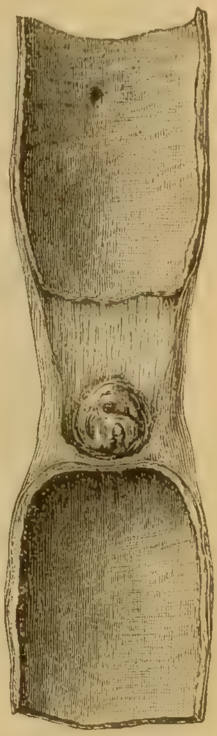

Fig. 133.—Contusion of an Artery, showing the division of the inner and middle coats and the curling up of these severed coats within the artery. (Middlesex Hospital Museum.)

The effects of contusion of an artery, therefore, are very varied. The most frequent result is occlusion of the lumen of the vessel by blood clot and displaced portions of the coats, with the risk of gangrene from diminished supply of blood. The division of the coats of the vessel causes the weakening of the artery and a lessened power of resisting the blood pressure, which may result in aneurysm or hæmorrhage. The impairment of the vitality of the vessel introduces the dangers of sloughing and ulceration, with the probability of secondary hæmorrhage.

Symptoms.—Contusion of an artery can only be recognised during life by its secondary effects—arterial obstruction, gangrene, aneurysm, or secondary hæmorrhage. Thus, if a contusion be immediately followed by loss of pulsation in the artery at the place struck, division of its inner coats, at any rate, with thrombosis, may be inferred; and, if these symptoms be delayed for some hours, the slighter degree of contusion with thrombosis may be diagnosed. As a striking example, the following case recently in the wards of the Middlesex Hospital may be quoted. A man struck his right groin against the corner of a table; a very slight bruise was found by the house-surgeon exactly over the upper part of the common femoral artery. Strong pulsation could be felt in the iliac artery above this point, but it ceased abruptly at the seat of injury. When he left the hospital, in three weeks' time, pulsation had returned in the superficial femoral and tibial arteries, but the common femoral was a firm pulseless cord. The exact limitation of the injury in this case to the contusion and permanent obliteration of a short length of the common femoral artery made the case a very striking and valuable one. The effect of this obstruction upon the parts supplied by the artery depends upon the vessel involved and the other injuries sustained; in the worst cases gangrene ensues in others the local anæmia is more or less rapidly recovered from. (*See* page 534.) If an aneurysm develop quickly after a blow upon an artery, and at the point struck, this may be attributed to the effects of contusion of the artery. If, again, secondary hæmorrhage ensues upon a contusion, and the blood flows from a vessel which did not bleed at the time of the injury, it is to be regarded as due primarily to contusion of an artery.

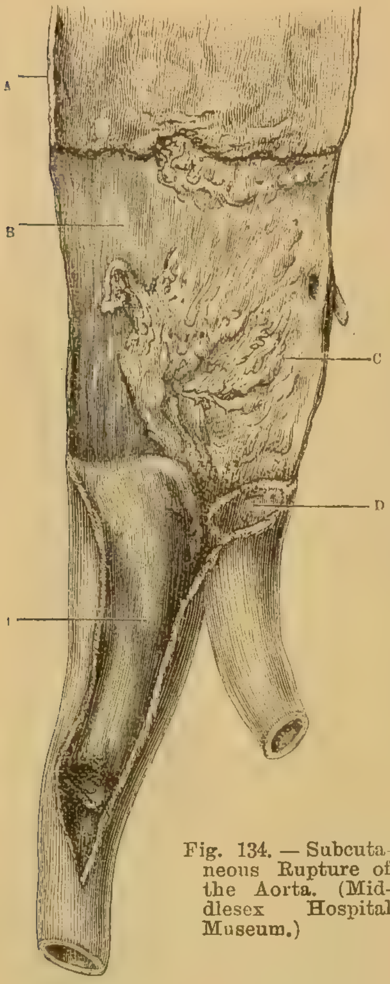

Fig. 134. — Subcutaneous Rupture of the Aorta. (Middlesex Hospital Museum.)

A, Artery laid open, showing the coats intact; B, unruptured outer coat, very thin, with a thin layer of clot (C) adherent to it; D, left common iliac artery, coats entire; E, right common iliac artery, plugged with the inner and middle coats of the aorta which have been detached and forced into the artery.

Subcutaneous rupture of an artery is recognised by sudden pallor, coldness, and benumbing of the limb beyond the rupture, and by the rapid formation of a tense swelling about the seat of rupture.

This swelling, as a rule, has no pulsation, but when very tense a slight pulsation may be detected by it; as a rule, too, there is neither bruit nor thrill; both may, however, be present. The swelling rapidly increases, is usually acutely painful, but the local heat is not increased. The arteries beyond the rupture are pulseless, and the tissues here become œdematous, livid, and cold, and if the condition be not relieved moist gangrene results. The patient may also show the signs of considerable hæmorrhage—pallor, weakness, and a rapid, feeble pulse.

Treatment.—Where, as the immediate or later result of contusion, an artery is obstructed by a thrombus, the treatment is directed to maintaining the vitality of the parts rendered anæmic. They must be kept at rest, warm, and protected from all constriction, pressure and other injury. If the anastomotic circulation is established, nothing else is required; if, however, it fails—owing to contusion of the collateral vessels, as well as of the main trunk, or to disease of these vessels—and gangrene ensues, amputation at the level of the obstruction in the artery should be performed as soon as the patient's general condition permits of it.

In cases of contusion and obstruction of a large artery such as the common femoral or axillary, when an entire limb is bloodless, there is sometimes considerable shock for some hours after the accident, which may even prove fatal. In the case of contused wounds great care must be taken to render the parts aseptic and to secure rapid healing by first intention. The patient must be closely watched for the first appearance of secondary hæmorrhage, which must be arrested by ligature of the artery above and below its contused area.

Where an artery has been severed by crushing or tearing, if its ends are closed it should not be ligatured or twisted; but if any hæmorrhage is occurring from either end, that end should be carefully tied. Special care should be taken to examine the distal end of such a vessel. Some advise that an obviously contused artery which remains patent should be at once subjected to the double ligature without waiting for bleeding to occur. The cases in which the diagnosis can be made before hæmorrhage occurs are very few.

When an *artery* is *ruptured subcutaneously* the principles of treatment are the same. Thus, if there is no subcutaneous hæmorrhage, the surgeon's efforts are all directed to maintaining the vitality of the anæmic parts until the anastomotic circulation is established. Should this fail, amputation at the seat of rupture must be performed. If blood is escaping from either end of the ruptured artery the surgeon must cut down upon it, turn out all the effused blood, and ligature both ends of the vessel. Supposing that there has been a considerable extravasation of blood from the wounded artery, but the hæmorrhage from it has ceased, an incision to turn out the clots must be made, because these clots form a serious obstruction to the venous return from the parts beyond, and may even impede the establishment of the arterial anastomotic circulation. In either case the clots may lead to gangrene, and it is to prevent this that they should be removed at once. When gangrene has occurred, amputation is the only course open. (For the treatment of traumatic aneurysm *see* page 632.)

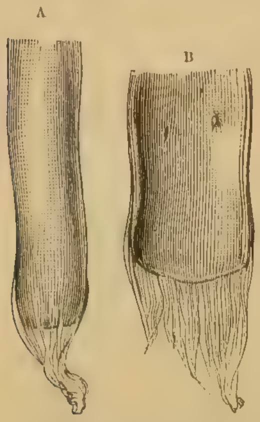

Fig. 135.—An Artery severed by Tearing.

A, The unopened vessel, showing the frayed-out outer coat over the end of the divided inner coats; B, the artery laid open, showing the clean-cut inner coats.

2. Laceration of arteries.—It is well known that lacerated wounds are attended with very little, if any, hæmorrhage, and for this reason an écraseur is used in certain operations upon vascular tissues. When an artery is torn across, the brittle inner and middle coats quickly snap and curl up within the artery, and on them a clot forms. The outer coat is drawn out and stretched beyond the degree of its elasticity, and then breaks, leaving a frayed end to fall over the end of the artery (Fig. 135). Such vessels often protrude beyond the general surface of the wound, and then they can be seen pulsating almost to their extremity.

Avulsion of the entire upper limb with the scapula has occurred more than once from the hand getting caught in revolving machinery; even in such an extensive wound as this there may be no bleeding.

The only direct *treatment* for a lacerated artery which has ceased to bleed is to keep the part at rest and secure aseptic repair. If the artery is bleeding, it must be tied. Watch must be kept upon the patient that, if secondary hæmorrhage occur, it may be arrested and treated at once.

3. Punctured wounds of arteries.—Punctured wounds of arteries form an extremely important class of injuries, for while of apparently trivial nature, their consequences are often very grave. They are generally produced by stabs and punctured wounds of various kinds, occasionally by the surgeon in operating. Pins and fish-bones have been known to transfix the œsophagus and pierce the thoracic aorta, and cause fatal hæmorrhage. In fractures, sharp fragments of bone may pierce a neighbouring artery, and in removing a sequestrum from the femur its sharp point has been known to puncture the popliteal artery. A fine clean needle may be passed through an artery without any ill-effect.

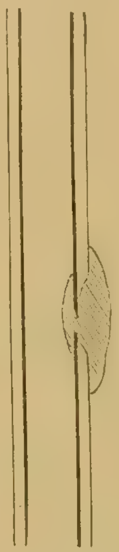

Fig. 136.—Diagram of a Punctured Wound of an Artery, showing the blood-clot (A) occupying the puncture, overlapping it in the sheath of the artery and project-into but *not obliterating* the lumen of the artery.

Effects.—The disastrous consequences of a punctured wound of an artery are due in the first place to the fact that the clot in the puncture does not project into the artery and occlude it. The flow of blood through the artery, therefore, is uninterrupted, and the full blood pressure continues to be exerted upon the puncture all through the process of healing. The clot that forms in the puncture is supported, however, by a clot outside the artery in its sheath (Fig. 136). When the blood pressure rises during recovery from shock, during movement or excitement, or from the effects of stimulants, it often displaces this clot and causes a renewal of the hæmorrhage.

Another very important fact is that punctured wounds of arteries are often deep wounds, and are very liable to be oblique; the blood which escapes from the artery does not find a ready exit, and it accumulates in the tissues, coagulates there, and, the hæmorrhage still continuing, a traumatic aneurysm is formed. (*See* page 629.) In these cases, too, the surgeon is liable to close the external wound with his dressings without arresting the escape of blood from the artery, and the blood, collecting in the tissues, produces an aneurysm. The third element of danger arises from the risk of infection of the wound in the artery, either from the puncturing body or from secondary infection of the deep, irregular, oblique, ill-drained wound.

The effects of a punctured wound of an artery, therefore, are hæmorrhage, which is very prone to recur owing to displacement of the clot (intermediate hæmorrhage), traumatic aneurysm, and septic arteritis leading to secondary hæmorrhage. Where the companion vein is punctured at the same time as the artery, arterio-venous aneurysm may result. (*See* page 633.)

Treatment.—The only reliable treatment of a punctured wound of an artery is the aseptic ligature of the vessel above and below the puncture. This should be carried out at once, and for this purpose it is generally necessary to enlarge the wound. If the companion vein is found punctured, it, too, is to be tied if the hæmorrhage continues after the artery is secured. Pressure with a graduated compress or pad and bandage has often been employed, and its results have, as a rule, been disastrous, owing to the formation of an aneurysm or to recurrence of the bleeding. If the pressure is sufficiently firm to arrest the flow of blood through the artery until the puncture is firmly healed, it may be successful. (*See also* page 513.)

4. Incised wounds of arteries.—Incised wounds are the most frequent of all wounds of arteries; they are characterised by the excessive hæmorrhage they occasion. If the wound in the artery is longitudinal, it gapes but little, and chiefly during the ventricular systole; its healing may take place quickly. If the wound is transverse to the long axis of the artery, it gapes widely, and when it involves more than half the circumference of the vessel the retraction of the divided part makes a very large opening in the vessel. An oblique wound in an artery is intermediate in character (Fig. 137). When an artery is completely divided across, its ends contract and retract within its sheath, as will be more fully described when we speak of the natural arrest of

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hæmorrhage. Non-penetrating wounds of arteries are very occasionally met with. The outer coat, or the outer and middle coats only, may be severed, but in these cases the inner coat subsequently gives way; it is said to form a hernial swelling before it yields. A wound with a blunt knife has been known to divide the inner and middle coats without severing the tougher outer coat.

Effects.—A longitudinal wound and a transverse one not exceeding one-fourth of the circumference of the artery, heal—if at all—without occluding the lumen of the vessel; they are, therefore, in this respect like punctured wounds, and, like them, liable to frequent repetitions of the hæmorrhage from displacement of clot, and rupture of the forming cicatrix. If the wound down to an artery is oblique, or is closed by the treatment employed, blood may escape into the tissue about the incised artery, and an aneurysm form.

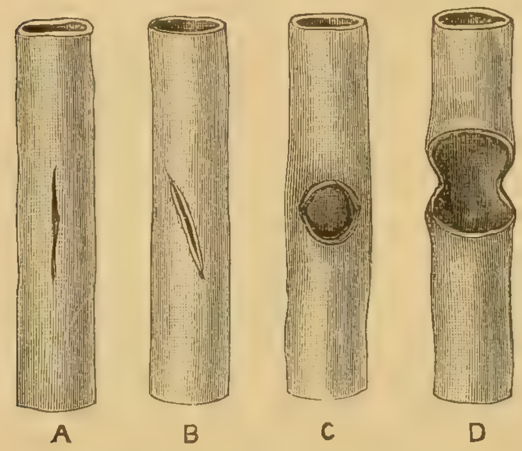

Fig. 137.
A, Longitudinal wound of an artery; B, oblique wound of an artery; C, transverse wound of artery, extending about one-quarter of the circumference; D, transverse wound extending more than half across the artery.

The great symptom of these wounds is free external hæmorrhage. In the case of the largest arteries this is quickly fatal; in vessels of smaller calibre the fatal result is longer delayed, and may be prevented by coagulation of the blood when syncope occurs; even small arteries, when incompletely severed and unable to contract and retract, may be the source of very grave hæmorrhage. When the main artery of a part is widely opened, the tissues it supplies are rendered more or less completely bloodless, and its branches pulseless; the degree to which this follows depends not only upon the size of the wound in the artery, but also upon the presence or absence of other channels of supply to the part.

Treatment.—A completely-divided artery should be twisted or tied if blood is escaping from it; and this applies with special force to the distal end of the artery. In all cases of incomplete division of an artery—independently of the actual occurrence of hæmorrhage—the treatment is to ligature the artery above and below the wound in it, and divide the vessel between the two ligatures. The continuance of the stream of blood through an artery, which is the seat of a lateral wound, exposes the patient to serious danger, and should always be avoided.

The important points to be specially borne in mind in the treatment of injured arteries are:—(1) To secure the total occlusion of every wounded artery; no attempt must be made to obtain healing of the wound in the vessel without obliteration of its lumen.

(2) When nature has closed the end of a divided artery, the surgeon should not interfere with it.

(3) Every open artery—proximal or distal end—should be closed as quickly as possible.

(4) All obstructions to the circulation through other than the injured vessels should be carefully removed.

(5) Gangrenous parts should be removed at the level of the injury to the artery.

5. Gunshot wounds of arteries.—These injuries are rare in civil practice, but are a very frequent cause of death in warfare. (*See* page 240.) A bullet may cause contusion of an artery of any degree of intensity, or it may completely sever a vessel, and in certain situations such an injury involves a great risk of gangrene, especially if the companion vein is also wounded and subsequently tied. The special lesions caused by bullets are two: one is the scooping out of a portion of the circumference of an artery, and the other is the double perforation of a large artery, the projectile passing quite through it. Portions of clothing and accoutrements may be carried by a bullet into the deep parts of a wound, and even into the injured vessel and act as a plug, so that there is no hæmorrhage until these foreign bodies are removed. Such wounds are often very deep, and the arteries involved inaccessible, while the risk of complication with other injuries, such as fracture of bone, wound of vein, nerve, joint, or viscus, and the danger of wound-infection from foreign matter carried in with the bullet or from extension from the contaminated surface of the wound, are very great.

The **treatment** of gunshot wounds is discussed elsewhere (page 247). Gunshot wounds of arteries must be treated like other injuries; foreign matters should all be carefully removed; partially-severed vessels should be secured by double circular ligatures; completely-severed arteries should be tied if bleeding. The utmost care should be taken to render, and to keep, the part aseptic. Where it is certain, from the nature of the injury, that a useful limb cannot be obtained, primary amputation should be practised. If gangrene occurs, amputation should be at once performed at the level of the injury to the artery.

6. Injuries of veins. — These lesions are produced by the same forms of violence as injuries of arteries. **Contusions** of veins lead to coagulation of the blood within them; when more severe, death of all the coats of the vein is caused, with thrombosis; this occurs in severe crushes of parts, such as are followed by local gangrene. Owing to the lower blood pressure within the veins, the danger of dilatation of the vessel or of hæmorrhage—primary or secondary—is very much less than in contusion of arteries.

Lacerations occur where limbs or parts of limbs are violently torn off, and in the reduction of dislocations, especially when these are of old standing. Laceration may completely occlude a vein as an artery, but, owing to the smaller amount of elastic and muscular tissue in its walls, a torn vein may continue to bleed when its companion artery is completely closed. Thus it happens that very extensive and even fatal hæmorrhage may occur from a subcutaneous laceration or rupture of a vein. In certain situations the veins are unable to collapse, owing to their walls being adherent to rigid structures, *e.g.* the cranial sinuses and the hepatic veins; and from these veins furious hæmorrhage may occur.

Wounds of veins are of frequent occurrence, and if they involve deep and large vessels they are very serious injuries. Owing to the low pressure of the blood in the veins, and to the tendency of divided veins to collapse, the hæmorrhage is less rapid than it is from wounds of the corresponding arteries. For this reason, also, it is easy and safe to arrest venous hæmorrhage by external pressure.

Treatment.—Wherever possible injured veins should be allowed to heal without obliteration of their lumen; lateral wounds and punctures should be closed by a lateral ligature, and as the blood-pressure is so little, pressure may be safely employed to occlude veins. The cardiac end of a divided vein need only be ligatured when it is in the "dangerous area," or above valves so that air may be drawn in or blood escape from it. When the main vein of a limb has to be tied, the main artery, if not wounded, should not be also tied in order to prevent the occurrence of moist gangrene. Indeed, the chance of gangrene is increased when the *vis a tergo* of the circulation is diminished at the same time as an obstruction is placed to the venous return from a part. Where both vessels are wounded, both should be tied, if the wound in the vein is more than a small puncture.

7. Entrance of air into veins.—This is a rare accident. The suddenness with which it occurs and its alarming and often fatal consequences invest it with special interest and importance. Experiments on animals have shown that a small quantity of air may be injected into a vein without ill-effect, and, provided the injection is carried out very slowly and gradually, even a large quantity of air may be introduced without the disastrous consequences that ensue from the sudden entrance of a smaller amount.

Causes.—In the large veins near the heart there is negative blood pressure, the blood being drawn towards the right auricle by the aspiratory effect of inspiration. This negative pressure is at its maximum in the intra-thoracic veins and in those at the root of the neck, and lessens as we pass farther away from the heart. The influence of deep and laboured inspiration, however, may be manifest at some distance. If a vein in which the blood pressure is negative is wounded, and the wound held open and exposed in the air; air is sucked into the heart. If, however, the wound in the vein collapses, air is not sucked in. We thus see that the cause of the entrance of air into a vein is twofold: (1) the aspiration of the chest, and (2) the patency of a wounded vein open to the air. The patency of a wounded vein—or, as it has been well called, its "canalisation"—may be caused by thickening and rigidity of the vein itself, by infiltration of the sheath of the vein and surrounding tissues, by adhesion of the vein to some unyielding fascia, or to the wound being held agape by the surgeon. This may arise in two ways. A vein is sometimes partially divided in the angle of a wound, and then, when the edges are held apart, the wound of the vein is held open; or, in removing a tumour, if it is lifted up, and the parts held on the stretch as they are divided, any partially-divided vein is "canalised." As this accident is especially liable to occur in wounds and operations about the root of the neck, this region is called "the dangerous area." It may follow wounds of veins so far away from the heart as the facial and subscapular, and even the common femoral and uterine.

Effects.—The sudden entrance of a large quantity of air into the heart may cause its instant paralysis and sudden death of the patient. More commonly when air is drawn to the heart in quantity the right auricle and ventricle and the pulmonary artery are found full of frothy blood, and the left side of the heart empty. The right ventricle is unable to force the mixture of blood and air through the pulmonary capillaries, and the left ventricle, not being filled, is unable to send sufficient blood to the brain, and the patient dies from syncope. When only a small quantity of air is introduced into the circulation, it gradually becomes dissolved in the blood, and possibly some of it escapes through the lungs.

Symptoms.—These may be described as local and general. The sucking of air into a wounded vein is attended with a peculiar hissing or sucking sound, and frothy blood is then seen to issue from the vessel. The general symptoms vary much in intensity. There may be instant death from syncope. More commonly the patient suddenly becomes pale, the pupils dilate, the pulse is flickering or imperceptible, and the movements of respiration are exaggerated, the heart's action is powerful and turbulent, and on listening over the præcordia a churning sound is heard. These alarming symptoms may gradually subside, and the patient recover; or the heart's action may become feebler, and death occur, often preceded by a convulsion. When the accident is fatal, death usually occurs in a few minutes; but if successive quantities of air are sucked in, the fatal issue may be longer delayed. If the patient escapes the immediate danger, he may subsequently die from bronchitis or pneumonia.

Treatment.—In operating in the "dangerous area," particularly in the removal of tumours, care should be taken not to divide a vein incompletely at either end of the wound, and not to hold the parts on the stretch while dividing them. Veins when stretched collapse, and emptied of blood are pale in colour, and then are easily mistaken for bands of fascia. Another way of preventing the entrance of air during such operations is to flood the wound with a harmless and colourless fluid, such as boiled water, saline solution, or boric acid solution; then if a vein be cut this fluid, and not air, is drawn into the heart, and no ill-effect follows.

If, while operating, the peculiar sucking sound of air entering a vein is heard, or frothy blood is seen in the wound, the finger should be instantly placed over the vein until it can be secured with a forceps or ligature. This is to prevent the further entrance of air and the loss of blood. If the vein cannot be thus instantly secured, the wound should be immediately flooded with fluid, which will, at least, prevent all further suction of air. Having done this, the surgeon's aim is to *maintain the heart's action* until the obstruction in the pulmonary vessels is overcome by the gradual solution of the air or its escape into the air cells, and to furnish a *sufficient supply of blood to the brain* to prevent fatal syncope. The patient should be placed flat on his back with his head hanging over the end of the table, and the axillary and common femoral arteries should be compressed, so as to increase the proportion of arterial blood going to the brain. Stimulants should be administered freely—ammonia to the nostrils, ether subcutaneously, brandy by enema, and a sinapism over the præcordia. Attempts to suck the air out of the right side of the heart are useless; in children firm compression of the chest may succeed in expelling air from the wounded vein—not from the heart—but it embarrasses respiration, and, if at all, should only be practised once very rapidly. Artificial respiration, with a view of aiding the circulation through the pulmonary capillaries, has been recommended, but Treves considers this a dangerous measure to adopt.

II. HÆMORRHAGE.

The natural arrest of hæmorrhage and the repair of injured vessels.—Hæmorrhage, or the escape of blood from the heart and blood-vessels, is the most constant effect of injury, a frequent result of disease, and the invariable accompaniment of a surgical operation. Any loss of blood may be serious, and to the untutored eye it is always alarming. When furious and uncontrolled it is quickly fatal, and in some circumstances its arrest taxes the courage and resource of the boldest and most experienced surgeon. In certain positions, as within the skull and spinal canal, and in the mouth, air-passages, and thorax, the escaped blood causes the most serious indirect effects. For no other condition is surgical treatment so entirely efficient, and there is none for the relief of which patients are so dependent upon skilled assistance. All these circumstances combine to render the study of hæmorrhage and its treatment of supreme importance.

In studying the repair of injured vessels, we have to notice two distinct processes—the *arrest of hæmorrhage* and the *formation of a cicatrix* in the wounded vessel. These processes are of great scientific interest, but they are also of vital practical importance. It is true that Nature unaided can, in a large proportion of cases, arrest hæmorrhage before a fatal loss of blood is sustained;

but often, if life is saved, it is only at the expense of great or even profound anæmia, which may never be recovered from, and in certain cases the blood flowing from the wounded vessels becomes a source of new danger to the patient by collecting in the body, compressing and obstructing important parts, such as veins, arteries, brain, spinal cord, bronchi, and lungs. By surgical means we are able to arrest hæmorrhage instantly and certainly where Nature can do so only tardily and doubtfully, if at all, and so to save life and health and preserve intact the other parts of the body. Indeed, there is no single result of injury or disease for which treatment is so immediately and widely beneficial as in the arrest of hæmorrhage. But as the surgical means employed are all modelled on Nature's pattern, it is extremely important to understand her ways thoroughly and to appreciate the manner in which her efforts may be supplemented and aided. The cicatrisation of a wounded blood-vessel, however, like that of any other tissue, is effected by Nature alone; the surgeon cannot make a scar, nor can he do much to further the natural processes, although he may do much to hinder or impair them, and it becomes of the highest importance for him while aiding Nature to arrest hæmorrhage to use only those means which do not impede the process of repair of the wounded vessel. It is for this reason that we speak of the treatment of hæmorrhage rather than of the treatment of wounded vessels.

We shall consider, in the first place (1) how unaided Nature arrests hæmorrhage; this is sometimes spoken of as the temporary arrest of hæmorrhage. We shall then describe (2) the cicatrisation or true repair of wounded blood-vessels; this is sometimes called the permanent arrest of hæmorrhage; and in the next section (3) deal with the surgical means of arresting hæmorrhage.

1. The natural (or temporary) arrest of hæmorrhage.—When an artery is cut across, its *muscular coat contracts* and narrows or even closes the orifice; the instant closure of a cut artery is often seen when small arteries are divided in an operation. This contraction results from the direct stimulus to the muscular fibres of the injury, and is a vital effect; its influence is greatest in those arteries in which the muscular coat is best developed.

At the same time, the elasticity of the artery causes it to *retract within its sheath.* This retraction is both longitudinal and circular. The circular retraction narrows the orifice of the cut artery and the lumen of the vessel for some distance above. The longitudinal retraction withdraws the artery from the surface of the wound within its cellular sheath. This is a physical phenomenon, and can be demonstrated on the dead body.

These changes in the cut vessel—contraction and retraction—are immediate, and they are quickly followed by *coagulation of the blood.* As the blood flows through the narrowed mouth of the artery and over the inner surface of its cellular sheath, the blood platelets are entangled, disintegrate, set free their ferment, and fibrin is formed. The clot thus arising forms first in the sheath of the artery, and increases until it covers over and surrounds the end of the vessel, and stops all escape of blood as by a pad. This is the *external clot.* Blood coagulates also on the divided end of the artery and occludes it, and for a variable distance within the artery—often up to the branch next above or below the wound. This is the *internal clot.* The coagulation of the blood is the essential part of the process; by itself, without contraction or retraction of the vessel, it suffices. The contraction of the artery by narrowing the orifice lessens the force and volume of the stream of blood, and, together with the retraction, by drawing the artery within its sheath, makes room for the external clot, and also favours the occurrence of coagulation. By themselves, neither contraction nor retraction, nor the two together, suffice to arrest hæmorrhage, for even in those cases where the sudden contraction of the artery closes its cut end, it is only by the formation of an internal clot that the orifice is actually sealed over. The clot, then, is the essential means that Nature employs to arrest hæmorrhage. The most vital part of it is the portion that actually occupies the orifice of the vessel, and this may well be called the *central clot;* until that has formed the bleeding is not arrested, and with that, the rest of the clot may be, and sometimes is, dispensed with. The external clot is like a pad over the end of the vessel, holding in place the central portion, and resisting the force of the blood, which tends to displace it; it is a most valuable adjunct to the central clot. The internal clot acts as a buffer to protect the central clot from the full force of the circulation. The internal and external clots, therefore, though not essential, materially add to the security of the closure of the vessel; and the clot, as a whole, plays an important part in the cicatrisation of the vessel.

The natural (temporary) arrest of hæmorrhage is *aided* in particular cases by certain other conditions.

1. **Contusion of the artery.**—If, in place of an artery being cut across with a knife, it is bruised and crushed, the more brittle inner and middle coats give way first and curl up inside the vessel before the tougher, but thinner, outer coat is severed. On the curled-up ends of the inner coats coagulation of the blood quickly takes place; and so it happens that in severe contusions, or by the use of an écraseur which slowly crushes through the tissues, arteries of some size may be divided without even a drop of blood escaping from them. In such cases there is no external clot, the internal clot extends up the vessel as usual, and the curling up of the inner coats within the artery helps to occlude it, and also gives a wide surface for the attachment of the central clot; this makes it as secure as if supported by an external clot.

2. **Laceration of the artery** acts in much the same way. The tearing force soon snaps across the inner and middle coats, which curl up within the vessel, partially occlude it, and excite coagulation upon their upturned edges. The outer coat is drawn out until its elasticity is destroyed, and then it yields, and falls over the inner

coats as a long film. An artery as large as the brachial may be torn in this way without any blood escaping from it; of all the accessories to the one essential factor in the natural arrest of hæmorrhage—the formation of a clot in the wound in the vessel—none are so potent as contusion and laceration of the vessel.

3. **Diminished arterial tension.**—It is evident that with less pressure in the wounded artery the blood will escape more slowly, and will, therefore, coagulate more readily. A rapid forcible stream of blood tends to wash away the forming clot, while the slower gentler stream of blood does not displace the clot, and itself readily coagulates. This diminished tension may be suddenly induced by the physical shock of the injury, or by the sight of the blood escaping from the wound. Or it may be more slowly induced by the loss of blood weakening the heart and emptying the arteries.

4. **Increased coagulability of the blood.**—We shall have to notice (page 525) that one result of an abundant hæmorrhage is the absorption of serous fluid from the soft tissues, rendering the blood more watery. It is well known that the addition of a moderate amount of water to normal blood outside the body, increases the rapidity with which it coagulates; and it is believed that the same result may be produced in the late stages of a severe hæmorrhage.

It is in the case of complete division of the largest vessels only that unaided Nature fails to effect the arrest of the hæmorrhage; for the longer the bleeding continues, and the more blood is lost, the greater becomes her power, until when life is almost extinct, and the force of the circulation barely sufficient to carry it on at all, the most favourable condition for the formation of a clot exists, and the hæmorrhage may be arrested. Thus it is that patients recover even when completely blanched, pulseless at the wrist, and unconscious, and death from hæmorrhage alone is rare, except in the case of a wound of a very large vessel, causing a great rush of blood and rapid syncope.

The natural (temporary) arrest of hæmorrhage is *retarded* by the following conditions:—

(*a*) **Partial division of the artery.**—This altogether prevents longitudinal retraction, and the contraction of the muscular coat opens out instead of closing transverse and oblique wounds of the vessel. (*See* Fig. 137.) The stream of blood coursing along the artery also tends to displace any clot that forms in the wound, and prevents the formation of an "internal clot" to protect the "central clot." Further reference is made to this point in the section on punctured wounds of arteries. (*See* page 500.)

The complete division of an artery thus wounded and bleeding will sometimes be followed by an immediate arrest of the hæmorrhage.

(*b*) **Adhesion to its sheath** is another frequent mode of preventing the contraction and retraction of a cut artery. In certain situations this occurs normally—*e.g.* the scalp and the penis—and it is met with in disease as the result of chronic periarteritis and œdema. Wounds in such parts and cases bleed to an extent and for a length of time quite out of proportion to the size of the vessels divided.

(*c*) **Movement of the part,** if slight, may displace a forming clot, and so prolong the hæmorrhage. If violent and excited, it also acts by increasing the force of the heart's action. In sponging a wound, if the sponge is rubbed over the surface instead of being gently pressed upon it, a similar displacement of clot is often occasioned.

(*d*) **Stimulation of the heart,** by increasing the blood pressure in the arteries, directly impedes the arrest of hæmorrhage, or starts it afresh. This stimulation may be physical, as by movement, change of posture, the exhibition of "stimulants"—alcohol, ether, ammonia, etc.—or emotional. The importance of this fact will be insisted on when we speak of the treatment of hæmorrhage.

(*e*) **Division of an artery in a muscle.**—When a muscle is cut transversely to its fibres, the muscular tissue often contracts farther than the arteries in it retract, and the ends of the vessels are left projecting from the cut surface of the muscle. This prevents the formation of a good external clot. Such vessels are, however, usually quickly closed by the formation of a central clot in their contracted mouths.

(*f*) **Hæmophilia.**—(*See* page 376.)

The arrest of hæmorrhage from a **wounded vein** is effected in a similar way by the coagulation of blood in the orifice of the vein—the *central clot*—supported by an *external clot*, and protected by the *internal clot* extending a short distance along the vein. The internal clot is shorter in a vein than in an artery, and, owing to the lessened blood pressure in a vein, there is not the same necessity for it. Contraction and retraction are very slightly marked in veins, and are of no practical importance. But, on the other hand, the *walls of a cut vein collapse;* and this greatly aids in the formation of a central clot. The low blood pressure in a vein greatly favours the coagulation of the blood, and the adhesion of the clot to the cut end of the vein. When the walls of a vein cannot collapse, and when the blood pressure is greater, venous hæmorrhage may be fatal. It is to be remembered that in many cases there is no hæmorrhage from wounded veins, or the pressure of the blood in them is reduced to a minimum. Thus, when a vein is cut across, valves above the wound may prevent any blood from flowing from the cardiac end; and if the companion artery is severed at the same time, the stream of blood through the capillaries is arrested, and none reaches the distal end.

2. The repair of wounded vessels (or the natural permanent arrest of hæmorrhage).—This is accomplished by the formation of a *cicatrix* in the vessel, which seals over the wound, and in its main features the healing of a divided artery or vein resembles the healing of a wound in any other tissue. This process in other tissues has been already fully described; and here we need only note the features peculiar to the healing of blood-vessels.

In the first place, it is necessary to observe that while the clot plays an all-important part in the temporary closure of the vessel, it takes no direct active share in its permanent closure or healing. It affords a certain amount of pabulum for the cells from which the cicatrix is developed; it protects the formative tissue from the impact of the blood when it is soft and unable to withstand it; it may form to some extent a mould in which the cicatrix is developed; but eventually the whole of the clot—fibrin and blood corpuscles—disappears. The "external clot" is sometimes partly washed away with the abundant serous exudation from the wound; into what remains leucocytes wander from the vessels of the sheath and surrounding tissues, and they are quickly followed by the true plasma cells, derived from the hyperplasia of the cells of the sheath and other divided tissues. These plasma cells are found at first in the minute spaces or channels which are formed in the clot, probably by its contraction (Fig. 138). They rapidly multiply, and absorb and replace the clot. They then organise into vascular connective tissue, at first richly cellular and very vascular, gradually becoming more fibrous and less vascular. This external cicatrix is continuous with that of the neighbouring severed tissues, which it resembles in its origin and developmental changes.

Fig. 138.—A Portion of Clot in an Artery fourteen Days after Ligature, showing cracks and fissures. × 90. This part of the clot was not adherent to the artery, and had not been as yet invaded by leucocytes or plasma-corpuscles. (Ballance and Edmunds.)

The artery itself contracts upon the tapering internal clot, and as the clot fills out its divided end a bulbous form is sometimes given to the vessel. This appearance of a bulbous enlargement of the end of the vessel is not the result of yielding of the artery, but of contraction of its upper part. The internal clot at first is adherent to the artery at its divided end only, and here a change of colour from black-red to grey is early noticed. The grey colour gradually extends through the clot, and at the same time it is found to adhere to the lining of the vessel more and more extensively. Gradually now the line of demarcation between the arterial wall and the altered clot becomes less distinct, and the clot is found to be vascular; the whole vessel as far as the clot narrows more and more, until at last a vascular cord of cicatricial tissue is all that exists to mark the site of the former vessel, and this scar is continuous with that of the surface of the wound. The obliteration of the lumen of the artery extends as far as the internal clot, and thus the length of this clot is of real moment.

These are the changes which can be observed with the naked eye. The *minute processes* appear to be as follows:—The vessels in the outer coats of the artery dilate, and an exudation of serum and leucocytes occurs from them which infiltrates the coats of the artery and the contained clot. Following this exudation, and apparently resulting from it, the connective tissue-cells of the artery rapidly proliferate, and the plasma cells thus formed pass into the clot, first of all at its attachment to the end of the vessel—the central clot—and later at other points of its circumference, the cells passing through the coats of the artery in more or less vertical columns. At the same time the endothelium lining the tunica intima undergoes rapid hyperplasia until it forms a layer many cells deep. This hyperplasia extends as far as the internal clot; the cells resulting from it seal over the outer surface and fine end of the clot, and, together with those coming from the outer coats of the vessel, wander into the clot, passing along the intercommunicating fissures and spaces in the clot which result from the contraction of the clot, and which appear to have some relation with the nodal points around which the fibrin forms. Concurrently with this infiltration of the clot with these plasma cells, the red corpuscles are found to be disintegrated, and they are ultimately absorbed, and the clot is replaced by "lymph." This plastic lymph organises into a vascular connective tissue, which gradually becomes more fibrous and less vascular, and shrinks and blends with the surrounding cicatrix. Concurrently with these changes, the muscular tissue of the artery slowly disappears, and at length there is little left to mark the site of the artery but a vascular fibrous cord. The vascularisation of the clot is brought about in a very interesting way. As mentioned above, a series of intercommunicating fissures appear in the clot, and those on the surface open into the lumen of the vessel. Through these communications blood is forced from the artery; the spaces receive a lining from the endothelial cells, and later new capillaries connect them with the vasa-vasorum of the vessel wall. The cicatricial tissue which replaces the wounded artery receives its blood supply, therefore, partly from the blood in the artery itself and partly from the vasa-vasorum. In an artery the stream is from the vessel; in a vein it is probable that the stream is towards the vessel. This fact is important. The organising clot is traversed by blood channels rather than distinct vessels, in which the blood pressure is equal to that in the artery, and this is why the healing process in an artery may be interrupted, and secondary hæmorrhage occur, even apart from septic infection and inflammatory disintegration. In the healing of a lateral wound or puncture in an artery, the scar is formed in the same way; but being exposed to the full force of the blood stream, it is exceedingly liable to be torn through before development has advanced, or subsequently to yield, and to form an aneurysm.

The *healing of a wounded vein* differs but very little from the same process in an artery. When a vein is severed the process is identical. But in the case of a lateral wound in a vein, the scar is formed without obliteration of the lumen, and owing to the low blood pressure in the veins this process is a safe one. Indeed, there is much less tendency to obliterate the vessel in the healing of a wound in a vein than is the case in an artery. The most striking evidence of this used to be afforded by phlebotomy. Patients were often bled every spring and autumn for years, and from the same vein; repeated division of the vein failed to obliterate it. For the same reason, the operation of "pinning" a vein often fails to obliterate it.

3. **The surgical means of arresting hæmorrhage.**—The surgical methods of arresting hæmorrhage are designed to produce the immediate closure of the wounded vessel in ways closely resembling those of Nature. The means employed cause either contraction of the cut artery, or contusion or laceration of its coats, diminution of the force of the blood stream in the wounded vessel, coagulation of the blood, or closure of the opening in the vessel by external pressure. A knowledge of the mode in which they effect their purpose is essential to the proper employment of these means, and we must therefore study each of them separately.

1. **Pressure** is the most widely applicable and readily available of all hæmostatics; at the same time, it may be the most misleading and most mischievous. It is exceedingly important, therefore, to understand clearly its limits of usefulness and the elements of success and of failure.

It can be employed as a *temporary expedient*, either to close the wound in a vessel until it can be more securely dealt with, or to arrest the circulation in the wounded part, and prevent blood from reaching and escaping from the open vessel.

In operating, if an artery or vein is divided and bleeds, the surgeon or his assistant at once puts his finger over the vessel until it can be seized in a forceps and tied. The largest vessels can be thus compressed, and the most furious hæmorrhage at once stayed; it is only when pads and plugs are substituted for the surgeon's finger that difficulties and dangers arise. By compression of the main artery supplying a part, or circular compression of a limb firm enough to arrest all circulation in it, hæmorrhage can be prevented or temporarily arrested. For the prevention or for the temporary arrest of bleeding, pressure is the best of all the means known to the surgeon.

When used as the *sole expedient* for arresting hæmorrhage, pressure must close the mouth of a bleeding vessel until a clot has formed within it and is so firmly adherent that the force of the circulation will not detach it. The force required will, therefore, vary with the blood pressure in the wounded vessel, and also with the directness with which it can be applied. More force is needed to stop bleeding from an artery than from a vein, and a little more from a large artery than from a small one. Where the compression can be brought to bear directly upon the vessel, and this is supported by a resisting structure, such as a bone, quite moderate force only is sufficient. But in many of the cases where pressure is or may be applied, the conditions are just the reverse of this. The wounded vessel—probably an artery—is surrounded by a thick mass of soft elastic tissue, and the pressure to act efficiently through this has to be very great, and must of necessity render anæmic all the tissue between the compress and the artery; and as the compress is usually fixed in place by a circular bandage, this involves the entire arrest of the circulation in the limb.

The *dangers* incident to the use of pressure are two—the conversion of an open into a concealed hæmorrhage, and the production of gangrene. When a pad is placed over a wound from which arterial blood is flowing, and it does not entirely close the bleeding vessel, the blood continues to escape, and collects around the artery until the resistance of the tissues equals the pressure of the blood; in this way the concealed hæmorrhage has produced a traumatic aneurysm. It must also be remembered that the pressure in such a collection of blood is very great—as many times greater than the pressure with which the blood escapes from the artery as the area of the blood tumour exceeds the area of the artery opening into it. This pressure often becomes so great that it displaces the pad, and then renewed external hæmorrhage occurs. For example: in a case of hæmorrhage from the socket of an extracted tooth, pressure to be effectual must be applied to the bleeding artery by a plug passed quite to the bottom of the socket. If the plug be merely fitted over the top of the alveolus, this quickly fills with blood and becomes a miniature Bramah press, the plug is displaced, and the hæmorrhage continues.

The *gangrene* may be *local*, due to the pressure of a firm pad; where a graduated compress is used for the treatment of wound of the palmar arch, sloughing of the palm is liable to occur. But the gangrene may be *total* if a tight constricting bandage is placed around a limb so firmly as to arrest all circulation for many hours.

It follows, therefore, that *pressure should only be used as a permanent hæmostatic* in the following circumstances:—

(*a*) In cases of capillary oozing and parenchymatous hæmorrhage, where the pressure required does not imperil the vitality of the tissues: *e.g.* a cut finger or the oozing from cutaneous flaps.

(*b*) In cases of hæmorrhage from a superficial vein, where, again, only slight pressure is needed to close the vessel and resist the force of the blood: *e.g.* a burst varicose vein, or after phlebotomy.

(*c*) In cases of arterial hæmorrhage from a bony cavity, in which the pressure, if applied to the vessel itself, easily controls it, and does not act injuriously upon the bone: *e.g.* hæmorrhage from the socket of a tooth, or after removal of carious or necrosed bone.

(*d*) In cases of hæmorrhage from the walls of cysts or hollow organs, which cannot be otherwise controlled; here the compress must be applied directly to the bleeding vessel, and the whole cyst tightly filled: *e.g.* bleeding from an opened cystic bronchocele, some cases of ovarian cyst; plugging the uterus for hæmorrhage comes under this head. The end of a long strip of lint or gauze must be carried to the farthest part of the cyst to be stuffed, and then carefully paid in, so as to fill up the cavity uniformly from the bottom, every part of the cavity being carefully filled.

(*e*) In cases of deep hæmorrhage where other means, such as ligature, cannot be employed, and where the pressure can be applied directly to the bleeding vessel, and against structures firm enough to afford counter-pressure : *e.g.* deep hæmorrhage in perineal cystotomy.

Pressure should not be employed in any case of deep hæmorrhage unless it can be applied directly to the bleeding vessels, and without injury to the surrounding parts; it should never be used for deep arterial bleeding in the form of a pad applied on the surface with a thick layer of soft elastic tissue between it and the vessel to be occluded. The graduated compress is a relic of antiquity that should very rarely, if indeed ever, be used. Pressure is sometimes made over the main artery or arteries of a part as an accessory to other modes of treatment; for example : if a pad is used to control bleeding from the palm, a piece of catheter may be bandaged on over the radial and ulnar arteries in the fore-arm, to lessen the force of the circulation in the hand.

2. **Cold** acts as a hæmostatic by exciting contraction of the muscular coat of vessels. It is, therefore, of no avail in vessels which are only partly severed except as it diminishes the blood supply to the part; and it is more effectual in arterial than in venous hæmorrhage. It acts quickly. Sucking ice is used to arrest bleeding from the mouth and throat; iced injections will control many cases of epistaxis ; a stream of iced water over a wound is often efficient in bleeding from small arteries or capillaries; and an ice-bag or poultice applied to the surface will moderate bleeding in deeper parts beneath. The mere exposure of a wound to the air will sometimes suffice to arrest bleeding that had been kept up by the natural warmth of the body. It is important to remember that prolonged cold exerts a depressing influence upon the heart.

3. **Heat** acts like cold, by causing contraction of the muscular coat of a blood-vessel, and its effect is quickly produced. A temperature between 80° F. and 105° F. favours vascular engorgement, and over 180° F. it injures (burns) the tissues. As a hæmostatic, water, sponges, or cloths of a temperature between 120° F. and 150° F. are used. Heat is extremely useful in bleeding from very vascular parts, as the face, the external genitals, the uterus, and inflamed bone. It cannot be relied upon to control bleeding from veins or large arteries. Under a stream of hot water, operations upon the face and other very vascular parts become almost bloodless. Irrigation with hot boiled water is an admirable means of arresting bleeding from the uterus, abscess cavities, cysts, or cavities in bone.

4. **Styptics** are chemical agents that arrest hæmorrhage by causing contraction of arteries, coagulation of albumen, and separation of fibrin. Unless the clot they produce fills and adheres to the opening in the bleeding vessel, it is of no value; and it is for this reason that styptics so often fail, for the stream of blood washes them

away from the bleeding point. To obtain success, the wound should be carefully dried, and the styptic immediately applied to the actual bleeding-point with sufficient pressure for a few minutes to control the flow of blood. The most frequently used styptics are powdered alum, persulphate of iron, tannin, or gallic acid, the solid stick of nitrate of silver, and solutions of alum, matico, perchloride or persulphate of iron, and turpentine. They are of service chiefly in bleeding from cavities, as the mouth, nose, and uterus, where other means are difficult to apply, and in parenchymatous hæmorrhage from fungating tumours. Turpentine, acetate of lead, the astringent salts of iron, and ergot are given internally, with a view of causing contraction of arteries and "increased coagulability" of the blood.

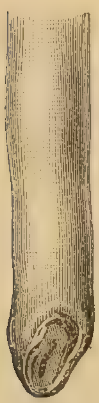

Fig. 139.—Large Artery closed by the Cautery, showing all the coats of the artery charred into an eschar over its end.

5. **The cautery** is a very powerful hæmostatic. It acts in one of two ways. In some cases, especially in very large arteries, it chars the end of the wounded vessel into a tough, solid eschar, which closes over the lumen of the artery, and on the inner surface of which the blood quickly coagulates (Fig. 139). Very often, however, a very remarkable effect is produced. At the first touch of the cautery the end of the artery is narrowed and folded up within the vessel, and with the further contact of the hot metal the end of the vessel is completely inverted, and this inversion increases rapidly until its further progress is arrested by undivided branches of the artery. This inversion is unattended with any separation of the coats, and it forms a very firm closure of the vessel: it is well shown in Fig. 140. Paquelin's cautery is the most convenient form to use. It should be at a black or dull-red heat, and be applied accurately to the bleeding point, and held in contact with it for some moments. Neglect of these precautions often leads to failure; for a bright red or a white heat is not hæmostatic, and it can easily be demonstrated outside the body that the closure of an artery by the cautery takes many seconds, the time varying with the size of the vessel. The cautery is chiefly employed in parenchymatous oozing, in hæmorrhage from sloughing surfaces in which a ligature will not hold, in some other cases where ligature is difficult or impossible, as after removal of the upper jaw, and to seal the vessels in the pedicles of tumours—especially internal piles. The eschar produced by the cautery is aseptic.

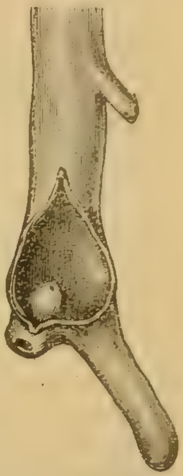

Fig. 140. — Medium-sized Artery closed by the Cautery, showing the whole artery inverted.

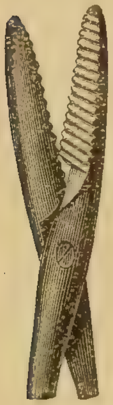

Fig. 141. — Forcipressure Forceps.

6. **Forcipressure** has superseded to a very great extent both

torsion and ligature of divided vessels. Catch forceps with strong bluntly-serrated ends are used, and with them the bleeding vessel is seized as cleanly as possible (Fig. 141). The immediate effect is the crushing of the part grasped, and the brittle inner coats of an artery break across and curl up inside it. The crushed part forms a flat band, closing over the end of the vessel and the blood clots within it, and becomes adherent to the curled-up inner coats (Fig. 142). Arteries up to the size of the ranine are easily sealed by these forceps if two precautions are taken,—the forceps must be left on for some few minutes, to allow the blood in the artery to coagulate and the clot to adhere, and they must be removed gently so as not to open out the crushed end of the vessel and displace the internal clot. Arteries of any size can be closed by the forceps if they are allowed to remain on for a few hours thus; if, in a case of nephrectomy, the renal artery cannot be tied, it should be seized in a large pressure-forceps, and held for twelve to twenty-four hours. In vaginal hysterectomy the same plan is followed if the uterine artery cannot be secured in a ligature.

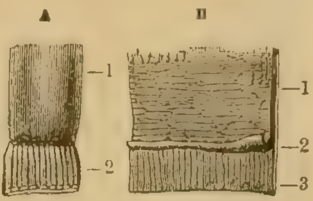

Fig. 142.—A, An Artery closed by Forcipressure.

1, Normal artery; 2, crushed extremity. B, The same artery laid open—1, Normal artery; 2, crushed and serrated outer coat; 3, inner coats separated and curled up.

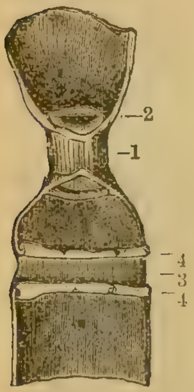

Fig. 143. — An Artery which has been crushed with Forceps in two places.

1, Compressed outer coat; 2, two inner coats, rolled up, partially occluding lumen. Below, the vessel is laid open; 3, outer coat; 4, inner and middle coats.

Pressure-forceps are used in several ways. Small bleeding arteries are seized as divided, and held until the completion of the operation, when, if the forceps are removed carefully, the hæmorrhage is found arrested. Larger arteries are seized temporarily until twisted or tied. Bands of vascular tissue are clamped in the forceps before division. Bleeding veins are occluded by these forceps, as well as arteries. In fact any vessel that can be seized in a pressure-forceps can be closed by it if the forceps is left on long enough to secure an internal clot so firmly adherent to the inner coat that it can resist the pressure of the blood.

7. **Clamps and écraseurs** may be spoken of together, as their action is similar. A clamp causes a division of the two inner coats of a vessel, on the ends of which an internal clot forms, as in an artery divided by contusion. If the action of the clamp is continued and intensified, as in hysterectomy and similar operations, the outer coat of the vessels and the other tissues are partly torn and partly ulcerated through. As a hæmostatic agent, a clamp need only be applied firmly enough to occlude the vessels, and long enough to cause a firmly adherent internal coagulum.

Écraseurs tear and crush their way through vessels, dividing first the inner coats, and then drawing out and overstretching the outer coat until it yields. If this is done quickly, the vessels bleed freely, as if cut with a knife; if slowly, the blood has time to coagulate within the vessels before they are completely severed, and the action is a tearing rather than a cutting one. Even large arteries may be thus divided without any hæmorrhage if sufficient time is taken. Écraseurs are only used where there is special difficulty in using other surgical hæmostatics; for example, in removal of the tongue or of uterine polypi.

8. **Acupressure** is now seldom used, and only as a suture as well as a hæmostatic. It is employed in this way in uniting the lips in the operation for hare-lip, etc., and may be employed in the scalp. A straight steel needle is passed under the bleeding vessel on each side of the wound, the edges of which are then held in apposition, and fixed by a figure-of-8 suture over the ends of the needle. Care must be taken not to leave in the pin too long, lest it cause ulceration, nor to apply the ligature too tight, lest it cause sloughing. If the pressure of the pin is slight, it simply occludes the vessel, without rupture of any of its coats; a clot forms within it, and the usual reparative changes ensue. If the pressure of the needle is firmer, it may rupture the inner coats in the scalp. A suture of silk or catgut is often used to unite a wound and compress a bleeding vessel in it. Suture is the best means of arresting bleeding from wounds of the liver, kidney, and spleen.

9. **Forced flexion** of the elbow or knee closes the brachial or femoral artery. It may be used as a temporary expedient to stop arterial bleeding from the hand or foot, or as an adjunct to other means. The flexion must be pushed to its extreme limit to be efficient, and this position is so inconvenient that it is rarely resorted to.

10. **Torsion** is one of the best means of arresting arterial hæmorrhage. When the cut end of an artery is seized in suitable forceps (Fig. 144), gently drawn upon and then twisted about eight half turns, its brittle inner and middle coats quickly snap across and roll up inside, whilst its tough and elastic outer coat curls up into a knob at its extremity (Fig. 145). An internal coagulum forms, and is protected against expulsion by the knob of outer coat. If the artery be insufficiently twisted, the outer coat untwists, and the vessel opens out again. If the vessel be overtwisted, the coiled-up outer coat is separated, and the clot loses its protection, and is apt to be displaced. The forceps should have broad ends and be bluntly serrated, so as to seize the whole artery and not tear it. Care should be taken to seize the artery cleanly, and in the case of a large vessel not to introduce one blade into its lumen. Torsion may be trusted to close even the largest arteries, and it has the advantages over ligature that the operator requires no assistance, that it is done very rapidly, and that it does not introduce any foreign material into the wound. Disease of the inner and middle coat of an artery is not a bar to

Fig. 144.—Torsion Forceps.

successful torsion, but extreme fragility and loss of elasticity of the outer coat is a bar, and so also is adhesion of an artery to its sheath, for it prevents the vessel from twisting freely. Veins can be closed by careful torsion as well as arteries. It has been suggested that torsion should be used for small arteries and the ligature for large vessels, and that the torsion forceps should be twisted off. Both suggestions are bad. Torsion is perhaps better adapted for large than for small arteries, because they can be seized more cleanly. Most certainly the forceps should not be twisted off, for the curled-up knob of the vessel wall is a great security, resembling in its usefulness a firmly adherent external clot.

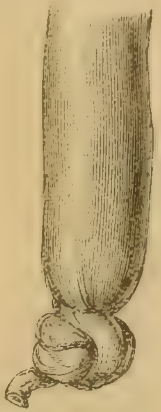

Fig. 145.—Artery closed by Torsion, showing the curled-up outer Coat of the Artery forming a twisted Knot over the end of the Artery.

The late changes occurring in a twisted artery are closely like those met with in an aseptic vessel. The thrombus is organised in the way already described, and the vessel converted into a fibrous cord (page 512). The twisted end of the vessel is quickly embedded in "lymph," and if it is not infective or infected, it is absorbed by the plasma cells, and replaced by cicatricial tissue over the end of the artery. Exactly similar changes occur in vessels closed by forcipressure and the cautery.

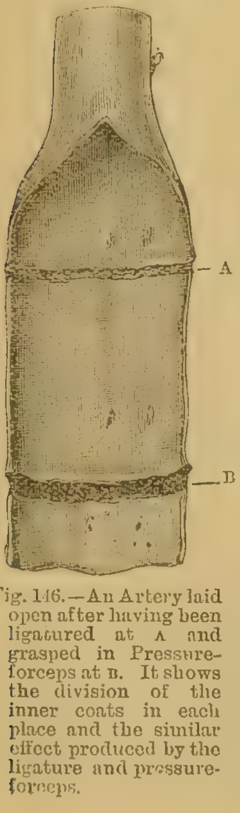

Fig. 146.—An Artery laid open after having been ligatured at A and grasped in Pressure-forceps at B. It shows the division of the inner coats in each place and the similar effect produced by the ligature and pressure-forceps.

11. **The ligature** of vessels is the most important of all hæmostatic agents. It is employed to occlude arteries in their continuity as well as where wounded. But we shall postpone to a subsequent section the discussion of the special points arising in connection with ligature of arteries in their continuity.

(*a*) *The immediate effects.*—When a ligature is tied with moderate force, the inner and middle coats of an artery are divided as if cut with a knife, and curl up within the vessel, and the portion of outer coat within the noose is strangled (Fig. 146). In the case of a divided artery tied in a wound, all the vessel beyond the noose is bloodless, and dies. If the artery be tied with still greater force, the outer coat also is cut through opposite the knot, and if it is a large artery, in all probability hæmorrhage recurs. If the ligature be tied with less force, however, the artery may be occluded without any laceration of its tissue. We shall subsequently see (page 530) that for the permanent obliteration of the artery it is neither necessary nor desirable to divide any of the coats of an artery when ligating it. In the case of a divided artery tied in a wound, the force of the blood tends not only to open it, but to push off the ligature, and the movement of the part, the twitchings of muscles, the adaptation of flaps, etc., may have the same tendency. To guard against this, it is customary to tie with such an amount of force as will certainly sever the two inner coats; but it should be clearly appreciated that this is merely to get a sufficiently firm hold upon the vessel—to prevent the ligature from being pushed or dragged off—and not at all for any advantage accruing from division of these coats. Happily, the disadvantage of the partial division of the vessel is reduced to a minimum in cases of amputation or of the removal of parts, for the blood pressure in the ligated arteries falls, as a result of the removal of the parts supplied by them.

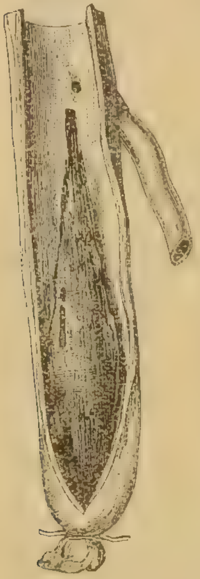
Fig. 147.—Ligatured Artery laid open, showing the internal Clot adherent to the Artery below, free and tapering above where it reaches up to a branch.

The closure of the artery at once stops all bleeding from it, and quickly the blood within it coagulates upon the ends of the divided coats. This clot gradually extends, until, in about three days, it fills the vessel, more or less exactly, as far as the lateral branch nearest to the ligature. It is conical in shape, and adheres first and most firmly to the divided vessel wall (Fig. 147); when the wound and the ligature are aseptic, the clot may be very limited in extent, and not reach so far as the nearest branch. It may, later, disintegrate and disappear.

(*b*) *The repair of a ligatured artery.*—When an artery has been divided and its ends tied, its repair is modified in two or three important ways. In the first place, its inner coats are severed by the ligature, and the end of the vessel is closed in by the thin outer coat only, surrounded and supported by the ligature. This wound in the coats forms a large surface for the attachment of the internal clot, and the leucocytes and plasma cells enter the clot freely and easily at the same spot. In the next place, the constricted ring of the outer coat and the end of the vessel beyond it die, as the result of their total anæmia, and are absorbed by the myriads of leucocytes which are poured out around them, and quickly pass into them. Thirdly, the ligature itself is either absorbed, encapsuled, or cast off. In an aseptic wound, a readily absorbable ligature, such as catgut, is rapidly absorbed; a more resistant structure, such as silk or unspun silk, may be slowly absorbed, or it may lie at the side of the vessel encapsuled in firm cicatricial tissue. In a septic wound, catgut may be quickly liquefied, and only the knot come away in the discharge; but sometimes the ligature comes away entire, having "ulcerated" through the tied end of the artery. Silk, tendon, and wire ligatures are always thus separated in septic wounds.

The ligature of a divided artery, therefore, complicates the process of repair by cutting across the two inner coats of the vessel, and to that extent weakening it, by necessitating the absorption or separation of the end of the vessel and of the ligature, and by introducing a fresh source of possible wound infection. The repair of a tied artery takes a little longer time to perfect, and is attended by the effusion of a greater amount of "lymph" than that of a vessel closed by natural processes alone.

In the case of gold or platinum wire ligature, no absorption whatever takes place, but in all other ligature materials, absorption, more or less rapid and complete, occurs if asepsis is preserved. In the case of carbolised catgut the absorption is rapid; chromic acid gut is more resistant; silk, silkworm gut and tendons are still more slowly absorbed. The unabsorbed parts are surrounded by cicatricial tissue, which also replaces the ligature as it is absorbed.

(*c*) *The results of sepsis.*—A divided artery tied in a wound may be infected through the ligature, or by other infective matter introduced into the wound at the time of the injury or later, or, rarely, through the general blood-stream. The results of such infection are a septic arteritis and periarteritis; the strangled portion of the artery is not absorbed, but is separated from the living artery by a process of ulceration. This sets free the ligature, which is neither absorbed nor encapsuled, but comes away with the discharge. There is, therefore, a destructive ulcerating process going on at the end of the ligatured vessel. If this be so intense as to arrest all repair, or if it so impair the artery that it cannot resist the pressure of the blood, secondary hæmorrhage occurs. Previous to the introduction of antiseptic surgery and aseptic ligatures, secondary hæmorrhage, about the time of the separation of the ligatures—one to three weeks after application—was very common; now it is a rare event.

Ligature materials.—Very many materials have been used as ligatures, such as silk or hempen threads, and gold, silver, iron, lead, or platinum wire, untanned skin, catgut, unspun silk (silkworm gut), tendon, nerve, ox-aorta, and peritoneum. A good ligature must be strong, inelastic, pliable, smooth, round, aseptic, and slowly absorbable. Wire is no longer used; it is inconvenient to apply, and not absorbable. Silkworm gut is also rejected, because deficient in pliability, although its smooth surface without pores renders it easy to make aseptic, and it is also very strong and slowly absorbed. Strips of untanned skin and of nerve are no longer used. To-day the choice really lies between catgut, silk, tendon, artery, and peritoneum.

Catgut.—Commercial catgut consists of the intestine of the sheep from which the mucous and muscular coats have been more or less perfectly removed by scraping. Owing to imperfection in its manufacture, much of the muscular and mucous coats is left behind, and the villi can often be detected in microscopical sections. Hence it is an infected material; cocci, bacteria, and their spores have been demonstrated in it, and it is a possible source of wound-infection—some say a frequent source. Volkmann has recorded a case in which a catgut ligature conveyed the infection of anthrax. In its dry state it is too rigid to knot securely, while if soaked in warm water or the serum of a wound it swells up, becomes soft, pulpy, and useless For surgical purposes, therefore, it must be specially prepared with a view of making it first aseptic, and then sufficiently supple to tie easily and securely without, at the same time, becoming too readily absorbable.

The following methods of preparing catgut for surgeons' use have been recommended :—

(1) Lister's original method was to soak loose hanks of catgut for at least two months—the longer the better—in an emulsion of one part of crystallised carbolic acid deliquesced by water, in five parts of olive oil. The catgut thus prepared may be kept in the same emulsion, from which the water gradually separates, or in simple carbolic oil 20 per cent., or in corrosive sublimate and glycerine 1 in 500.

(2) Catgut wound on a reel of some kind is soaked for forty-eight hours in a 5 per cent. watery solution of pure carbolic acid, to which chromic acid is added in the proportion of 1 in 4,000; the weight of catgut must equal that of the carbolic acid in the solution. The catgut may be kept dried, and must then be soaked in carbolic solution 5 per cent. for half-an-hour before being used; or after it has been carefully dried, it may be kept in carbolic oil 20 per cent. or corrosive sublimate and glycerine 1 in 1,000.

(3) A third plan is as follows:—Five parts of catgut on the stretch is immersed for twelve hours in a solution of 1 part of chromic acid in 100 parts of distilled water. It is then partly dried by wiping it with a clean cloth, and placed for twelve hours in 100 parts of sulphurous acid. The catgut is now dried and kept dry. Before being used, it is placed for fifteen minutes in carbolic acid solution 5 per cent. This "sulpho-chromic catgut" is also called "green catgut," from its colour.

(4) Catgut may be sterilised by exposure for two hours to a dry heat of 284° F., and then preserved in carbolic oil or sublimate glycerine.

It has been shown that much of the so-called "antiseptic catgut" that is supplied to surgeons is not aseptic, and the difficulty of obtaining a certainly aseptic catgut has led many surgeons to agree with Kocher in discarding it altogether. When prepared by the first of the methods above described, the catgut is liable to soften prematurely and slip, and allow the vessel to become patent.

Catgut in an aseptic wound is gradually absorbed by the leucocytes, which invade it from the outside and pass in between its constituent fibres. In a septic wound the ligature may be thrown off unaltered, or it may be rapidly softened by the peptic action of the micrococci. Catgut is largely used as a ligature for vessels divided in a wound; it should not be used for ligaturing vessels in their continuity. Its faulty preparation and the great difficulty in making it at once certainly aseptic, sufficiently supple, and neither too readily nor too slowly absorbable, render catgut inferior to other materials.

Silk is used in the form of floss silk or Chinese twist. To make the pure white silk used by surgeons, the raw material is first boiled to remove the gum, then washed with soap, then placed in sulphur kilns for several hours, and finally bleached. The boiling, sulphuring, and bleaching are all antiseptic in their action, and silk is, therefore, less likely to be infective when supplied to the surgeon than is catgut. It is rendered aseptic by prolonged boiling in water, carbolic lotion, or mercuric chloride solution, and it is kept in carbolic lotion 5 per cent. or in sublimate glycerine. It can be obtained of any required size; some prefer the softer floss silk, others the stronger twist. In an aseptic wound it becomes encapsuled, and then slowly absorbed, the leucocytes and plasma cells wandering in between the fibres of the silk. Silk is a thoroughly reliable material for ligature, and its special preparation can easily be conducted by every surgeon. This is a great safeguard and advantage.

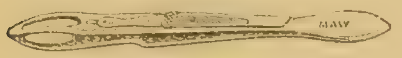

Fig. 148.—Fenestrated Artery Forceps for Ligature.

Tendons, arteries, and peritoneum.—The long tail tendons of the kangaroo, dried, and then soaked in carbolic or mercuric solution for forty-eight hours before use, make excellent ligatures. By splitting, they can be obtained of any required degree of fineness; they are strong, supple, and absorbable—but not too readily. The knot is bulky, and will slip if not tied very firmly.

The middle coat of the aorta of the ox can be cut in long spiral bands of any desired breadth, and dried under extension. It makes a strong, flat, absorbable ligature, which knots well, and is easily made aseptic by soaking in carbolic solution 5 per cent. for some hours. Mr. Barwell has had great success in its use.

Peritoneum, cut into strips and dried, is recommended, because it consists of pure white fibrous tissue, like the outer coat of an artery. It is rendered aseptic by soaking in carbolic solution. It is said to be an excellent ligature, but has not come into general use.

To apply a ligature to a divided vessel, it should be seized in a forceps (Fig. 148) and drawn gently out, care being taken to seize the vessel cleanly without muscle, fascia, nerve, or other structure. The ligature is tied in a reef-knot (Fig. 149) or a surgeon's knot (Fig. 150), the fingers being pressed well down on the vessel; it is tied so tight as to rupture the inner coats in order to give it a secure hold upon the vessel, and both ends are cut off short.

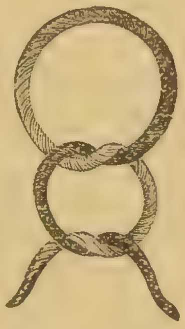

Fig. 149—Reef-knot.

12. **Complete division** of a partially-severed artery by allowing of its retraction and contraction will sometimes at once arrest a hæmorrhage. This means is not of wide application, but it is of use

in some cases of hæmorrhage from the *frenum præputii* and from the scalp. The point of a lancet drawn across the bottom of the bleeding vessel is the best means to employ.

III. THE EFFECTS OF LOSS OF BLOOD.

The quantity lost.—Uncontrolled hæmorrhage, however produced and wherever occurring, is inevitably fatal; but the quantity of blood that may be lost without sacrifice of life varies very much according to the age, sex, and constitutional state of the individual, and to the nature and rapidity of the hæmorrhage. Infants and young children bear any loss of blood badly, a relatively small loss being either fatal or attended with profound syncope; but the resulting anæmia is quickly repaired. After puberty and up to middle life hæmorrhage is borne well, and its effects are most quickly recovered from. With advancing age, loss of blood becomes increasingly serious and more permanent in its effects. Some have thought that women bear the loss of blood better than men. It is certain that a hæmorrhage—say, the loss of a pint of blood—will cause much more marked effects in one individual than in another of the same age, sex, and general condition, and a constitutional peculiarity is assumed to account for the difference.

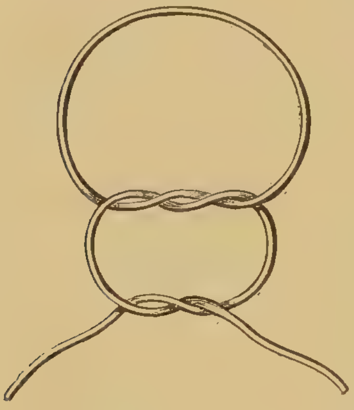

Fig. 150.—Surgeon's Knot.

Effects.—The loss of a given amount of arterial blood produces a greater effect than the same amount escaping from a vein. This may be in part due to the greater rapidity of the flow from the artery, and in part to their relative oxygenation and carbonisation. The local anæmia and the loss of the force which affects the venous return from the part may also have an adverse influence.

Very striking is the difference due to the rapidity of the hæmorrhage. When an artery of the first magnitude is freely opened, the blood in the general arterial system flows out through the wound, as it is the direction of least resistance; and the venous circulation is at the same time arrested, owing to the loss of the *vis a tergo*. There is, therefore, sudden fatal syncope, and the corpse has a leaden hue from the blood in the more or less well-filled veins.

When an artery of less than the first magnitude or a large vein is wounded, and continuous hæmorrhage, ending in death, occurs, the first effect produced is a lessening of the arterial tension and an increase in the force and frequency of the heart's action, which, however, fails to compensate for the progressive emptying of the vessels. As a result, all the tissues of the body, including the muscular substance of the heart, become acutely anæmic, and their special functions impaired and eventually destroyed. This result is also partly brought about by the want of oxygen in the tissues. A certain amount of watery fluid is absorbed from the tissues into the blood-vessels, as an attempt to keep the vessels full enough to carry on the circulation. The symptoms that the patient exhibits in these circumstances are as follows :—The pulse becomes increasingly rapid, smaller, and weaker, until it cannot be felt in the smaller arteries, such as the radial, and is only a feeble flicker in the common femoral and common carotid. The mucous membranes are blanched, and the skin becomes white, cold, and bathed in sweat. The patient experiences a sense of nausea and faintness, palpitation and dyspnœa, noises in the ears and deafness, dimness of vision, increasing to a sense of darkness with occasional flashes of light, and then he becomes unconscious. The breathing is hurried, gasping, with a distressing sense of "want of breath," until, when syncope comes on, the respiratory movements are very shallow and only diaphragmatic. There is great muscular weakness, the voice is very feeble, the limbs are flaccid; and in the late stages great restlessness, with throwing about of the arms and legs, becomes a prominent and alarming symptom. Convulsions occasionally occur. After death the body is shrunken, with a peculiarly white and wax-like appearance, and all the organs are pale and bloodless.

When the hæmorrhage is *recurrent* instead of continuous, a much larger total loss occurs before life is extinguished. Serum is absorbed from all the soft tissues of the body, which become thereby very shrunken. The fluid in the arterioles is so dilute that it filters through them, and a certain amount of œdema of the eyelids and extremities occurs; and the "blood" that escapes in the final hæmorrhage looks thin and watery: more like coloured serum than blood. In such cases the patient faints with each return of the hæmorrhage, and the pulse becomes very feeble and rapid, with a soft artery which feels empty between the successive pulse waves. The temperature falls two degrees or more below the normal, and the patient is so weak that he is liable to faint or die if raised in bed, or if his arms are allowed suddenly to drop over the bed-side. There is great thirst, but absorption of fluid from the stomach is slow, owing to the very watery condition of the blood.

The early period of recovery from a severe hæmorrhage is often marked by a condition called *hæmorrhagic fever*. The temperature is raised, the pulse is frequent and soft, with a large wave, but an empty artery between the cardiac systoles. The patient is very weak and thirsty, the urine is scanty, the bowels are confined, and complaint is made of a sense of throbbing in the temples. For some time after a severe loss of blood there is marked anæmia, which may be permanent, and render the patient an easy prey to infective diseases; or the anæmia may be slowly recovered from; after middle life the recovery is rarely complete.

IV. THE CONSTITUTIONAL TREATMENT OF HÆMORRHAGE.

In a case of severe hæmorrhage constitutional treatment becomes of great importance, and it is directed to three special ends: (1) the prevention of a recurrence of the bleeding; (2) the compensation, as far as possible, of the immediate effects of the hæmorrhage; and (3) the speedy repair of the loss of blood. In every case of hæmorrhage care should be taken to avoid everything liable to renew the bleeding; but it is only when the loss of blood has been serious that it is important to try to compensate for its immediate effects, and to secure its speedy repair. What follows applies to cases in which a serious loss of blood has occurred.

Indications.—(1) The first great indication is to secure for the patient the most **complete rest,** in the horizontal position if possible. This protects injured vessels from the local effects of movement, which may displace ligatures and coagula, but it is especially valuable as quieting the heart's action, lessening arterial tension, and diminishing nervous, muscular, and cardiac exhaustion. The quiet and the lessened heart's action produced by the "shock" of every severe injury and operation are most beneficent in their influence in arresting and preventing the recurrence of hæmorrhage, and care should be taken not to hurry on "reaction," lest it lead to renewed bleeding. After a great loss of blood, restlessness is often a marked effect, but it must be checked as far as possible by gentle, quiet restraint.

(2) The next indication is to **maintain the temperature** of the patient by covering him with warm blankets, and by placing hot bottles to the feet, between the thighs, and on each side of the trunk. The clothing must not be heavy nor tucked in tightly, especially over the chest and abdomen, lest the respiratory movements be in any way hindered. After severe hæmorrhage there is great difficulty in supplying the tissues with sufficient oxygen, owing to the loss of the oxygen-carrying red corpuscles; the movements of respiration, therefore, must not be in any way impeded by clothing or pressure, especially as the respiratory muscles, in common with others, are weakened by the loss of blood. For the same reason, the apartment in which the patient lies should be thoroughly ventilated, no unnecessary screens or other hindrances to the free movement of air should be tolerated, and as few people and lights as possible should be allowed in the room.

(3) The third great point to attend to is the **prevention of syncope,** by favouring the blood-supply to the brain. The patient must be kept flat on his back, and all attempts to raise the head must be checked; in very bad cases the head may be allowed to be a little lower than the chest, and in extreme cases the limbs may be firmly bandaged and raised, to drive the blood out of them, and secure a more liberal supply to the brain.

(4) **The food** of these patients should be warm fluid, given in small quantities frequently; hot water, hot water and milk in equal parts, and beef-tea are the most suitable. As recovery progresses the diet must be improved, farinaceous foods, white fish, chicken, and meat being gradually added. Constipation will probably be troublesome, and is best combated by a daily enema, or by a pill containing iron, aloes, and nux vomica. If sleeplessness and restlessness are marked features, bromidia in ʒss doses two or three times a day will be found serviceable.

(5) For the **resulting anæmia,** the main indications are prolonged rest, a carefully-regulated, light, nutritious diet, residence at the seaside or in moorland air, with as much sunshine as possible, thorough ventilation of the rooms both by night and by day, and such tonics as the milder preparations of iron, arsenic, cod-liver oil, quinine, and nux vomica. A sea voyage is often beneficial.

When, in spite of the arrest of the bleeding and the treatment above described, the patient gets worse, his pulse weaker, and the syncope more marked, *stimulants* may be given. Subcutaneous injection of ether (♏xx to ♏xxx for a dose) is the best, because the most rapid in its action, and it may be repeated at short intervals. Other good means are a sinapism over the heart, brandy and hot water by mouth, by enema, or rubbed on the tongue and lips. An ounce of brandy or other spirit in two or three ounces of hot water may be thrown into the bowel, and is more efficacious than pure spirit; for the heat is a stimulant, and the water may be absorbed and add to the fulness of the vessels. Where hæmorrhage is still continuing, stimulants can only do harm by increasing the rapidity of the flow. Where open vessels are closed by blood-clot only—not a ligature, suture, or by torsion—stimulants may excite fresh bleeding by displacing the clots. Their use, therefore, requires great care and discrimination. In cases where death is threatened from extreme loss of blood, transfusion should be practised.

Transfusion and saline injection.—Where life is endangered by a severe hæmorrhage, the most valuable means of averting death is the injection of suitable fluid into a vein. At first it was thought necessary to inject blood; then it was shown that defibrinated blood was as good; and lately a saline solution has been shown to be as effective in its immediate results as blood, and far safer and simpler to use. A certain degree of arterial tension is essential to the carrying on of the circulation, and when the vessels have been emptied beyond a given point, the heart's action is entirely ineffective; any innocuous fluid which will then restore the adequate arterial tension enables the heart to act and the circulation to proceed.

When pure blood is used—**immediate transfusion**—great care must be taken not to introduce any coagulum, which would form an embolism, and might cause instant death. Roussel's apparatus is very ingenious, and successful in the inventor's hands, but it requires considerable practice to use it properly. Aveling's apparatus is the simplest and the best (Fig. 151). It consists of a miniature Higginson's syringe without valves, and fitted with a proper silver cannula at each end of the rubber tube. The whole is immersed in and filled with warm saline solution, and then the cannulæ are passed into the prepared veins in the "giver" and "receiver" of the blood. By closing the "giver" tube with the finger and thumb, and gently compressing the ball, it is emptied into the "receiver's" vein; if now the "receiver" tube is compressed, and the ball allowed to expand, it fills from the "receiver's" vein. By repeating this manœuvre, the desired amount of blood can be passed from the "giver" to the "receiver." If any difficulty is experienced in filling or emptying the syringe, it shows that clotting is occurring, and the transfusion must be at once stopped.

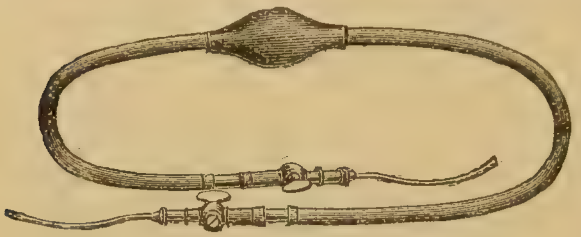

Fig. 151.—Aveling's Transfusion Apparatus.

When defibrinated blood is used—**mediate transfusion**—the simplest plan is to draw the blood into a wide-mouthed enamelled receptacle standing in a vessel of water at a temperature of 105°. After thoroughly "whipping" it in this vessel, the blood is strained through fine muslin into a similar jar, also standing in water at 105°. This blood is then passed into the receiver's vein by means of a silver cannula, with a tube and funnel attached, or a syringe, such as that of an aspirator, or an ordinary glass syringe.

Since such excellent results have been obtained with **saline injections,** both forms of transfusion have been abandoned. The much greater simplicity of this plan, its freedom from the risk of embolism, and the fact that it entails no sacrifice of blood by another, all commend it as superior to transfusion. The fluid used may be either boiled water containing a teaspoonful of common salt in the pint, or a solution containing 50 grs. of chloride of sodium, 3 grs. of chloride of potassium, 25 grs. each of sulphate of soda and carbonate of soda, and 2 grs. of phosphate of soda, in a pint of boiled water. The solution should be injected at a temperature of 100° F., and at least as much as 30 fl. oz. should be introduced; in some cases several pints have been injected. The injection should be made steadily, with little force and without the admission of air. A vein exposed in a wound may be chosen, or one of the subcutaneous veins at the elbow or the dorsum of the foot may be opened. Into the open vein a glass vulcanite or silver cannula is passed, to which a short length of rubber tube is attached, and the solution is injected by means of a glass syringe with a nozzle fitting the tube. Or a glass funnel and two feet of rubber tube may be attached to the cannula, and the solution poured into the funnel, raised eighteen inches above the patient. The syringe is, perhaps, the safer instrument, as there is less chance of an admixture of air bubbles than when the fluid is poured into a funnel.

Whatever plan is employed, all the apparatus must be previously thoroughly cleansed with a suitable antiseptic, and then flushed with boiled water at 100° F. The injection may be followed by a sharp, but short, rise of temperature.

Injection of fluid into the vessels should only be employed when the loss of blood has been stopped, and it is particularly indicated in cases of hæmorrhage pure and simple, as from a ruptured varicose vein, a punctured wound of an artery, etc. Mr. Mayo Robson has shown, however, that it is a very valuable means of combating the profound shock which is liable to occur after certain prolonged abdominal operations.

The ligature of an artery in its continuity.—When an artery is tied in its continuity, the circulation through it is suddenly arrested, and certain changes occur in the walls of the vessel itself. We will first notice the changes that occur in the artery, and then the effects of a sudden block in the arterial circulation of a part.

(*a*) **Changes in the artery.**—The effects of the ligature vary with the degree of tightness with which it is tied. The coats of the artery may be merely thrown into several longitudinal folds which occlude the lumen. If more force is used, limited lacerations of the inner and middle coats are produced. If the ligature is drawn tight the inner and middle coats are entirely severed, and the portion of the outer coat encircled by the thread is compressed and anæmic. Opposite the knot even the tough outer coat of the artery may be divided. These injuries of tightly tied arteries are well shown in Fig. 152, taken from Ballance and Edmunds' valuable monograph on "Ligation in Continuity." (I am greatly indebted to the authors of this work for much valuable matter which I have incorporated in this Article and in Article XXV., and also for the permission, most readily granted, to use several of their original illustrations.) The blood coagulates upon the divided arterial coats, and the thrombus gradually extends on each side of the ligature, in some cases as far as the next branch of the artery. As a rule, the clot is slower in formation and smaller in size on the distal side of the ligature. As the result of the injury, the vessels in the sheath of the artery and in the adjacent arterial wall itself dilate, and serous fluid exudes into the sheath and artery, and leucocytes migrate from the dilated vessels, and pass in numbers into the arterial walls and into the clot. Stimulated by this excess of pabulum, the fixed cells of the part—sheath and artery, including the endothelium—proliferate, and plasma cells thus arising, infiltrate the arterial wall and the clot within it. The clot gradually disintegrates and disappears, the strangled zone of outer coat is also absorbed, and the occluded artery is converted into a vascular fibrocellular cord; the organisation of the clot and its vascularisation occurring in the way that has been previously described (page 520). The ligature is either absorbed, encysted, or thrown off. (*See* page 521.)

By this process, then, the artery is divided, and its ends are sealed over by a cicatrix. Experience shows that even with aseptic ligatures and aseptic wounds this process is attended with a certain risk of secondary hæmorrhage; this becomes a very serious danger in those arteries where the branches are large, and so placed that only a short length of artery intervenes between them, and can be thus obliterated. In the physiological occlusion of arteries, such as occurs in the ductus arteriosus, and in pathological occlusion from the pressure of tumours or of contracting cicatrices, hæmorrhage never occurs; it is therefore evident that this danger is introduced by the method employed by surgeons. A study of the *physiological process* shows that it differs from the surgical mainly in this: that no injury is inflicted upon the arterial wall; none of its tissues are divided or strangled, but the endothelium undergoes great proliferation, this excites coagulation of the blood in the vessel, and the thrombus thus formed is replaced by connective tissue with certainty and safety.

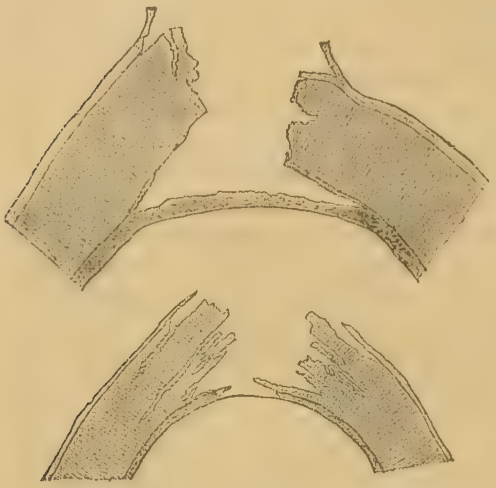

Fig. 152.—Drawings of longitudinal Sections of Human Arteries after Ligature with Kangaroo Tendon. The upper artery is the superficial femoral artery; the lower is the third part of the subclavian artery, in this case all the coats of the artery are severed and the thin sheath only remains. (Ballance and Edmunds.)

It has been shown that a ligature can be tied so as to occlude an artery without severing any of its coats, and that when it is thus tied a thrombus forms within it, and is replaced by a connective tissue scar in the artery. It is urged in favour of this mode of ligature that it is unattended with the risk of secondary hæmorrhage inherent in any method which divides the coats of the artery, and that it is capable of safe application in situations where the older form of ligature has been found to be inapplicable—*e.g.* the innominate artery. Against this form of ligature is urged the difficulty of applying it with just the amount of force required to occlude the vessel, and yet not to lacerate its wall; if not tied tight enough, the vessel may become pervious again, and the same

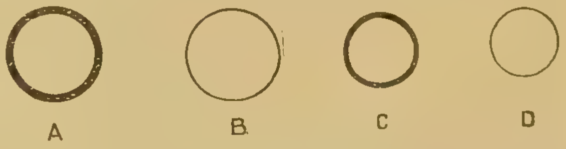

Fig. 153.—A, Transverse Section of first part of Subclavian when collapsed; B, the same under a Pressure of 240 mm. of Hg; C, Transverse Section of collapsed superficial Femoral Artery; D, the same Artery under the Pressure of 240 mm. Hg. (Ballance and Edmunds.)

result may follow too rapid absorption of the ligature. While admitting the justice of these objections, they do not appear to outweigh the important advantage of safety against hæmorrhage, and the balance of evidence is, therefore, decidedly in favour of applying ligatures to arteries in continuity without division of their inner coats. Although this method should be used in all cases, it is of especial value and importance where the ligature has to be applied near a large branch or to a diseased artery. This form of ligature, no doubt, requires greater care than the other method; but this is no argument against its employment. The ligature should be one of the less absorbable forms—silk or tendon—and, of course, it should be carefully prepared to render it aseptic, and every pains taken to secure primary aseptic healing of the wound.

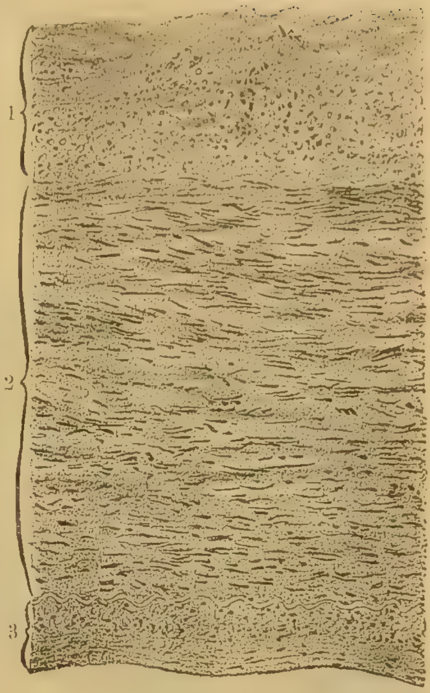

Fig. 154.—Transverse Section of superficial Femoral Artery (× 100), showing the thickness of the three coats. (Ballance and Edmunds.)

1, Outer; 2, middle; and 3. inner coat. The outer coat is much thicker than in other large arteries.

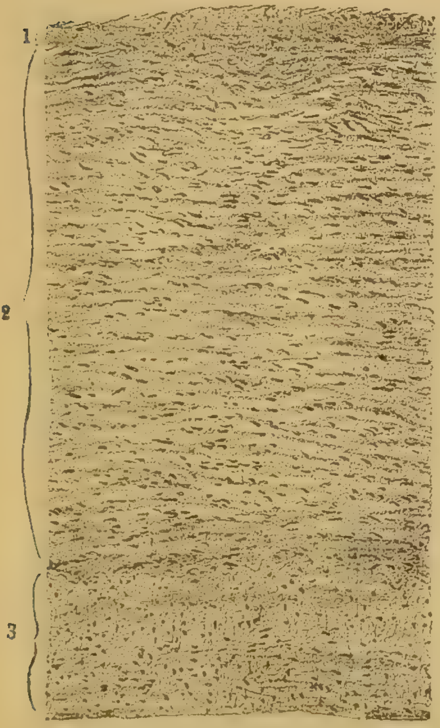

Fig. 155.—Transverse Section of the Common Carotid Artery (× 190), showing the thickness of the three coats. (Ballance and Edmunds.)

1, Outer; 2, middle and 3, inner coat.

It must be remembered that the arterial walls are much thinner during life when exposed to the normal blood pressure than they are when empty and collapsed after death. The wavy outline of the elastic membrane of Henle is entirely due to this shrinking of the empty vessel, and is lost when the artery is distended by the blood. Ballance and Edmunds found that the thickness of the common carotid artery of man, when collapsed, is ·78 mm., but when distended with a pressure of 240 mm. of mercury it is reduced to ·29 mm. The thickness of the superficial femoral artery is reduced in the same way from ·72 mm. to ·51 mm. (Fig. 153). These differences represent the extremes met with in large arteries. It is very important to remember the tenuity of the walls of living functional arteries. Still more noteworthy is the extreme thinness of the outer coat of large arteries. Thus in the innominate artery the outer coat is only about one-fourteenth of the whole, in the common carotid artery one-twenty-fifth, and in the superficial femoral artery a little more than a quarter of the entire arterial wall. For the smaller arteries the proportion is considerably modified, but in the larger vessels, which are most often the seat of ligature in their continuity, if the ligature severs the inner and middle coats it leaves only a very thin layer of tissue to withstand the force of the circulation (Figs. 152, 154, 155).

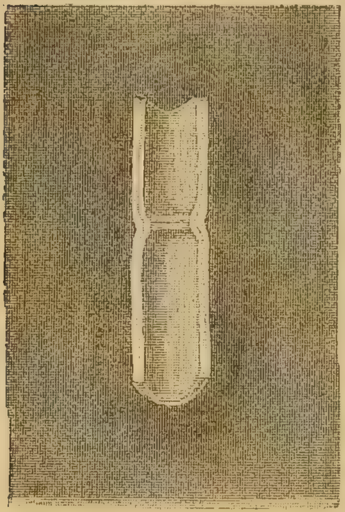
Fig. 156.—Naked-eye Appearance of a Human Carotid Artery 108 Days after Ligature with stout Catgut. The artery is patent but its lumen is only 3 mm. in diameter, being contracted by a diaphragm.

If, from imperfect tying of the ligature or its too rapid softening, the lumen of the artery is not occluded, a ring of fibrous tissue, or even a complete diaphragm, may form in the interior of the vessel without alteration in the vessel above and below. These "diaphragms" are caused by proliferation of the cells of the intima, as a result of the compression of the ligature, and the subsequent development of this cushion of thickened intima into fibrous tissue. This effect may be produced when the lumen of the artery has been merely narrowed, or temporarily occluded, or temporarily occluded with rupture more or less complete of its inner coats. (*See* Fig. 156.) When an artery is tied in its continuity, the pressure of the blood within it tends to loosen the first knot while the second is being made, and this in the case of the surgical knot as well as the reef knot. For this reason Ballance and Edmunds have recommended the "stay knot" (Fig. 157), which, they say, can be applied without any risk of this accident. The "stay knot" is made by using two strands of ligature material, making a simple knot with each singly, and then tying together each pair of ends. If the reef or surgical knot be used, the artery above should be compressed while it is being tied, so that the knot may not be exposed to the pressure of the blood until it is completed and secure against slipping.

This liability for an artery which has been tied to become pervious again is of great practical importance. It may render the operation a failure in its effects, as when an artery is tied for the cure of aneurysm. It also affords a very strong argument against so tying an artery as to rupture its inner coats, and leave only the thin and weak outer coat to withstand the full force of the arterial blood pressure, with the danger of its yielding and forming an aneurysm, or causing secondary hæmorrhage.

There are a few cases recorded where the ligature of an artery in its continuity has been followed by the development of an aneurysm: as a result, no doubt, of the weakening of the vessel by division of its inner tunics, and failure of the ligature to occlude the vessel permanently. It appears that the condition of asepsis tends to lessen the length of clot formed in a tied artery. In this indirect mode it may increase the liability to repatency of the vessel.

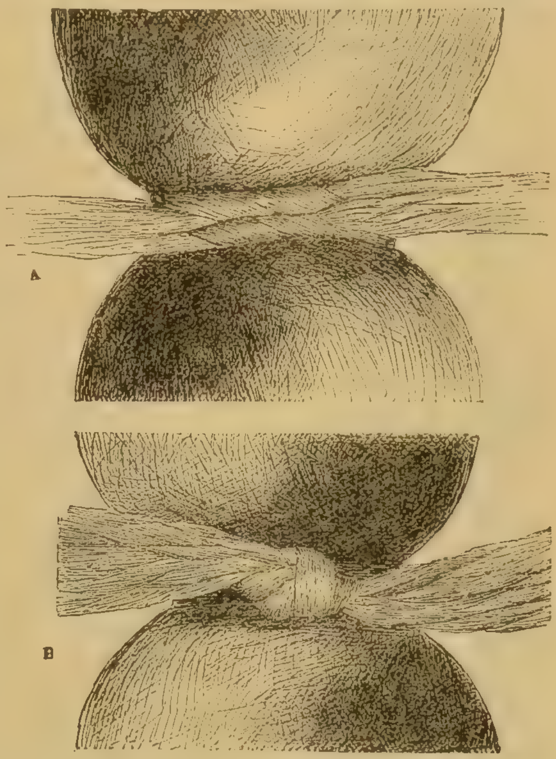

Fig. 157.—The first (A) and second (B) Stages of the Stay knot. (Ballance and Edmunds.)

(*b*) **Effects on the circulation.** — When from any cause, ligature, embolism, thrombosis, or the pressure of a tumour or cicatrix, an arterial trunk is obliterated, the parts supplied with blood by it are blanched, and the branches arising below the obstruction are pulseless. The part quickly becomes cold and benumbed, and if, from the extent or position of the arterial obstruction, or from any other cause, the anastomotic circulation is not established, the part dies, undergoing the changes described as "dry" gangrene. More usually blood is carried into the empty vessels by the numerous anastomoses that exist almost universally in the body. At first all the communicating vessels dilate and participate in this, for the resistance they offer to the passage of blood is less than that offered by the capillaries, and the blood flows from the full arteries in the direction of least resistance. Certain special vessels gradually enlarge, undergoing a true hypertrophy, and serve as the main channels of the blood, while the others contract to their normal size. (*See* Fig. 158.) The pulseless arteries are felt to beat again, and sometimes the enlarged anastomosing vessels can be felt beating under the finger. The temporary deprivation of blood causes the vessels beyond the ligature to yield to the pressure of the blood first brought to them through the anastomosing channels, and thus the pallor and coldness are followed by increased redness and heat in the part, which gradually subside as the tissues recover their full vitality. A similar effect is seen after the removal of an Esmarch's bandage: the pallid anæmic tissues blush, and then gradually resume their natural appearance. In some cases the sudden anæmia is attended with severe pain in the part, and in the case of an embolus the sudden acute pain may be associated with tenderness of the empty arteries. Finally, the part is generally a little shrunken and cold, owing to the blood supply being below the normal.

The treatment of such a part requires care. It should be placed in the position most favourable to the circulation. As a rule, this is slightly raised, with the joints a little flexed. It should be swathed in cotton wool, kept in place by a loosely-applied flannel bandage, and outside this a hot bottle should be placed. Great care must be taken not to have any tight bandage, splint, or other application, nor any hot bottle next the surface, nor exposure of the part to cold. Any such injury may lead to gangrene from interference with the circulation, or from its direct lethal influence upon the anæmic, almost lifeless, tissues. In the later stages gentle massage promotes the circulation in the part.

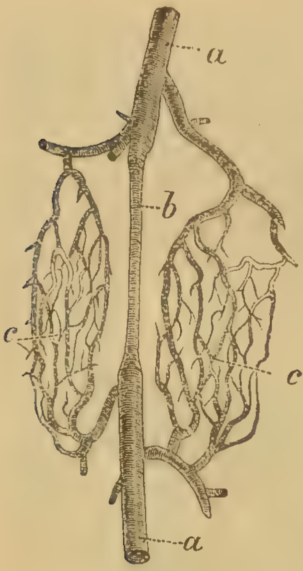

Fig. 158.—Showing Effects of Ligature of Femoral Artery. (After Porta.)

a, Femoral artery of a dog; *b*, obliterated portion of artery; *c*, *c*, anastomosing vessels in sartorius and pectineus.

Gangrene after ligature or wound of an artery.

Causes.—One of the dangers of the ligature of a main artery in its continuity or of its division in a wound is gangrene. It may be of the "dry" or the "moist" variety, the tissues in the one case dying from the insufficient supply of blood, and in the other from failure of the venous circulation. The causes of "*dry gangrene*" after ligature or sudden occlusion of an artery are disease or degeneration of the heart weakening the force of the arterial circulation, great hæmorrhage lessening the blood pressure, extensive disease of the arteries—either atheroma or calcification—preventing their enlargement for the anastomotic circulation, the obstruction of a considerable length of an artery, or the formation of a thrombus in a diseased artery when the feeble stream of blood first reaches it through the anastomosing vessels. In certain situations there is special difficulty in enlarging the anastomosing vessels, as when they run, like the vertebral and internal carotid, through bony channels; tight bandaging would have the same effect. Dry gangrene most often occurs, therefore, in the aged, or after a profuse hæmorrhage. Horsley has shown that when the common or internal carotid is ligatured the communication between the two anterior cerebral arteries is free enough to maintain the vitality of the front part of the brain, but that there is a great danger of fatal anæmia of the parts supplied by the middle cerebral artery, as the posterior communicating artery is unable to carry sufficient blood into it. Hemiplegia is a frequent result of ligature of the common carotid artery, and after death an abscess is frequently found in the parts that have been rendered anæmic, the bloodless tissues offering no resistance to the development of pyogenic organisms which have reached them through the feeble stream of blood with which they are supplied.

Moist gangrene is more often met with after ligature of an artery than is the dry form; it occurs when venous obstruction is superadded to the block in the artery. This may be due to wound or ligature of the companion vein; or to a thrombus forming in it, as the result of contusion or compression; or to pressure on the vein or veins by effused blood or a tumour—especially an aneurysm. In these cases the force of the heart is expended, first, in propelling the blood through the anastomotic channels, and then in overcoming the resistance of the capillaries; and there is no force remaining to propel the blood through the veins if any obstruction is offered.

The application of heat or cold or an attack of erysipelas may cause death of a part whose vitality is lowered by ligature of its main artery, when no such effect would be produced on healthy tissues by influences of the same intensity. Either form of gangrene occurs more often in the lower than the upper limb.

Extent.—The features of dry and moist gangrene are elsewhere described (page 129). Here we need only note that dry gangrene from obstruction in an artery may be of quite limited extent, or may involve the whole part up to the seat of ligature. Thus the circulation may only fail in the most distant parts—*e.g.* the toes—or a patch of dry gangrene may be occasioned by a thrombus forming in an artery, the seat of advanced calcification; or some particular anastomotic vessel may fail to enlarge: *e.g.* the posterior communicating cerebral artery. In such cases the gangrene is limited to a particular area of the primarily anæmic part. But in other instances, from great loss of blood or very feeble action of the heart, or advanced and general disease of the arterial system, or division or compression of many of the channels of anastomosis, the circulation in the entire part fails, and death occurs up to the seat of the ligature, or embolus, or other source of obstruction. Owing to the freedom of anastomosis around joints, the gangrene is not infrequently arrested at a joint.

Moist gangrene, on the other hand, usually extends at once up to the level of the arterial obstruction, for its exciting cause is generally some form of venous obstruction at this level, which affects the circulation in the entire limb below. By infection of the tissues it may spread up the limb above the seat of ligature.

The gangrene is usually noticed soon after the ligature—from the third to the tenth day is the most frequent time—but its occurrence may be delayed even so long as three or four weeks.

The utmost care must be taken to prevent gangrene by carrying out the treatment already specified for anæmic parts. (*See* page 534.)

Treatment.—In the case of *dry gangrene* involving only a limited area of the anæmic part, the surgeon should do his utmost to "nurse up" the neighbouring parts by local warmth and by judicious feeding of the patient, and, if necessary, the exhibition of appropriate cardiac tonics and stimulants, or narcotics and anodynes. It must be remembered that the parts adjacent to the gangrenous tissues are in a condition of "suspended animation;" their life is hanging in the balance, and no operation should be performed upon them. Gradually, if all goes well, the circulation becomes more vigorous, and the increased vitality of these parts is shown by the separation of the sphacelated portion When this evidence of the activity and vigour of the living parts exists, the surgeon should, with the least disturbance and injury, divide the bones and tendons—the most resistant structures—and so hasten what would otherwise be a tedious process; a little "trimming" of a stump may also be required. This waiting for the "line of demarcation" is unattended with danger, as absorption of poisonous material does not take place from the dry gangrenous parts.

Where the entire member up to the level of the arterial obstruction is in a condition of dry gangrene, amputation should be performed as soon as the patient's general condition renders it wise. Delay is not dangerous, as we have seen, but there is no local reason for delay, as the parts above the gangrene are not affected and "half killed" by the same injurious influence that has produced the gangrene. But if the patient is suffering from a severe loss of blood or any grave general disturbance, delay in operating is, of course, wise.

In *moist gangrene* the conditions are entirely different; the dead tissues are a source of continuous toxic infection of the patient, and there may be also a progressive local infection and spread of the gangrene. The extent of the gangrene, also, is known—up to the ligature. For these reasons, as soon as its existence is certain, the surgeon should amputate at the level of the obstruction; that will usually necessitate the division of the bone at a higher level, as the flaps must be fashioned from unquestionably healthy tissue.

V. SURGICAL HÆMORRHAGE: ITS VARIETIES AND PRINCIPLES OF TREATMENT.

The varieties of hæmorrhage, or bleeding, are classified, according to their *cause*, into pathological and traumatic; according to their *seat*, into cerebral, pulmonary, uterine, cutaneous, etc.; and these are grouped into external and internal, according as the blood flows from the surface or collects in some cavity or tissue of the body. External hæmorrhage is of chief interest to the surgeon; it is spoken of as *arterial, venous, or capillary*, according to the nature of the vessel from which the blood escapes; and another very important classification is made, according to the time after an injury at which bleeding occurs, into *primary*, *intermediary*, and *secondary*.

External hæmorrhage.—When blood is seen to flow from the whole surface of a wound, oozing out like juice from a cut orange, and not escaping from definite points, which can be recognised as the mouths of severed vessels, it is usually spoken of as *capillary hæmorrhage*. The blood is generally bright red in colour, and really escapes from the arterioles of the part; the bleeding from a cut finger is a familiar instance of this kind of hæmorrhage. It is the least important variety, as the loss of blood is slower, and is more easily arrested than when vessels of larger calibre are opened. When blood flows very freely from the whole surface of a very vascular tissue, it is sometimes spoken of as *parenchymatous hæmorrhage;* hæmorrhage from the lung, liver, spleen, kidney, erectile tissue, and very vascular tumours is of this nature.

When an *artery* is divided, bright red blood is propelled from its cardiac end in a forcible pulsatile stream, which rises with every beat of the heart, and falls in the interval. It can be arrested by firm pressure on the cardiac side of the wound. As the loss of blood continues, and the heart's power becomes less, or the opening in the vessel becomes narrowed, the distance to which the blood is propelled diminishes, and the effects of the ventricular systole become less marked, until the blood merely trickles from the end of the vessel. Sometimes the spontaneous closure of the artery suddenly arrests the flow of blood.

The characteristic features of arterial hæmorrhage may be lost; thus, in cases of asphyxia, whether from obstruction to respiration or an anæsthetic, the blood flowing from an artery is dark in colour. When the blood does not escape directly, but flows along a narrow or sinuous channel, as in the case of a punctured wound of a deep artery, it no longer escapes in jets propelled to a distance from the body, but flows evenly and continuously from the surface; but in such a case the rapid loss of bright red blood indicates the source of the hæmorrhage. Where anastomoses are very free, as in the head, face, and hand, blood will flow and even spurt out from the distal end as well as the proximal, either at once or after a short interval.

When a *vein* is wounded, dark red or purple-black blood wells up in a constant stream, uninfluenced by the cardiac contractions. It flows from the distal end of the divided vessel, and in the case of a large vein the flow is very rapid, and the blood may be projected to a short distance from the wound. The hæmorrhage is arrested by pressure on the distal side of the wound; it is often seen to be increased by expiratory efforts and by muscular strain. In the case of varicose veins, or of healthy veins wounded above their valves—*e.g.* the common femoral vein—blood flows from both ends of the divided vessel, and the hæmorrhage may be very profuse.

The force and rapidity with which blood flows from a wounded vessel depend upon the size of the vessel and its nearness to the heart, the size and direction of the wound in the vessel, and the force of the heart's contractions. The complete division of a vessel tends to close it and arrest the hæmorrhage: in the case of an artery by allowing its retraction and contraction, and in the case of a vein by its collapse.

Internal hæmorrhage is of great importance, not only from the loss of blood from the circulation, and in many cases the great difficulty of arresting it, but also from the disastrous local effects of the effusion of blood, particularly in the brain. This subject, so far as it is surgical, is dealt with in other chapters of this work, and here we need only refer to *subcutaneous hæmorrhages*, or hæmorrhages into the connective tissues of the body. When the infiltrated blood is in small quantity, filling out the potential cavities of the connective tissue, and not forming a distinct tumour, it is known as an *extravasation* or *suggillation;* when it is massed into a distinct tumour, but without communication with an artery, it is a *hæmatoma;* and when the communication between the artery and the blood tumour persists, it is a traumatic aneurysm. (*See* page 629.)

Primary hæmorrhage is that which occurs as the direct and immediate result of a wound of a vessel. The term is employed exclusively in reference to bleeding from injury; where disease, such as ulceration, opens up a vessel, the conditions from the first are those of secondary hæmorrhage.

The principles of treatment of primary hæmorrhage. (*a*) *Arterial.*—If the hæmorrhage has been *arrested by natural processes*, the surgeon should not interfere, and should be careful not to displace any of the clots in the wounded vessels by careless handling of the wound—especially rubbing. There are two exceptions to this rule:—In the case of an artery divided in a wound, the distal end may not bleed at first; but yet it is desirable to seek for it, and secure it in a ligature or by torsion, lest it bleed when the anastomotic circulation is established in full vigour. In a punctured wound of an artery, even when a clot has formed and stayed the hæmorrhage, the artery should be tied above and below the puncture, and the vessel divided between the two ligatures, to avoid the dangers specially connected with punctured wounds, viz. recurrent and secondary hæmorrhage and traumatic aneurysm.

The second great principle of treatment of wounded arteries is to *secure wounded vessels in situ;* for this purpose the wound should be enlarged, if necessary, even when this involves much greater difficulty than would ligature of the main artery of the part on the cardiac side of the wound. This principle rests upon the following facts:—Until the bleeding point is actually exposed, it is often impossible to determine from what artery a given hæmorrhage is occurring, and, therefore, if the main artery is tied on the cardiac side of the wound, the bleeding may not be arrested, or the operation may entail a much greater interference with the arterial supply of the part than is necessary.

For example, a gentleman was shot in the groin with a small bullet: a great effusion of blood resulted: instead of opening up the wound and searching for the wounded vessel, the surgeon proceeded to tie the external iliac artery. After death it was found that the blood had all been poured out from a wound of one of the small superficial branches of the common femoral artery. Take, again, the case of a stab in the axilla, with free arterial bleeding. The bleeding may be from wound of the axillary trunk or of one of its larger branches, such as the circumflex or subscapular, or from an intercostal artery. Without enlargement of the wound and actual exposure of the bleeding point, it may be impossible to determine the source of the hæmorrhage. Suppose that in place of this the surgeon were to ligate the subclavian artery in the third part of its course: if the wound had involved an intercostal artery, the hæmorrhage would continue unaffected. If, however, the wounded artery were a branch of the main trunk, the operation would have interfered with the arterial supply of the whole upper limb without any necessity; and even if the axillary trunk itself were wounded, such a method of treatment might still fail to arrest the hæmorrhage, because blood might flow through anastomosing channels into the artery between the ligature and the wound. This principle is of extreme importance, and the exceptions to its application are very few, and only arise where to carry it out would be impracticable; those exceptions are mentioned in the section on the wounds of special vessels.

The third cardinal principle of treatment is to *secure both ends of the wounded artery*, because the anastomoses of the branches of arteries are so numerous and free, and are so capable of enlargement, that blood readily finds its way into the distal portion of a divided artery, and escapes from the end if it is not securely closed. In certain situations, where the arterial anastomoses are particularly free—as in the scalp, face, and hand—blood will at once spurt out from the distal end of a divided artery almost as forcibly as from the cardiac end. But in other situations, even when there is no flow of blood at first, the dilatation of anastomosing channels soon brings blood freely into the wounded artery, and then hæmorrhage is liable to occur from the distal end of the vessel. It must be borne in mind that when an artery is divided or ligated in continuity, and its distal portion is for the time empty, the resistance offered by the anastomosing channels opening into it is less than that of the terminal capillaries supplied by the communicating arteries, and this accounts for the rapid establishment of the "anastomotic circulation." The necessity for securing the distal end of a divided artery is an additional reason for tying a wounded artery *in situ*. In some cases, when there is no hæmorrhage from it, it may be impossible to find the distal end of a divided artery; but diligent search should always be made for it in the case of a vessel of any size.

In applying these cardinal principles for the treatment of wounded arteries, torsion or the aseptic ligature is the best means to adopt.

(*b*) *Venous.*—The treatment of venous hæmorrhage presents far less difficulty, as a rule, than does arterial bleeding. The collapsibility of a vein, the low blood pressure within it, and the negative pressure in the veins near the heart, combine to remove most of the difficulties presented by wounded arteries. As a rule, only the distal end of a wounded vein requires to be secured. But the exceptions to this rule are very important exceptions; they are first, all veins influenced by the aspiration of the chest (*see* page 504); secondly, all varicose veins; and thirdly, all veins devoid of valves on the cardiac side of the wound or which bleed from the cardiac end. Superficial veins are easily controlled by a pad and bandage; deep veins should be ligatured. A punctured or lateral wound of a vein should be closed, if possible, by a lateral suture, or by a compress, without division of the vein. In the case of a wound of the main vein of a limb, it has been suggested that the companion artery should be tied as well as the vein, to prevent moist gangrene; but the suggestion is based on imperfect premises, and experience has shown it to be wrong.

On the other hand, when a surgeon is ligating an artery in its continuity for any purpose, and he happens to wound the companion vein, it has been shown to be the best practice to tie the artery at another spot, either just above or just below, and to leave alone the puncture in the vein, unless blood continues to flow from it after the ligature on the artery is tied. The explanation of this point of practice is twofold: in the first place, a puncture or lateral wound in a vein heals securely without occlusion of the main channel; and, secondly, the ligature of a main artery in its continuity so lessens the pressure of the blood in the main veins of the part, that the feeblest adherent clot is not displaced, and the process of cicatrisation of the vein is not imperilled.

(*c*) *Capillary hæmorrhage* is readily arrested by the application of cold, of heat, or of pressure; none of these means must be used of such intensity as to injure the living tissues.

(*d*) *Parenchymatous hæmorrhage* is more difficult to deal with, for it only occurs in circumstances in which the natural means of arresting hæmorrhage are at a minimum. The best means to employ are heat, the actual cautery, or the compression of a careful suture, or by means of a sponge or pad; styptics should be avoided, as a rule. The free hæmorrhage from an incision into the kidney can often be arrested either by sponge pressure or by irrigation with hot water, and, if these fail, by a suture; hæmorrhage from the placental site is best controlled by flushing the uterus with hot water; bleeding from the liver or spleen may be arrested by closing the wound accurately with sutures; hæmorrhage from very vascular tumours is best arrested by styptics or the actual cautery.

Intermediary hæmorrhage is that which is due to interruption in the temporary closure of a wounded vessel. It comes on within twenty-four hours of the infliction of a wound, before the process which results in the cicatrisation of the vessel is at all advanced. *It is, in fact, a recurrence of the primary bleeding*, and it most often arises from imperfect arrest of the primary hæmorrhage.

Causes.—(1) The first is *reaction* from the shock of an injury or operation; this is attended with increased heart-power and dilatation of arteries, and therefore soft, recently-formed coagula are liable to be forced out of divided vessels. The frequency with which this form of hæmorrhage arises from premature or excessive reaction has led to its being also called *reactionary hæmorrhage.*

(2) The other great cause is *movement of the part* displacing an external clot, and dragging upon the central clot and opening up the vessel again. Movement may also displace a badly-applied ligature; it is also injurious, by stimulating the heart and increasing the arterial tension.

(3) *Slipping of a ligature* from faulty application, or its very rapid softening, may allow the vessel to open up again.

(4) *Failure to secure the distal end of a divided artery.*—It sometimes happens that the distal end of a divided artery does not bleed at the time, but after an interval, when the heart has recovered its power, and the anastomosing vessels have dilated, blood reaches and flows from the open distal end of the artery.

(5) *Failure to occlude the lumen of a wounded artery* which is the seat of a punctured wound.—We have already mentioned the great liability to recurrence of the bleeding from punctured wounds of an artery if the stream of blood through the vessel is not arrested. The clot which forms in the puncture is very liable to be forced out during reaction from the shock of the injury, when the blood pressure rises to the normal, or even above the normal.

As intermediary hæmorrhage is a recurrence of primary hæmorrhage, and has no special features of its own, it must be treated in all cases in accordance with the principles that guide in the treatment of primary bleeding. The wound must be opened, and the bleeding vessel sought and secured by a ligature.

Secondary hæmorrhage.—Secondary hæmorrhage is usually defined as occurring at any time between twenty-four hours after the infliction of a wound and its complete healing. Such a definition is meaningless and useless, and ought to be abandoned. The true definition of this variety of hæmorrhage is "bleeding arising from failure in the repair of an injured or ligatured vessel, or from ulceration into a vessel." Most of the cases are traumatic in origin, but hæmorrhage arising from ulceration into an artery is of the same nature, and requires the same treatment as secondary traumatic hæmorrhage. No form of bleeding is so insidious or so terrible as secondary hæmorrhage, and its treatment is beset with difficulties. It is very important for the surgeon to be thoroughly acquainted with its causation and phenomena, and to have a firm grasp of the principles to be applied in its treatment. Formerly it was of frequent occurrence, but the introduction of aseptic surgery has rendered it rare, although it has not entirely abolished it from surgical practice.

Causes.—Secondary hæmorrhage occurs under one of two conditions:

(1) When the lumen of a vessel is opened up by ulceration of its walls (septic arteritis).

(2) When the pressure of the blood within it ruptures a healing artery before a strong resisting cicatrix has been formed.

1. **Septic arteritis.**—The infective material may reach the artery from the general wound surface, on the weapon inflicting the wound, in the ligature applied around the artery, from an abscess cavity or infective ulcer adjacent to the artery, or from the blood within the artery. The results of such infection will vary with the intensity of the infective material and with the vital resistance of the arterial walls. Even when the ligature is septic, and suppuration occurs around the artery, there may be no interference with the plastic healing processes in the artery and its contained clot, and the vessel may be permanently and securely obliterated above and below the line of ulceration about the ligature. But if the infective material in the wound be very virulent or contains the streptococcus pyogenes, a spreading destructive inflammation of the part may occur, and expose the patient to grave danger from bleeding. On the other hand, age, cachexia—especially from septicæmia and pyæmia, diabetes, chronic renal disease, or alcoholism—and advanced atheromatous or calcareous disease of the artery, are conditions which may seriously lessen the vital resistance of an artery to septic infection and destroy its power of resisting the attacks of pyogenic organisms. Of equal or even greater importance in this respect is the severance of some of the coats of an artery by a ligature or other hæmostatic. With all its coats intact an artery may be bathed in pus and stand proof against ulceration; but if the middle and inner coats are ruptured and the outer coat contused or strangled, the tissues become an easy prey to infective organisms, and, in any case, the process of ulceration is greatly facilitated.

2. **Rupture of a healing artery.**—We have already seen that the clot inside a divided or ligatured artery becomes honeycombed by intercommunicating fissures into which blood flows from the open artery, and that these channels form communications with new vessels developed from the columns of plasma cells passing through the arterial walls into the clot. The blood in these channels is flowing with the same pressure as the blood in the artery, and thus it comes about that the young cellular tissue, which is destined to repair the artery and develop into firm resistant cicatricial tissue, is exposed to considerable force; in adverse circumstances it is ruptured by it, and secondary hæmorrhage results. The conditions which tend to facilitate this accident are threefold: increase in the blood pressure in the repairing artery, hindrance to or impairment of the reparative process, and weakening of the arterial wall.

The blood pressure may be increased by plethora, by cardiac hypertrophy, by chronic renal disease, or by excited action of the heart from mental or physical stimulation.

The rapid and perfect repair of an artery is impeded by advanced disease of the vessel, by cachexia, by hæmophilia, by adjacent inflammation, by the proximity of a large branch of the artery through which blood continues to flow, and, in the case of punctured wound or incomplete division of an artery, by the forcible stream of blood flowing through the vessel.

The weakening of the arterial wall is caused by division of any of its coats; and when it is borne in mind how thin the outer coat of an artery—particularly of some arteries—is, and how great a proportion of the total thickness of an artery is due to the inner and middle coats, it is apparent that a ligature or other hæmostatic agent which severs the inner coats does thereby greatly lessen the strength of the artery and its power to withstand the pressure of the blood. (*See* page 531.) We may repeat here that the physiological occlusion of such an artery as the ductus arteriosus is effected by Nature without the severance of any part of its walls, and without any danger whatever of secondary hæmorrhage. That severance of part of the thickness of an artery should facilitate, and render quicker and safer the repair of the vessel, is contrary to all we know of the repair of other parts and tissues. Nowhere does increase of an injury promote its repair.

It must be remembered that while we have spoken of two conditions leading to secondary hæmorrhage, and discussed them separately, they may, and often do, *co-exist.* Septic arteritis may destroy the end of a divided vessel, but above it a plastic process may obliterate the vessel, and save the patient from hæmorrhage when a septic ligature separates and the end of the vessel sloughs off. On the other hand, the influence of the inflammatory process may be seen in such a delay of the reparative process in the artery beyond, that when the ligature separates, the soft reparative material yields before the blood pressure and hæmorrhage occurs. In other cases the septic inflammation is so intense that it arrests all efforts at repair and the artery is directly ulcerated into.

One or two other special causes must be noticed. Thus the knot of a ligature, if very hard and tied firmly, may quickly ulcerate through the thin outer coat of the artery, and open up the vessel before the organisation of the thrombus has advanced far enough to enable it to withstand the force of the circulation. For this reason a flat ligature is preferred by many surgeons, and in any case a supple thread. (*See* Fig. 152.)

In tying an artery in its continuity, a branch may be wounded just below the seat of ligature. At the time no hæmorrhage occurs, because the vessel is empty, but gradually, as the anastomotic circulation becomes more vigorous, the tension rises in the wounded vessel until the organising thrombus in it is displaced or torn through, and secondary hæmorrhage occurs. Premature softening of a ligature which has severed the inner and middle coats of an artery, by leaving the thin outer coat unsupported at a time when the healing process is not sufficiently advanced to withstand the blood pressure, is another occasional cause of secondary hæmorrhage.

Idiopathic secondary hæmorrhage occurs in all cases from disintegration of the arterial wall by septic arteritis. It is met with in abscesses, ulcers, and infected wounds opening into a neighbouring artery.

Thus we may enumerate the causes of secondary hæmorrhage as follows:—(1) Septic infection or sloughing of the artery, due to infection of the wound, of the ligature, or of the blood clot; (2) yielding of an arterial cicatrix; (3) division of the inner coats of an artery, weakening its walls; (4) advanced disease of the arterial wall; (5) cachexia, hæmophilia, chronic renal disease, diabetes; (6) plethora; (7) increased force of the ventricular contractions; (8) punctured wound or incomplete division of an artery; (9) proximity to the ligature of a large branch; (10) special thinness of the outer coat of an artery; (11) wound of a branch arising just below a ligature in continuity; (12) faulty ligature.

As the cause of secondary hæmorrhage is in all cases a change in the wall of an artery, causing it to yield to the internal blood pressure, it often happens that the bleeding is preceded by an aneurysmal dilatation of the end of the vessel. This is well seen in the case of hæmorrhage from phthisical lung cavities, where arteritis is the cause of the bleeding, and is usually attended with marked aneurysmal dilatation of the vessel before the bleeding occurs. This occurrence of aneurysm must not be confused with the bulbous form of a tied artery, which is due to the contraction of the artery above the tied end from lessening of its function; the actual tied part cannot at once contract, because it is filled with clot.

Secondary hæmorrhage very often occurs from the distal side of an artery ligated in its continuity. Various reasons have been given for this. It has been stated that the application of a ligature must necessarily interfere with the vasa vasorum supplying the vessel wall immediately below it, while it need not affect at all those supplying the vessel above it. Again, the internal clot is smaller and later in being formed on the distal than the proximal side of a ligature. Another and probably more important explanation lies in the fact that the obstruction offered by the capillaries of a part is greater than that offered by the anastomosing channels through which the blood flows when a main artery is tied. The blood pressure in the artery above the ligature is determined by the obstruction offered by the channels of anastomosis; but the blood pressure in the artery below the ligature is determined by the obstruction offered by the capillaries of the part it supplies. The pressure may, therefore, be greater in the artery on the distal than on the proximal side of a ligature.

Phenomena of secondary hæmorrhage.—Secondary hæmorrhage is always caused by progressive molecular disintegration of an artery, and the breach in its walls is at first minute, and allows only a tiny stream of blood to escape. In and around this tiny aperture a clot forms and stops the loss; but as the disintegration proceeds, this clot is detached, and fresh hæmorrhage results. This also may be arrested by a clot, and again a further disintegration taking place, the hæmorrhage is renewed. This recurrent character, with a progressive increase in the amount of blood lost, until it escapes in a full stream, is peculiar to secondary hæmorrhage, and is exceedingly important to bear in mind, for serious secondary hæmorrhage is always preceded by a slight loss, which should warn the surgeon of what is about to occur.

The earliest sign, therefore, is the escape of a few drops of blood, merely staining the pus or other discharge, and this may be mistaken for oozing from granulations. The blood, however, will be noticed to come from the depth of the wound, and to be unconnected with anything that could injure granulations, such as probing. This oozing stops, and in a few hours, or longer, is followed by another oozing —more free this time—and again the bleeding stops. But after a short interval another bleeding occurs, and this time, perhaps, the loss is so rapid that unless quickly arrested by the surgeon it proves fatal. Too much importance, therefore, cannot be attached to the occurrence of a slight oozing of blood from the deep part of a wound in which an artery has been tied or exposed, or from an abscess cavity near a large artery. It is the herald of the dreaded secondary bleeding, and the surgeon who observes it should at once make his preparations to deal with the yielding artery, and the patient should be continuously watched by a skilled attendant.

In some cases the hæmorrhage is preceded by a rise of temperature and by pain and deep swelling in the part, due to escape of blood around the artery.

Principles of treatment of secondary hæmorrhage.—As soon as ever it is clear that secondary hæmorrhage has occurred, active measures should be taken to deal with it. The fact that the bleeding has ceased when the surgeon reaches his patient in no way modifies this injunction. In the case of primary hæmorrhage, when the bleeding has ceased the surgeon should not interfere; but in a case of secondary hæmorrhage no such rule must be followed, for Nature is unable to deal permanently with a yielding and ulcerating vessel, and the cessation of bleeding is but temporary, and will surely be followed by a more profuse hæmorrhage. For the same reason, the amount of blood lost must not be allowed to decide the course to be pursued. The slight hæmorrhage is but the prelude to the more profuse, and if the surgeon waits to interfere until the bleeding is serious in intensity, he exposes his patient to grave danger, and will certainly be called upon to act in less favourable circumstances. It is necessary to insist upon this point, because in its early stages secondary hæmorrhage is very deceptive, and the surgeon may easily be thrown off his guard; and the means to be adopted may appear to the unthinking out of proportion to the obvious necessities of the case. If success is to be attained, the proper treatment cannot be carried out too early or too thoroughly.

When the very first trace of hæmorrhage is noticed, the main artery above should be controlled by digital compression or an artery compressor, in the hope that, by diminishing the blood pressure in the injured artery, repair may progress and seal over the wound with an unyielding cicatrix. The pressure, if successful, must be continued for several days, and left off gradually. This treatment is likely to succeed in those cases where the hæmorrhage is due to the proximity to the ligature of a large branch of the artery, or to the weakening of the arterial walls by disease, or their severance by the ligature. It is not so likely to succeed where septic arteritis is disintegrating the vessel. Where it fails, or where a free hæmorrhage has already occurred, and in situations where it cannot be employed, the artery should, if possible, be ligatured.

For secondary hæmorrhage from wounded arteries, such as those in amputation stumps, or from arteries in the walls of abscesses and ulcers, the treatment that is almost always necessary is to open the wound—however far advanced healing may be—turn out the clots, and expose the bleeding vessel, which must then be secured by a carefully-applied ligature. Until this is done, the hæmorrhage is stayed by the use of a tourniquet, where that is possible. The wound itself should be opened up rather than the main artery be cut down upon at a distance, because it is often uncertain exactly what vessel may be bleeding; and to ligature the main artery of a stump or part may be unnecessary, while the anastomotic circulation may quickly lead to further loss from the unobliterated vessel. When the wound is opened up, a careful search may be needed to find the artery at fault, for the opening in it may be closed by a clot; the surgeon must carefully look for such a clot, may then rub the wound well with a sponge, and particularly follow up any stream of bright arterial blood, however small. Having found the vessel, he must proceed to ligature it. This is not so easy as in the case of a primary wound. The artery is more or less perfectly embedded in inflammatory exudation or granulation tissue, which fixes it to its companion vein, nerves, and other structures. Its own wall may also be so attenuated by the former ligature, or by septic arteritis, that it tears through at once if seized in a forceps. The blunt end of a director should be used to separate the artery rather than a knife or any sharp instrument. When the artery has been previously divided in a wound, it is separated well above the wound surface, and there carefully tied. When the artery has been previously tied in its continuity, or is ulcerated into in its continuity, a double ligature should be placed on it, above and below the leak, and well away from the surface of the wound. The ligatures should be of aseptic silk, and tied so as not to divide the inner and middle coats of the vessel. Torsion, acupressure, and forcipressure should not be employed in the treatment of secondary hæmorrhage.

To secure the artery with a ligature is, however, only half the surgeon's aim. He must also endeavour to render the part aseptic; for if he does not succeed in this, the continuation of the process which led to the bleeding will lead to its recurrence. The greatest care should be taken to apply efficient antiseptics to every part of the wound, and especially to the vessel and the tissues around it.

A light antiseptic dressing should be applied, through which bleeding, if it recurs, will at once be detected, and the patient should be constantly watched. Great care should be taken to keep the arterial tension as uniform as possible; to this end, strict rest should be enjoined; the diet should be light and unstimulating; the bowels should be carefully regulated and relieved, if necessary, by enema; while pain and restlessness must be subdued by opium. As far as possible, all excitement must be allayed, and the patient must be encouraged to believe that no recurrence of the bleeding will occur.

In certain conditions the *treatment by ligature cannot be carried out:—*

(*a*) Where the tissues are sloughy, and a ligature cannot be made to hold, but at once cuts through the vessel, the actual cautery should be employed. The cautery must be at a dull red heat, and applied for a long time, so as to char the artery and surrounding tissues deeply. This not only secures a firm temporary closure of the vessel, but it renders its end aseptic, and it is this fact which explains the success yielded by the cautery in some apparently desperate cases.

(*b*) In certain suppurating and sloughy wounds, the hæmorrhage occurs not from any one large artery, but blood oozes out from the whole surface—"parenchymatous oozing." The bleeding has the recurrent character of arterial secondary hæmorrhage, and, if unchecked, is ultimately fatal. To tie the bleeding vessels is impossible, and pressure is of only temporary avail. Such cases may be dealt with by searing the whole surface with a hot iron, by daily application of finely-powdered dried sulphate of iron, or, best of all, by continuous irrigation with ice-cold boric acid solution.

(*c*) There are rare cases in which, a few days after an operation, venous bleeding occurs, and recurs day after day, delaying healing, and eventually becoming serious from loss of blood. Where it can be exerted, firm pressure is the best means to adopt in these cases; in other instances styptics may be injected into the wound. The writer has met with two instances of this hæmorrhage. In one the venous bleeding, gradually becoming more abundant, persisted for more than a week after excision of the mamma for scirrhus; in the other it occurred after amputation through the lower third of the thigh.

(*d*) In certain other cases the opening up of the wound and ligature of the bleeding artery is impracticable, on account of the position of the vessel. The deep arteries of the neck, groin, and abdomen are instances of this. In such cases the only course open to the surgeon is the careful plugging of the wound with aseptic gauze, which must be kept in position undisturbed for several days. In the case of cancerous ulceration into arteries, which occurs most often in the neck and groin, any treatment is unavailing to save life; and many surgeons regard active efforts to stop the bleeding as uncalled-for and inexpedient if they give pain to the patient. In the groin success may be obtained by passing a large curved armed needle deeply beneath the artery and vein and ligaturing them *en masse*.

(*e*) In the case of amputation at the shoulder joint or hip joint, where secondary hæmorrhage comes on when the stump is almost healed, it has been proposed to ligature the main artery higher up rather than cause severe shock and extensive wound surface by opening up the stump. Such a practice has been followed by success, but the ligature of the bleeding vessel at the seat of injury is the more satisfactory procedure, as already stated.

(*f*) In some instances, when it is known what artery is bleeding, instead of opening up the original wound, and finding a healthy portion of vessel to tie above or below the leak in it, it is better to cut down on the artery through a fresh wound, and apply the ligature. This may not only be an easier means of reaching and of tying the vessel, but it may be safer, inasmuch as the second wound can be kept free from infection from the first. For example, in a case of amputation through the lower end of the femur with secondary hæmorrhage, known to be from the popliteal artery, it is sound practice to tie the superficial femoral artery in Hunter's canal.

(*g*) In certain cases where the ligature presents special difficulties —*e.g.* either tibial artery in the leg—firm compression of the part has been recommended; the pressure must be applied very evenly and cautiously, and should be left undisturbed for several days.

When secondary hæmorrhage occurs from an artery that has been tied in its continuity, careful compression of the artery where tied by a pad and bandage may be tried at the first occurrence of the bleeding; but if it is not wholly successful, valuable time must not be lost in readjusting the bandage, but a ligature should be put on above and below the bleeding point. If now we take the case of an artery tied in its continuity from which secondary hæmorrhage recurs even after renewed ligature, there are at least two courses open to the surgeon. He may tie the main artery higher up, or he may amputate the limb. In the lower limb a double block in the main artery exposes the patient to so great a risk from gangrene that it should not be made. In the upper limb the conditions are more favourable, and a second block in the arteries may not cause gangrene. The main artery may, therefore, be ligatured higher up, and only when that has failed should amputation be resorted to. In the lower limb, where re-ligature of the artery at the same spot has failed, amputation is the only resource. The amputation may be at a spot immediately above the seat of ligature. The explanation of the success attending amputation when ligature in continuity fails lies in the fact that removal of the limb greatly lessens the function of the main artery, and this leads to contraction of the arterial trunk for some distance above—as high as the subclavian or iliac trunk—and to diminution of the lumen of the vessel by endothelial overgrowth. In some cases the amputation substitutes an aseptic for a septic wound.

The constitutional treatment of hæmorrhage already described must be carefully carried out. The patient must be kept at rest, and a nurse must be in constant attendance both to attend to all his wants and to observe immediately any recurrence of the bleeding, and to arrest it by a tourniquet.

V. WOUNDS OF SPECIAL VESSELS.

Of the head and neck.—Wounds of the **common carotid** artery, if not immediately fatal, must be treated by a double ligature of the vessel, and if secondary hæmorrhage occurs, the artery may be re-ligatured above and below the first ligature; and if that fails to arrest the bleeding, the wound must be carefully plugged. Wounds of the **internal carotid** artery must, if possible, be dealt with by a double ligature at the seat of injury; if secondary hæmorrhage occurs, the common carotid artery and the orifice of the external carotid artery should be tied. When the vessel is wounded close to the skull it is quite impossible to ligature it there, and the surgeon must then trust to tying the artery lower down in the neck or the common trunk.

Wounds of the **external carotid** artery and its branches.—Wherever possible the divided ends of the artery should be picked up and tied, and care must be taken to tie the distal end, as the anastomoses of the branches of this artery are so free. In the case of hæmorrhage from the deep branches of the external carotid, such as the internal maxillary, where the local ligature cannot be applied, the external carotid trunk should be tied just beyond the origin of its thyroid branch; and if this fails to arrest the bleeding, the external carotid artery of the opposite side should be tied, as in such a case the continuance of the hæmorrhage will be due to blood reaching the wounded vessels across the middle line through the anastomoses from the other side. In no case should the common trunk be tied for wound of the external carotid or its branches. In the case of the lingual artery wounded in the mouth, when the bleeding point cannot be seized the vessel may be tied in the neck where it lies above the hyoid bone.

Wounds of the **palatine arteries** often bleed very freely: in the soft palate the vessel can be seized in pressure forceps; in the hard palate, if this be impracticable, a ligature may be passed by means of a fine curved needle, and tied; and if other means fail, a peg of ivory or wood may be forced up the posterior palatine canal.

The **arteries in the face and scalp** are often most conveniently occluded by a suture, which serves the double purpose of closing the wound and arresting the bleeding; harelip-pins or silk sutures may be employed, care being taken to pass the suture beneath each end of the vessel. For the scalp, pressure by pad and bandage may be safely employed, as the underlying bone forms a firm surface for counter-pressure. Bleeding from a punctured scalp artery may sometimes be arrested by completely severing the vessel; this method used to be employed after arteriotomy of the temporal artery.

Wound of **meningeal artery** must be closed by a fine double ligature; it is often impossible to seize the artery in forceps and tie it, and then the ligature must be carried round the artery by a needle passed under it. If wounded in a bony canal, it must be plugged by gauze or an ivory peg.

Wounds of the **subclavian artery** appear to be invariably fatal: wounds of the *suprascapular* or *transverse cervical* arteries must be treated by double ligature in the wound. Wounds of the *vertebral* artery present a special difficulty both in diagnosis and treatment; for as compression of the common carotid artery against the transverse process of the sixth cervical vertebra may occlude the vertebral trunk also, it is easy to see that the arrest of the bleeding from this pressure may be wrongly taken to indicate that the wounded vessel is the carotid or one of its derivatives when it is really the vertebral trunk. The only way to make sure of the source of the bleeding is to enlarge the wound until the finger can be introduced and feel the bleeding point. If the wound involves the vessel below its entrance into the cervical spine, the artery should be tied above and below the puncture; but where it is punctured between two transverse processes, there is no room to place a double ligature; the best plan in such a case appears to be to plug the wound very carefully, taking great pains to have the deepest part of the plug accurately fitted in between the transverse processes of the vertebræ. Ligature of the artery below the sixth vertebra and above the atlas has been suggested: the operation is attended with difficulty and some degree of danger.

Small wounds of the **internal jugular vein** should be closed by a lateral ligature or suture of the vein; extensive wounds of the vein require a double ligature. Wounds of a *cranial sinus* must be dealt with by careful plugging with a strip of aseptic gauze.

Hæmorrhage from the **socket of a tooth** may be long continued and serious in amount. It is to be arrested by removing all clots from the socket, and passing a narrow strip of lint firmly down to the very bottom of the socket from which the blood is coming, and then filling in the cavity lately occupied by the tooth with lint, until the little pad rises just above the level of the adjacent teeth. This pad is held in position by firmly closing the patient's mouth.

Of the trunk.—Wounds of the **internal mammary** and **intercostal arteries** are best treated by double ligature *in situ*. This subject is more fully discussed in Article XLIV., on THE SURGERY OF THE CHEST, Vol. II.

Wounds of the **epigastric** and **lumbar arteries** must be closed by double ligature, and it is often necessary to enlarge the wound considerably to expose the severed vessel; the ends of the epigastric artery, in particular, may be found considerably retracted.

The artery of the *frænum præputii* is sometimes torn, and continues to bleed freely. Complete division of the vessel in the wound often stops the hæmorrhage, especially with the aid of gentle pressure; if not, the proximal end of the vessel must be seized and tied. The artery of the *hymen,* when torn, may also lead to serious hæmorrhage; if pressure fails to arrest it, the vessel must be tied.

Of the upper limb.—If the **axillary artery** or one of its branches be wounded, the bleeding vessel must be exposed and a ligature applied to both ends. It is important to follow the cardinal rule in these cases, as it is often difficult, and even impossible, to determine by other means what vessel is wounded; and the anastomoses of the branches of the axillary artery are so free that if a ligature is not applied to the distal end of the wounded vessel the hæmorrhage is almost certain to recur. In such a case, considerable enlargement of the wound may be necessary; and if the wound is high up in the axillary trunk, the pectoral muscles may have to be divided. Until the vessel is securely ligatured the hæmorrhage must be controlled by compression of the third part of the subclavian artery. Wounds of the **axillary vein,** if not too extensive, should be closed by a lateral ligature.

Wounds of the **brachial artery** and of the *radial* and *ulnar arteries* in the fore-arm must be treated by double ligature *in situ.*

Wounds of the **palmar arches** often occasion considerable trouble. They may be dealt with by a carefully-applied graduated compress over the wound, combined with full flexion of the elbow. But the results of this plan of treatment have been in many cases disastrous. It is far better to enlarge the wound carefully, expose the bleeding artery, and tie it on each side of the wound in it. To expose the deep arch, the inner head of the abductor indicis may be detached from the second metacarpal bone. If treatment by compression has been tried, and has failed, the local ligature should be resorted to whenever possible. But if this is impracticable owing to the sloughing in the palm, the graduated compress may be tried once more; and if this fails, the radial and ulnar vessels may be tied close above the wrist, or the brachial artery at the bend of the elbow. Wherever possible, the vessel should be tied at the seat of injury.

Of the lower limb.—Wounds of the **gluteal** and **sciatic arteries** in the buttock are very serious injuries; they should not be treated by plugging the wound or the application of styptics. When the vessel is wounded outside the pelvis, it should be cut down upon, and a ligature applied to each end. If exploration of the wound in the buttock shows that the hæmorrhage is from a wound inside the pelvis, the surgeon may resort to compression of the common iliac artery by tourniquet or the rectal lever, or he may place a ligature on the internal iliac artery; and the best way to reach it is through the peritoneal cavity. Of these methods, the ligature is to be preferred whenever possible. Wounds of the **femoral artery or its branches** require double ligature at the seat of injury. The same method must be employed in wounds of the *popliteal or tibial arteries,* and wounds of the *plantar arch* are to be treated in the same way as wounds of the *palmar* arch.

In cases of secondary hæmorrhage from wounds of the femoral or popliteal artery, the wound must be opened up, and the vessel religatured above and below the bleeding point. When the hæmorrhage is threatening, it may be prevented by digital or other compression of the main artery higher up, or by careful aseptic plugging of the wound. If the bleeding recurs after a second ligature of the vessel, amputation at the level of the wound in the artery is the only resource, as ligature of the artery at a higher level exposes the patient to the gravest risk of gangrene.

Wounds of the **femoral vein** are very grave injuries. A small wound should be closed by a lateral ligature of the vessel, a large wound by a double circular ligature; the companion artery should not be ligated unless wounded. If the heart and vessels are healthy, the popliteal or the femoral artery and vein may be simultaneously ligatured without the occurrence of gangrene. **Rupture of a varicose vein** of the leg—or its wound—may be easily controlled by the pressure of a pad, fixed on by a bandage extending up from the roots of the toes.

XXIV. DISEASES OF BLOOD-VESSELS.

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I. ARTERITIS.

Several varieties of arteritis, or inflammation of arteries, have been described. These varieties are distinguished by their cause as traumatic, embolic, and spontaneous; by their intensity and course as acute and chronic, and by the coat of vessel most markedly or earliest affected, as endarteritis, mesarteritis, and periarteritis. As a rule, it is the inner coat which is the special seat of inflammatory change. The older surgeons described a diffuse acute or erysipelatous arteritis as a common disease. It is now known not to exist, the post-mortem staining of the tunica intima having been mistaken for the redness of inflammatory hyperæmia. Very occasionally, in acute rheumatism, the part of the aorta immediately adjacent to the heart may be the seat of changes exactly resembling acute endocarditis. Rarely, also, atheroma may develop acutely, run a rapid course, and lead to the production of scattered patches of swelling in the tunica intima of the aorta. These, however, are pathological curiosities of little or no importance in surgery. The following varieties of arteritis are of great practical moment :—

The acute varieties include *plastic* or *traumatic arteritis*, *suppurative arteritis*, and *embolic arteritis*. Several forms of *chronic arteritis* have been recognised. Of these the most important are—*atheroma*, *syphilitic arteritis*, *tuberculous arteritis*, and *obliterating arteritis*. In each form the changes are most marked in the inner coat of the artery.

1. **Plastic or traumatic arteritis.**—By this is meant the sum of the processes which lead to the healing of a wounded artery. It is characterised by the effusion of plastic lymph and its subsequent organisation. According to circumstances, this lymph either seals a wound in an artery, closes the end of a divided vessel, or infiltrates a thrombus and occludes some extent of the lumen of the tube. It is associated with similar changes in the sheath of the artery, for the injuries which it repairs always involve the sheath as well as the vessel. It is a strictly local process, with no tendency to spread far beyond the injured area. It is also a conservative constructive

process, giving rise to no symptoms, requiring no treatment; and it is hardly accurate to speak of it as a disease of arteries, but rather as a process of repair. We have already considered the details of this repair in the previous Article. (*See* page 512.)

2. Suppurative arteritis.—This is an intense acute inflammation of the coats of an artery, caused by the growth of pyogenic organisms, and is exactly analogous to acute suppuration in other tissues. The arterial wall is infected in one of three ways: most often by the spreading of suppuration and ulceration from the surrounding tissues, sometimes by an infected ligature, and sometimes the poison may be contained in the blood, and infect the vessel through the thrombus formed in it. However the disease originates, it is attended with dilatation of the vasa vasorum in the sheath and the outer coats of the vessel, and by a rapid exudation of serum and migration of leucocytes. The wall of the artery is swelled and softened, the endothelium is shed, and blood coagulates in the interior. By the peptic action of the pyogenic toxines, the cells and fibres of the arterial wall are broken up and dissolved, and the artery "ulcerates." In some cases the destructive process is marked by the formation of distinct sloughs of the vessel. The softening and destruction of the vessel-wall exposes the patient to grave risk of hæmorrhage—this disease is the chief cause of secondary hæmorrhage—and, indeed, this result would be invariable were it not that the blood coagulates within the vessel, and that often the clot completely fills the artery and extends beyond the infected portion of the vessel, which then becomes shut off by plastic arteritis. In small arteries this often occurs. In large arteries the rapid stream of blood interferes with coagulation, and may prevent complete obliteration of the lumen with a clot, and then hæmorrhage is sure to occur when the arterial wall softens. A striking instance of this is when a large artery is exposed in the wall of an acute abscess; if the artery becomes infected, that part of its wall which projects into the abscess, yields, forming an aneurysmal pouch, and then bursts into the abscess cavity. The hæmorrhage in the late stages of phthisis may be from this cause. A few years ago I had a case of suppuration in the neck, in which nearly the whole length of the common carotid artery was destroyed by this disease, and death occurred from secondary hæmorrhage. An abscess in the neck, into which the common carotid artery had opened, was incised by Liston with disastrous result. The liability for the suppurative process to extend to arteries is the reason why all acute abscesses near large vessels should be opened and disinfected at the earliest possible moment. Injury of an artery, as by a bullet or other foreign body, a sequestrum or a ligature, renders it less able to withstand the influence of infection, and the destructive process advances more rapidly.

3. Embolic arteritis may be considered separately, owing to its great interest. When a non-infective embolus lodges in an artery, the blood coagulates upon it until it completely blocks the vessel, the endothelium proliferates, and all the changes of plastic arteritis, ending in permanent obliteration of the vessel, ensue. But if the embolus is infective—*e.g.* a portion of fibrin containing micro cocci washed from an aortic valve in a case of ulcerative endocarditis—an acute inflammation of the artery ensues, which often ends in suppuration, and this is the explanation of the abscesses which occur in the course of ulcerative endocarditis and pyæmia. The arteritis may, however, stop short of suppuration, and merely cause softening of the arterial wall. Then, under the pressure of the blood, the artery yields, forming an aneurysm, or it may burst and occasion hæmorrhage. Idiopathic aneurysm in children and young people is probably always due to the softening effect of an irritant embolus; and some aneurysms in older patients are caused in the same way. This form of arteritis occasionally occurs in the course of acute rheumatism.

4. **Atheroma, arteritis deformans, chronic endarteritis.**—These names denote a chronic sub-inflammatory disease of the arteries, which is so common in old persons as to have been regarded as a constant effect of old age. The inflammatory products always undergo fatty or calcareous degeneration, and it has sometimes been considered a degeneration of arteries rather than an inflammation. But the disease is distinctly inflammatory in nature, and in its most intense form has been described by Cornil and Ranvier as *acute endarteritis.* All grades of intensity between this form of the disease and the least intense, which most nearly resembles a degeneration, are met with.

Causes.—*Old age* is the predisposing cause of atheroma, by lessening the power in the arteries to withstand the strain of the circulation and the the pressure of the blood within them. The exciting cause of the disease is *mechanical strain*, and we therefore find that all conditions which increase this strain tend to induce atheroma. The disease is more frequent in men than women, and in those whose occupation or habits expose them to sudden and severe or prolonged effort. *Plethora* acts in a similar way, and on the other hand, wasting disease protects from atheroma. The obstruction to the flow of blood through the arterioles from arterio-capillary fibrosis, or from spasm of the arterioles, which occurs in *chronic renal disease* with contracted kidney, by raising the blood pressure in the arteries excites this disease. *Gout* leads to atheroma, chiefly by causing renal disease and arterio-capillary change, but possibly also in this dyscrasia the blood itself acts as an irritant. *Alcoholic excess* is an important cause of chronic endarteritis, partly from the direct irritant influence of the alcohol, partly from the chronic renal disease it sets up, and partly from the cardiac excitement it induces. *Syphilis* has been asserted to cause atheroma, but this is doubtful; if, however, there is widespread syphilitic endarteritis of the smaller vessels, the obstruction to the circulation thus induced may lead to atheroma. That syphilis has any more direct influence in exciting the disease has not been proved.

The *distribution* of the disease is strong evidence of the influence of strain in its causation. It is most frequent in the aorta—especially its arch—and the large arteries. It is less common in the smaller arteries, excepting those at the base of the brain. It is more common in the arteries of the lower limb than of the upper, because the pressure in them is greater; and of all the visceral arteries the coronary of the heart and the splenic are the most liable to the disease. Atheroma is rare in the pulmonary artery, and is only met with when the pressure in the vessel is increased, owing to obstruction in the circulation in the lungs or at the mitral valve, with compensatory hypertrophy of the right ventricle. The disease attacks especially the curved parts of arteries and the parts around the mouths of lateral branches, and where the vessel is in contact with bone, as along the back of the descending aorta, at the back of the femoral artery where it passes over the pelvis, and at the front of the popliteal artery.

Naked-eye appearances.—The disease occurs in the form of circumscribed raised patches on the inner surface of the artery. These may be in the form of circles around the mouths of branches, or in linear streaks, but are often oval in form; they grow at their margins, and so tend to coalesce. When these patches are numerous and much raised they justify the term sometimes applied to the disease—*arteritis nodosa vel deformans.* The patches are firm in consistence, a dull white or yellow in colour, and they make the vessel less elastic and resilient. In the later stages flat calcareous plates are seen, and the edge or surface of a plate may be bared of endothelium, and then is liable to have fragments of clot adhering to it. In other cases, the atheromatous patches are softened down, and when cut into a thick grumous fluid escapes—an *atheromatous abscess*—or such an abscess having burst, a depressed pit is left in the arterial wall—an *atheromatous ulcer.*

Pathological changes.—The disease begins in the deeper layers of the tunica intima, in a proliferation of the small flattened cells lying between the layers of fibrous tissue; this tissue also increases in amount, and in all but the more acute forms of the disease—called sometimes acute endarteritis—the diseased patches are formed in greater part of thickened layers of fibrous tissue with a few oval, flat, or stellate cells between them. Over this diseased area the superficial layers of the intima, with the endothelium, are continued unaffected. No vessels are developed in the new tissue, and after a time, which varies much in different cases, degeneration sets in. This assumes two forms: most often it is a fatty degeneration of the tissue, but calcareous degeneration is quite common, and it may follow upon the fatty change, or occur independently. The fatty degeneration may be attended with softening of the tissue, even to complete liquefaction and the formation of the so-called "atheromatous abscess," the contents of which are not pus, but fatty granules, oil-drops, and crystals of margarine and cholesterine. If the liquefaction is less, a soft yellow patch is produced, and it may be so entirely absent that the yellow area is dry and firm. Lastly.

the fatty granules and *débris* may be absorbed and leave a cicatrix-like depression in the vessel. The thin layer of endothelium and superficial part of the intima over a softened atheromatous patch is very liable to be ruptured; the contents of the "abscess" are then swept away in the blood stream, the larger portions blocking up arterioles or capillaries, but owing to their non-infective nature, not giving rise to any serious consequences. The deep surface of the patch, which is then exposed, forms an "atheromatous ulcer"; its floor always shows fatty or calcareous change.

Calcareous degeneration appears first in the form of fine granules of lime-salts; these then coalesce into flat plates of varying size, and this form has given rise to the name of *laminar calcification*, to distinguish it from the *annular calcification* met with in the middle coat of arteries. These plates sometimes attain a considerable size; the endothelium covering them is apt to be disintegrated and expose sometimes the whole plate, sometimes a sharp edge only. Occasionally, such a plate gets partly or wholly detached, leading to thrombosis or embolism. The causes which determine in some cases fatty change, and in others calcareous degeneration, are not known; heredity may play some part: at any rate, some individuals appear to have a special tendency to calcareous deposit (Fig. 159).

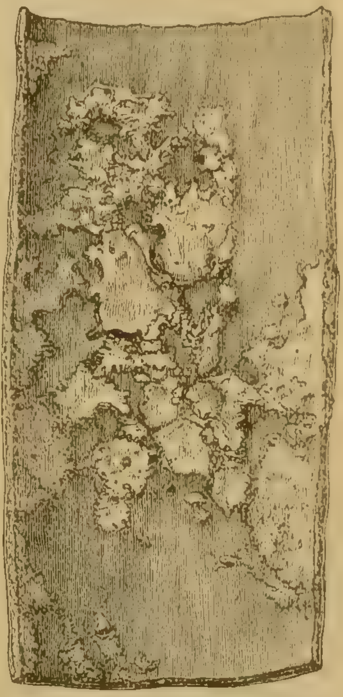

Fig. 159.—Laminar Calcification of the Aorta. (Middlesex Hospital Museum.)

The middle coat of the artery is invaded by the cellular growth in the deeper layers of the intima, and the muscle cells undergo fatty degeneration. It has been suggested that the primary disease is atrophy of the tunica media, and that the changes in the intima are of the nature of compensatory hypertrophy, but most authorities are now agreed in considering the disease as essentially a chronic endarteritis. The outer coat of the diseased artery is the seat of a varying degree of compensatory fibroid thickening, which is greatest where the other coats of the artery have been most destroyed by the disease. It is this thickening of the outer coat which prevents rupture of the artery when an "ulcer" has formed.

Effects.—The most constant effect of atheroma is a *loss of elasticity* in the artery, which entails a loss of force in the circulation, as the systole of the heart is not supplemented by the elastic recoil of the large arteries. The diseased vessel is also liable to *yield* under the pressure of the blood, and atheroma is therefore of great importance in the causation of aneurysm. In the smaller arteries the thickening of the intima partially *occludes the lumen* of the vessel, and in some situations, as in the coronary arteries of the heart, the cerebral arteries, and the arteries of the legs, this may lead to serious consequences from impairment of the blood supply. It is stated that the loss of power in the muscular coat of atheromatous arteries interferes with the due regulation of the local supply of blood, and is one cause of cerebral symptoms in old people with rigid arteries.

Exposed calcareous plates may excite *thrombosis*, and detached portions, as we have seen, may form emboli; either of these conditions may lead to gangrene. Arteries thus diseased are more liable to *rupture* under the influence of injury than are supple, healthy vessels; and when the affection is far advanced it interferes with the repair of injured vessels, and with the distension of arteries which occurs in the establishment of anastomotic circulation. When, from any cause, the force of the arterial circulation is markedly diminished—*e.g.* loss of blood, ligature of a main artery—this disease is very liable to excite coagulation of the feeble stream of blood. Atheroma, therefore, is an exceedingly important condition, and in many ways, directly and indirectly, leads to grave results.

Signs.—Well-marked atheroma can be recognised during life by the elongation and tortuosity of superficial arteries, such as the temporal and brachial, and by the too-marked "locomotion" of the vessel with each pulse-wave; the arteries are also less compressible than normal. The effects of advanced atheroma are also to be sometimes seen in coldness, wasting and diminished function of parts, or in special effects, such as giddiness.

Treatment.—There is no method known whereby any improvement or recovery can be obtained, but when atheroma is recognised, the individual should so regulate his life as to reduce the arterial strain to the minimum, and to sustain the nutrition of his tissues.

5. Syphilitic arteritis.—The disease is best known as it affects the smaller arteries, especially of the brain and other viscera, and in gummata; but it is stated to occur in larger arteries also, and in them to lead to aneurysm. The arteries become thickened, indurated, and narrowed: the lumen may be completely blocked. and the vessel reduced to a fibrous cord. The changes are most constant and most marked in the tunica intima. This becomes enormously thickened. Fine nuclei and cells appear beneath the endothelium, then multiply and enlarge, and at the same time become separated by fine fibre-cells and fibres. As the disease progresses, a tissue like loose granulation tissue is formed, which tends to become more fibrous; new capillaries are developed in it, which communicate with the vasa vasorum. By this growth of the inner coat the lumen of the artery is narrowed, generally distorted, often converted into a mere slit; and it may be completely obliterated in the case of small arteries, either by extension of the growth of the intima, or by the formation of a thrombus. The tunica media is generally unaffected; occasionally it is involved by an extension of the disease in the intima. The tunica adventitia is generally thickened by an addition of fibrous tissue; in rare cases the change is most marked in the outer coat. The disease is chronic, and probably recurrent; it is the cause of thrombosis and local anæmia, and so leads to cerebral softening and to the degenerative changes in a gumma; as already mentioned, it is believed by some to play an important part in the causation of aneurysm. A prolonged course of iodide of potassium is the best means of combating the disease (Fig. 160).

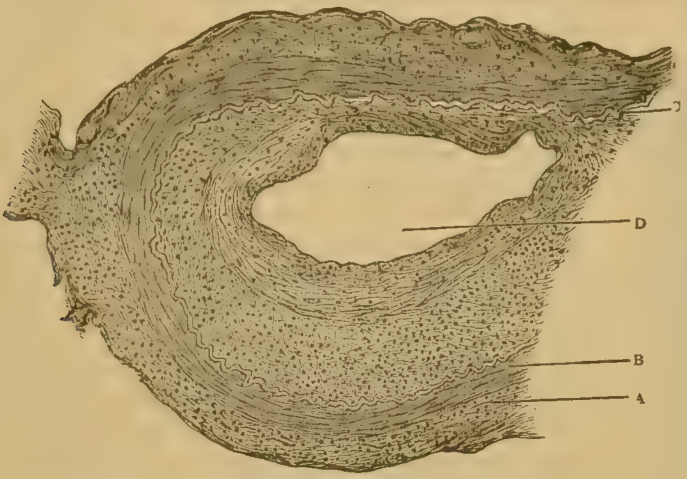

Fig. 160.—Syphilitic Arteritis. (From the Path. Soc. Trans., 1887.)

A, Outer coat; B, middle coat; C, fenestrated membrane of Henle: internal to which is the greatly thickened intima; D, greatly reduced lumen of the artery. (A specimen of Dr. Sharkey's.)

6. Tuberculous arteritis.—The arteries in tissues the seat of tubercular lesions show a change very similar to that in syphilis. The intima is enormously thickened by a fine cell growth, and the other coats are usually the seat of inflammatory change; but the essential and most constant and marked change is the thickening of the intima, which is caused by the tubercle bacilli finding their way into the vessel-wall. The lumen of the vessel may be reduced to a fine slit only, or be wholly obliterated by the growth of the intima.

7. Obliterating arteritis.—This name has been applied to a rare disease, occurring independently of syphilis, tubercle, gout, rheumatism, or other constitutional malady, and independently also of embolism or injury. The cause is unknown; it is most often seen in persons of middle age, but the writer has met with the disease in an otherwise healthy man of nineteen years of age and in a woman of twenty-seven. The disease is believed to commence in the small arteries, and thence to extend into the larger vessels. It is characterised by a great proliferation of the endothelium, which gradually narrows the vessel, until it occludes it; sometimes thrombosis occurs to complete the occlusion. The new tissue—and where present, the blood-clot—organises into fibrous tissue, new vessels passing down into it from the vasa vasorum. The arteries are felt first of all to be firmer than natural with feeble pulsation, and then they become hard, enlarged, and tender pulseless cords, and finally these cords shrink and lose their sensitiveness. The disease is a chronic one, often extending over years; but in some cases, at any rate, it is arrested spontaneously. The parts supplied by the affected arteries are pale and cold, livid on exposure, wasted, and the seat of pain, which may be intense. Dry gangrene of the extremities may occur. When the disease is very severe, the pain is excessive, and slight pyrexia may be produced. It has been met with in both the upper and the lower limb, more often the latter. No special treatment has been found of any avail. While the disease is in progress the part affected should be kept at rest and warmly swathed; anodynes may be required. Afterwards great care must be taken to preserve the anæmic parts from exposure to cold. Amputation has been practised when gangrene has occurred. This disease stands in need of a distinctive name, for other forms of arteritis are equally "obliterating," "proliferating," and "hyperplastic"; for this we must wait, until its cause has been ascertained.

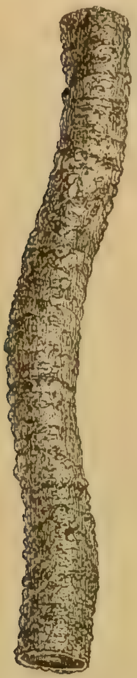
Fig. 161.—Tubular Calcification of an Artery. (Middlesex Hospital Museum.)

Other affections of arteries.—**Periarteritis** is an inflammation of the tunica adventitia and sheath of an artery. As an independent affection it is rare. It is stated to occur in the cerebral vessels, and cause such weakening of their walls as to lead to rupture, and Charcot stated that it is a frequent cause of senile apoplexy. Occurring in the limbs, it occasions swelling in the course of an artery with pain and tenderness, but owing to the disease not affecting the inner coats, no thrombosis occurs; the pulse is not lost, nor is the nutrition of the parts supplied by the artery interfered with.

Calcification of arteries.—Calcification is, as we have seen, a frequent termination of atheroma, large flat plates often being found in the aorta and other large arteries. This form is known as *laminar* or *secondary calcification* (Fig. 159). Calcareous degeneration is also met with as a primary change affecting the middle coat of arteries. The lime-salts are deposited in the muscular fibre cells, and as these run circularly round the arteries, calcareous rings are formed, and this variety is therefore known as *annular* or *primary calcification;* when, by the extension of the degeneration, adjacent rings are welded together, it is known as *tubular calcification* (Fig. 161). This disease is a senile change, it occurs symmetrically, and is often associated with atheroma. Its chief seat is in the small—not the smallest—arteries, but it is common in the brachial, popliteal and femoral trunks, and different portions of these vessels may be the seat of atheroma and of primary calcification. It can be recognised by the rigidity of the artery under the finger, and sometimes by the irregularity that can be felt when the finger is passed along the vessel. By lessening, and then destroying, the elasticity of the arteries, it impedes the circulation through the capillaries, and when the degeneration is advanced, it also narrows the lumen of the affected vessel. The part, therefore, is cold and slowly wastes, and sometimes it is the seat of ill-defined pain. This degeneration has great surgical importance, from three special effects that may follow it:—(1) The intima is liable to become detached, owing to the interference with its nutrition by the rigid barrier between it and the vasa vasorum in the adventitia; thrombosis is then very liable to occur, and as a result of this, gangrene. (2) Under the influence of strain or slight injury, it is believed that the calcified middle coat may snap, and that an aneurysm may form in the thus weakened artery. (3) When from any cause—thrombosis, embolism, pressure, or ligature—a main artery is occluded, if the channels by which the anastomotic circulation have to be carried on are calcified, they are unable to enlarge in response to the call made upon them, and gangrene may result. This affection, therefore, plays a very important part in the causation of senile gangrene and gangrene after wound or ligature of an artery. Portions of *bone* have occasionally been found in arteries; they are said to be formed in the outer coat adjacent to areas of calcification.

Primary fatty degeneration of arteries is a disease of very little, if any, surgical importance. It is met with principally in the aorta and the large arteries, and occurs even in young people—in them, it is said, from anæmia. It appears in the form of bright yellow spots and streaks in the intima, which, under the microscope, are found to be caused by fatty degeneration of the flat subendothelial cells. If very advanced, the fatty degeneration may cause a little softened spot, which may give way into the vessel and form a very superficial erosion, known as *fatty erosion.* It has been suggested that such an erosion may be the starting-point of a dissecting aneurysm.

II. DISEASES OF VEINS.

Thrombosis and phlebitis.—Inflammation of the coats of a vein is called *phlebitis;* when it affects the intima it always causes coagulation of the blood in the vessel, and this is called *thrombosis.* It was at one time held that the presence of a clot in a vein was evidence of previous inflammation of its coats, and every case of venous thrombosis was spoken of as phlebitis. But it has been shown that a thrombus may form in a vein quite independently of phlebitis, although it subsequently excites inflammation of the vein. The two conditions—thrombosis and phlebitis—are therefore very closely associated, but they must be considered separately.

Venous thrombosis.—A blood-clot formed within a vessel during life is known as a *thrombus,* and the process of its formation and the disease it characterises is called *thrombosis*. As we have already seen, thrombi form in arteries as a result of injury and disease, and play an important part in the repair of injured vessels. But they are of very common occurrence in veins, and especially frequent as primary diseased conditions, and for this reason, and from the fact that the disease in veins plays an important part in pyæmia and septicæmia, thrombosis is discussed under the heading of diseases of veins.

Causes.—Fibrin is formed by the union of the fibrinogen of the liquor sanguinis with the fibrino-plastin or paraglobulin of the white corpuscles, under the influence of a fibrin ferment, contained either in the same white blood cells or in certain special small colourless corpuscles, called blood platelets. The setting free of this ferment is an act of death; it occurs when the corpuscles are brought into contact with dead or imperfectly vitalised material, or when, from any other circumstance, the corpuscles lose their vitality. The pathological causes of venous thrombosis may be ranged under two heads as follows:—

(*a*) **Conditions affecting the physiological integrity of the walls of veins.**—These are *injuries*, such as incisions, contusions, lacerations, exposure in a wound, burns, and the action of caustics; *inflammation* of the vein, whether spreading to it from without, or lit up by an irritating thrombus or embolus; *degenerations*, which are much less common in veins than arteries, and are chiefly met with in varices; and the effects upon the veins of *blood stasis and exhaustion*. It is believed that the integrity of the endothelium of a vessel depends upon the rapid and uninterrupted flow of blood through the vessel, and upon the activity of the circulation in the vasa vasorum. In stasis both of these conditions are wanting.

(*b*) **Conditions affecting directly the physiological integrity of the blood,** particularly of its white corpuscles. These are less clearly determined. The experimental introduction of a sufficient quantity of "fibrin-ferment" into the circulation has been shown to cause widespread intravascular coagulation; and a smaller quantity of the "ferment" causes febrile disturbances without thrombosis. It is believed that in septicæmia and pyæmia a liberation of the "fibrin-ferment" occurs, causing fever and the multiple thromboses met with in these diseases. Other blood conditions, such as gout and the result of exhaustion, may have the same effect, and one way in which stasis leads to thrombosis may be by the white corpuscles being deprived of their proper supply of oxygen. *Foreign bodies* in the blood stream, such as a needle or a thread transfixing a vein, emboli, existing thrombi, and micrococci, cause coagulation of the blood by destroying its physiological integrity.

While these may be regarded as the pathological causes of thrombosis, certain clinical causes must be separately considered. Of these, the most important is interference with the circulation of the blood, especially when it amounts to *stasis.* Outside the body, complete rest of the blood retards its coagulation, but during life it is an important cause of thrombosis, owing to its interfering with the perfect vitality of the vessel wall and with the proper interchange of gases in the blood; it also favours the adhesion of the colourless corpuscles to the vessel wall and to one another. A slow venous circulation may be due to a weak heart, to loss of elasticity in the arteries, to dilatation of the veins, or to obstruction to the outflow from the vein, whether caused by a clot in the vessel above, constriction by a ligature or tight bandage, the pressure of a tumour, inflammatory exudation or cicatrix, or to disease of the heart or lungs. Compression of a vein, as of the femoral in long-standing flexion of the hip joint, may be followed by thrombosis. The influence of stasis is shown by the frequency with which coagulation begins in the sinuses behind the valves in a vein, where it is reasonable to suppose that there is least movement in the blood when the whole stream is slowed. In the longitudinal sinus, the clot forms first along the superior surface, at the part farthest removed from the openings of the cerebral veins, where, in conditions of marasmus, the flow of blood is slowest. A sudden alteration in the condition of the circulation may lead to thrombosis. For example, when a patient first gets up and moves about after an illness which has confined him to bed, the resumption of the upright position may be followed by thrombosis in a vein of the leg, especially in a varicose vein.

Exhaustion is a frequent cause of thrombosis. This is often exemplified in the later stages of phthisis and other suppurative diseases, after typhoid and rheumatic fever, and after severe operations. The slowing of the circulation due to the weak action of the heart, and the diminution in the quantity of the blood, the lowered vitality of the vessel walls, and probably also of the white corpuscles themselves, are the cause of the coagulation. When a clot has been produced by some strictly local process, such as a wound, ligature, or ulcer, exhaustion will often lead to its extension for a considerable distance from its starting-point.

Infective blood conditions, such as pyæmia and septicæmia, have already been mentioned. Apart from the debility they induce, and the possible liberation of "fibrin-ferment" they cause, the micrococci circulating in the blood have a powerful influence in exciting coagulation wherever they happen to lodge.

Gout is a frequent cause of thrombosis, but it is not determined whether the process starts in the blood—thrombosis, or as a gouty inflammation of the vein—phlebitis.

Many cases of thrombosis occur in which it is impossible to assign a cause, and these are, for want of exact knowledge, spoken of as *idiopathic* in origin.

Varieties of thrombus.—When blood at rest coagulates, all the corpuscles, both red and white, are enclosed in the meshes of the fibrin, and the clot is red in colour; it is a *red thrombus.* On the other hand, when the blood is in rapid motion, as in the case of a puncture in a vein, or a thread transfixing it, the white corpuscles adhere together, and then break up and lose their outline, and form a greyish granular mass of fibrin, in which an occasional white corpuscle may be recognised; this is a *white thrombus*. As such a clot extends, some of the red corpuscles may be entangled in it, and impart to it a deeper colour, and thus all grades between the white and the red thrombus may be met with. Sometimes a white thrombus grows until it occludes a vein and causes stasis above and below it, and then a red thrombus is formed on each side of the original white thrombus; such a clot is called a *mixed thrombus*. The colour of a recent clot in a vein, therefore, enables us to judge how it has been formed. The term *hyaline thrombus* has been suggested for a white thrombus, in which the fibrin is in the form of structureless hyaline material.

The growth of a thrombus.—An uninfected traumatic thrombus is usually limited to the portion of the vessel that is injured, but it is liable to extend upwards and downwards as far as the next branch, where the rapid flow of blood appears to stop the coagulation. In cases of exhaustion a thrombus, once formed, often extends for a considerable distance along the vein. Where the thrombus is infected there is also a special tendency to its extension, until it is closed in by a layer of uninfected clot.

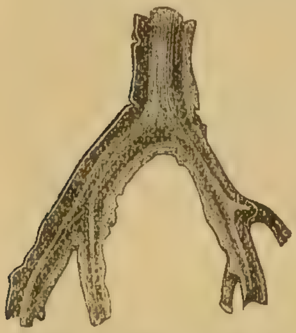

Fig. 162.—Thrombosis of Vena Cava and Iliac Veins.

The changes which occur in a thrombus vary in different cases, and upon them the importance of the condition very largely depends.

(1) *Organisation.*—The process of organisation of a clot in a vein is similar to that already described in an artery. (*See* page 512.) It may convert the vein into a fibrous cord, or the spaces which form in the contracting clot and open into the vein beyond may enlarge and coalesce, and eventually form a continuous channel and restore the patency of the vein. This channel may be placed centrally, the remains of the clot appearing as a circular thickening of the vein, or it may be excentric, the vein appearing greatly thickened on one side.

(2) *Calcification:* the product being known as a *phlebolith.*—Phleboliths are usually small, and are formed most often in clots lying behind the valves of varicose veins, and in the branches of the pelvic plexuses of veins. They may lie free in the channel of a vein, being attached to its wall by a narrow pedicle, or they may mark the site of an occluded vessel. They are composed chiefly of phosphate of lime, with a small quantity of the sulphates of lime and potash, together with about 20 per cent. of proteid matter.

(3) *Red softening, disintegration, absorption.*—A blood-clot may be changed into a greyish-red pulp, the softening commencing

in the centre and gradually involving the whole, until the products are poured into the circulating blood. The same process may occur at the tip of a projecting thrombus, gradually melting it away. In either case the detritus, or products of the softening or disintegration of the clot, may be arrested in the pulmonary capillaries, but being non-infective and unirritating, they give rise to no symptoms, nor any inflammation. The disappearance of a clot in this way, without obvious embolism, is sometimes spoken of as its "absorption," but the term is not then used accurately. In this process the blood-cells do not return to the circulation as living functional corpuscles—the detritus of the clot is dead matter.

(4) *Yellow or infective softening.*—This is the most important change that occurs in thrombi. It is only met with as the result of infection of the clot with septic micrococci, whether these organisms have reached it in the coagulating blood or from the wall of the vein. It is always associated with septic phlebitis, either as cause or effect. It is the result partly of the peptic action of the toxines of the cocci, and partly of the suppurative phlebitis which always accompanies such a change in the thrombus. The clot breaks down into a reddish-yellow pus-like creamy pulp, which consists of granular detritus, pus-cells, and micrococci. If this softening involves the whole clot, the products of it are poured into the circulating blood, and the particles are carried to the lungs and lodge in the arterioles; as they are infected emboli, and contain septic micrococci, they set up suppuration and "secondary abscesses." This process plays a very important part in most cases of pyæmia. A softened septic clot may be shut in by coagulation extending beyond the infected portion of the vein, and, happily, this often occurs in the less intense forms of infection, and so many cases of infected thrombosis end in a local abscess.

(5) *Embolism* may occur from either a portion or the whole of a clot being dislodged, or from softening of the thrombus. Its results depend upon the nature of the embolus, whether simple or infective, and upon its size and place of arrest. It is an accident of extreme danger, and often occurs under slight provocation, and without any warning. The possibility of its occurrence must not be lost sight of in every case of thrombosis, and it may be useful to mention two or three illustrative cases. A man in the out-patient room of the hospital with a thrombus in the saphena vein, slapped the part to show how little regard he paid to it—and fell dead upon the floor—the thrombus had been displaced by his blow and carried to the heart, whose action it at once arrested. A woman, who had been in Middlesex Hospital for six weeks for a fracture of the shaft of the femur, attempted to sit up in bed, and almost immediately died from a thrombus in the profunda vein getting displaced and carried to the pulmonary artery. A woman, who had thrombosis of the pelvic and iliac veins after delivery, when all the acute symptoms had subsided, turned in bed and lifted her infant from the cot by her side, and by this effort she displaced a large thrombus, which lodged in her heart

and caused immediate death. These cases are mentioned merely to indicate the extreme ease with which an embolus may be detached. In many cases, fortunately, the embolus is smaller, and lodges in a branch of the pulmonary artery, and after causing more or less intense dyspnœa with slight hæmoptysis, and perhaps a small area of pneumonia, the patient recovers.

Site of thrombi.—Thrombosis occurs most often in varicose veins of the lower extremity (*see* page 575) and in the pelvic veins. The thrombosis of marasmus is very prone to affect the superior longitudinal sinus. The lateral sinus is often thrombosed as the result of infection from chronic disease of the middle ear, and the profunda femoris vein from septic osteitis of the femur. No vein is exempt.

Effects of thrombosis.—The first effect is obstruction to the venous return of blood, and the result of this varies with the situation of the thrombus. There may be no appreciable result; the channels of the venous circulation are so large and so numerous, that in many situations the blocking up of one of them leads to no serious consequence. In other cases slight and temporary œdema occurs, which passes off as the anastomosing veins enlarge in response to the special need. If a main vein is blocked the œdema is more extensive, involving the whole thickness of the limb, and it is more persistent. In these cases it is very liable to lead to a condition of "solid œdema." Blocking of a deep vein may cause considerable swelling of a limb without any superficial œdema.

The presence of a thrombus in a vein sets up inflammation of the vein—phlebitis. If the thrombus is infective, this inflammation is suppurative and terminates in abscess; if it is non-infective, the inflammation is of a low grade of intensity, and does not end in suppuration.

The occurrence of embolism and pyæmia have been already sufficiently referred to. Lastly, thrombosis often plays an important part in bringing about gangrene.

Symptoms.—The symptoms of thrombosis are the presence of a firm cord-like swelling in the course of a vein, and local œdema. The cylindrical swelling caused by the distended vein is enlarged opposite each valve, but the exact outline of the vein may become more or less obscured by inflammatory swelling around it. The patient experiences a sense of stiffness or tightness in the part, or even acute pain, and it is tender to the touch, particularly when septic suppuration is occurring; occasionally superficial veins are dilated. The temperature at the outset is a little raised; in septic thrombosis it becomes of the septicæmic or pyæmic type. When suppuration occurs there are the usual signs of abscess.

Treatment.—As in phlebitis.

Phlebitis.—The various forms of inflammation of veins are sharply divided into two great classes: the non-suppurative or plastic, and the suppurative or septic.

1. **Plastic phlebitis** is an inflammation of the coats of a vein,

attended with effusion of plastic lymph, and terminating in resolution or in obliteration of the lumen of the vessel.

Causes.—The causes of plastic phlebitis are in the first place injuries of all kinds, and this variety is called *traumatic phlebitis.* The formation in a vein of a non-infective thrombus is a common cause of phlebitis—*thrombo-phlebitis.* Inflammation may extend to a vein from the tissues around it—*phlebitis by extension.* Certain other cases are due to gout—*gouty phlebitis ;* and other cases in which no definite cause can be determined are spoken of as *idiopathic phlebitis.* Heightened blood pressure in the veins produces a chronic inflammatory thickening of their walls, to which the name *phlebo-sclerosis* has been given.

Pathology.—The coats of the vein are affected in various degrees. When the disease is lit up by the presence of a thrombus the change is mainly seen in the inner coat, which is the seat of active proliferation of the endothelial cells, many of which pass into the clot. The middle coat shows but little change ; the outer coat is swelled, its vessels are enlarged, and the number of nuclei and cells in it is increased. In traumatic phlebitis, or phlebitis by extension, the swelling and softening of the outer coat are more marked, owing to a more abundant cell-infiltration ; the middle coat is also invaded by the "plastic lymph," and the proliferation of the endothelium is a marked feature. In all cases, a thrombus forms on the altered intima. Generally the vessel undergoes the same changes as occur in the permanent closure of a wounded artery (page 512), but in some cases the inflammation subsides, and the vein is restored to its former state. We have already mentioned that lateral wounds of veins are far less often followed by occlusion of the vessel than are similar injuries to arteries. In some cases this is due to the small extent of clot formed, but more often to the dilatation and coalescence of the spaces in the clot. (*See* page 511.) *Traumatic phlebitis* is limited in extent to the injured part of the vein, and has no tendency to spread. *Thrombo-phlebitis* also corresponds in extent with the clot within the vessel. We have seen that thrombi grow, and may sometimes extend far up a vein, until an active stream of blood is encountered, and so it happens that while thrombo-phlebitis is usually limited in extent, in some cases it spreads for some distance along a vein. *Gouty phlebitis* is usually symmetrical, often subsides in one vein to light up in another, and has a tendency to recur and to attack the same vein more than once. A very *chronic form* of plastic phlebitis, which slowly spreads along a vein in the direction of its current, converting it into a hard impervious cord, is sometimes met with. Phlebitis may attack any vein, but it is most common in those of the lower limb, and particularly in the saphena vein.

Symptoms.—The symptoms are primarily those of thrombosis—a firm knotted cord is felt in the position of a vein, with œdema of the tissues drained by it. The inflamed vein is tender and painful ; it is swelled, with an indistinct outline, owing to the inflammatory exudation in its sheath, and if the vein is superficial the skin over it is reddened, and often it pits on pressure. There may be slight febrile disturbance.

Gouty phlebitis may complicate an ordinary attack of gouty arthritis, or may occur independently; the areas of inflammation are often multiple, and the disease appears to be primarily an inflammation of the vein-wall, and secondarily thrombosis; the pain is often very severe, and it may last after the other acute symptoms have subsided. In chronic obliterative phlebitis there is very little pain, but the veins are felt as hard impervious cords, and this condition slowly spreads up along the vein. In phlebo-sclerosis of superficial veins the vessels stand out like tendons and roll under the finger, but the blood current in them is not obliterated.

Treatment.—A patient with phlebitis should be placed in bed, with the affected part at rest and evenly supported and raised on a pillow to favour the venous return. All sudden movement of the part, friction, or careless handling must be most carefully avoided, lest embolism occur, and owing to this danger the treatment must be continued after all acute symptoms have passed away. Heat relieves the pain and favours resolution, but poultices and fomentations are objectionable on account of the disturbance of the part attending their renewal. The best local application is a thick layer of cotton wool held in place by a "many-tailed" flannel bandage loosely adjusted; some surgeons recommend that the inflamed part should be thickly smeared with equal parts of extract of belladonna and glycerine under the wool. This dressing should only be disturbed at long intervals, and it has the merit of protecting the clot in the vein from the fingers of patient, surgeon and nurse.

The bowels should be unloaded, if necessary, by a saline purge, and the diet must be suitably regulated. In gouty phlebitis it should be restricted to fish, light farinaceous foods, and a moderate amount of milk; alcohol, sweets, meat, and butter are best withheld. In marasmic or "post-febrile" thrombosis, on the other hand, the diet must be liberal—as liberal as the patient's powers of digestion permit. It used to be thought that many cases of thrombosis and phlebitis were due to an excess of fibrin in the blood, and that to combat this condition a lowering diet was in all cases indicated, but this view is incorrect. With the same view, alkalies and iodide of potassium were generally prescribed "to keep the fibrin liquid"; in gouty phlebitis alkalies are undoubtedly indicated, and if the symptoms are very acute, colchicum may be added; but in marasmic thrombosis, tonics and cod-liver oil should be given; in traumatic phlebitis no drug need be exhibited; if there is any reason to suspect a syphilitic taint, iodide of potassium may do good. When the inflammation has subsided, if the œdema persists, a bandage should be worn, and for threatened "solid œdema," gentle upward frictions and hot douches are of service. In cases of slowly-spreading chronic phlebitis an attempt should be made to stop the process by excising a short portion of the vein above the diseased part.

2. **Suppurative phlebitis.**—This form of the disease is sometimes called "spreading phlebitis," but it is not a good name for it, as it does not always spread, and spreading phlebitis, as we have seen, may be adhesive or plastic in nature. It has also been called thrombophlebitis, but this is too narrow a use of the term, for thrombosis and phlebitis are associated in other conditions.

Causes.—The cause of suppurative phlebitis is infection of the wall of a vein with pyogenic organisms, chiefly micrococci. The infective material may be in the circulating blood, as in cases of puerperal phlebitis remote from the uterus, or may reach the vein in the blood returning from a focus of suppuration, as a wound, "acute necrosis," "facial carbuncle," or middle-ear disease. Infective inflammation may spread to a vein from the surrounding tissues, or a portion of a broken-down infective thrombus may lodge in another vein and set up inflammation—embolism. The poison, therefore, in some cases acts primarily upon the vein, setting up phlebitis, which excites thrombosis; and in others the poisoned blood coagulates within the vein—thrombosis—and the infection spreads thence to the vein, setting up inflammation. The disease occurs most often in connection with septic wounds, diffuse cellulitis, and "acute necrosis," and it plays an important part in most cases of septicæmia and pyæmia, as the poison of these diseases generally enters through the veins. In the secondary phlebitis of septicæmia, some local condition may be at work to determine the part attacked. A patient lately under the writer's care illustrates this: a woman, who had been confined a month before, was admitted to the hospital with suppurating phlebitis over the right shin, at the back of the right thigh, and in the two saphena veins in the right leg. The question arose, why the septic poison had attacked these particular veins only? We learned that the inflammation immediately followed the patient's going about after her confinement, and it appeared probable that the slight venous engorgement caused by the change of posture, or varicosity of the veins, or possibly some long-standing degeneration of the veins, had been the predisposing or localising cause.

Pathology.—The changes in the coats of the vein are those common to suppurative inflammation. The thrombus undergoes "yellow softening," and the products eventually blend with those of the disintegrated vessel itself. Septic micro-organisms are always found, both in the softened thrombus and in the inflamed vein. The infection spreads to the tissues around the vein, and in some cases large abscesses are thus formed. Very commonly, septic emboli pass into the blood stream from the softened thrombus, and each one becomes the starting-point of a "secondary abscess." Toxic products of the micro-organisms are also poured into the blood, or absorbed from the inflamed tissues; and so septic phlebitis leads to septicæmia and pyæmia. The infective nature of this form of phlebitis imparts to it a tendency to spread along the vein in the direction of the current of blood, a tendency so marked that it gave rise to the name "spreading phlebitis."

Symptoms.—When a superficial vein is attacked, it is felt as a solid cord with "knots" opposite the valves; the vein is tender, and its outline is quickly lost by inflammatory swelling around it, the skin over it becomes reddened, and later, fluctuation and all the signs of acute abscess or abscesses are recognised. The first onset is accompanied with a sense of stiffness in the part, and this increases to smarting and throbbing pain. The disease may be limited in area, or it may rapidly creep along the vein. The parts drained by the vein may be œdematous. With these local symptoms there is fever, often of high grade with rapid soft pulse, dry tongue, fetid diarrhœa, and low delirium; or a succession of rigors may mark the development of pyæmia. In some cases the septic poisoning is slight, and the fever is less intense, and subsides when the abscess bursts or is opened. When attacking a small deep vein, the only signs of the disease may be those of the septicæmia or pyæmia, to which it gives rise. Abscesses occurring in the course of septic inflammations are in many cases the result of suppurative phlebitis, and some of the secondary abscesses of pyæmia are of this nature.

Prognosis.—The prognosis of this disease is very grave. It varies to some extent with the position of the vein attacked, and with the virulence of the infection and the power of resistance in the patient's tissues. It largely depends upon the practicability of thorough removal or destruction of the infective material before it has infected the mass of the blood.

Treatment.—The prevention of this disease by perfect asepsis is one of the great advances of modern surgery. As soon as the disease is recognised, the inflamed vein should be freely laid open, all the infected thrombus removed, the whole cavity thoroughly cleansed, as by swabbing it well with pure carbolic acid, and the vein stuffed with an antiseptic gauze. Abscesses occurring in the course of the disease should be opened early, and the cavities thoroughly cleansed. To prevent blood-poisoning the vein has been divided and tied on the cardiac side of the clot. In some cases amputation may be the only means of preventing or arresting general infection. The constitutional treatment is that of septicæmia.

III. VARICOSE VEINS.

A permanent pathological dilatation of a vein is called a *varix*, and a vein so affected is said to be *varicose*, this name being given to the disease because of the tortuosity of such veins. A varicose vein must be distinguished from a merely engorged vein, and the difference is twofold, for the vein is enlarged beyond any mere physiological distension, and the enlargement is permanent. It is, in fact, an overgrown vein.

Pathology.—As to *situation*, the disease is most common in the veins of the lower extremity, but it is also frequently met with in the spermatic cord (varicocele), in the labia, and around the anus (hæmorrhoids), less often in the neck, upper limbs, and trunk. Very few veins are wholly exempt from it. It is chiefly observed as a disease of the superficial veins, but dissection shows that it affects very often the intermuscular and intramuscular veins also; but the main deep veins of the limbs, with the exception of the posterior tibial veins, are generally free. It has been met with in the internal jugular vein. The disease varies very greatly in its extent and distribution. It may be limited to one or more of the main subcutaneous veins, as either saphena, or to a limited portion of it; or, leaving these veins free, it may involve only some one or more branches opening into them, or some of the small venous radicles in the skin; while in some cases all the subcutaneous veins of a part appear to be affected. It is stated that a superficial varix often commences at the point where a deep vein communicates with a superficial one, and that the enlargement is apt to be most marked at such situations.

A varicose vein is not only enlarged transversely, but also in length. This elongation of the vein is often very great and more marked than the lateral dilatation; and as the ends of the vein are fixed points, it causes great sinuosity. It is not uncommon to find a varix three times as long as the healthy vein (Fig. 163). In some cases, however, the elongation of the vein is very slight. For example, the saphena vein may be greatly enlarged from the groin to the ankle, and may stand out under the skin as a straight tube, with perhaps only one short convolution near the knee. The lateral dilatation differs not only in degree in different cases, but in the same varix, producing often a marked pouching of the wall of the vein, and so adding to its varicosity. These pouches may, in large veins, attain to the size of a large cherry or a medlar. The best examples of them occur at the upper end of the internal saphena vein, where they form what may well be called "venous cysts" (Fig. 164).

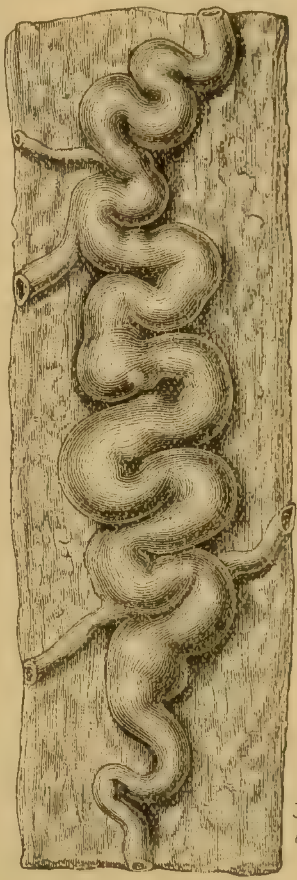

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Fig. 163.—A Varicose Vein. (Middlesex Hospital Museum.)

The wall of a varix is notably thickened, so that when cut across the vein gapes like a divided artery. The intima is thickened and shows longitudinal striæ. Sometimes it is atheromatous, or even calcified. The thickening of the intima may be general, or especially marked at one part of the circumference of the vein (Fig. 165). It probably has an important influence in the production of thrombosis.

In the middle coat there is an increase in both the muscular and the fibrous elements, especially the latter. The outer coat is also thickened, but to a less degree. The sheath of the vein is condensed, and the convolutions of a tortuous vein are often united together, and to the overlying skin, by slender but firm cords and bands of fibrous tissue. The valves of varices do not undergo compensatory enlargement, but, on the contrary, are often shrunken, or present in the form of cords, which may be adherent by one end only, or the valves may be entirely wanting. While the coats of a varix are generally thickened, it is very important to remember that localised thinning is a very frequent occurrence, particularly in the pouches. It appears to occur most markedly in certain individuals, and it is most frequent in the veins of the skin and the smaller subcutaneous veins. This thinning may be entirely absent.

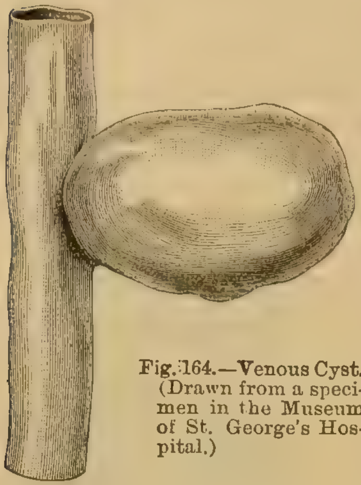

Fig. 164.—Venous Cyst. (Drawn from a specimen in the Museum of St. George's Hospital.)

The general view of the nature of varix is that it is the result of over-distension of the vein and that the changes in the wall are all the result of increased venous pressure. There is much reason to think, however, that the disease is more allied to a true venous overgrowth.

Ætiology.—The usual view is that varix results from a disturbance in the normal relation between the intravenous pressure and the resistance of the vein wall. This may be either an increase in the blood pressure, or a diminution in the tone of the vessel. The *weakness of the vein wall* may be an inherited condition, or the result of some occult error in development, or it may result from absorption of surrounding structures depriving the vessel of its wonted support, or from inflammatory changes in the outer coat of a vein, leading to its softening.

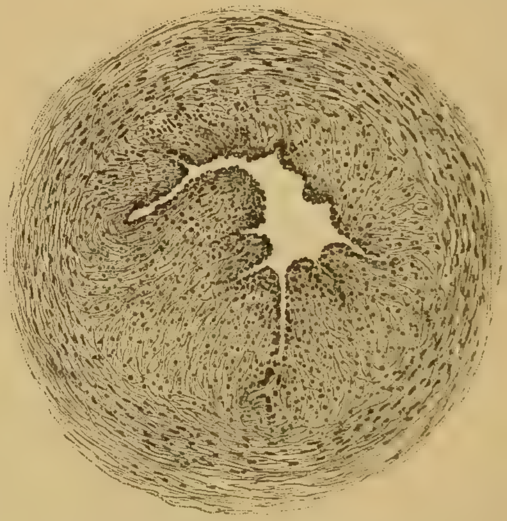

Fig. 165.—Transverse Section of a Varicose Vein showing great and irregular thickening of the inner coat.

It is generally held that *increased intravenous pressure* is the more important factor. This increased pressure may be brought about by several causes. It may be due to *general venous obstruction* from chronic heart or lung disease, or to local obstruction from the pressure of a tumour, as the pregnant uterus, or of an ill-fitting truss, or of a tight garter. It is believed that defect in the *valves* of a vein may have the same result. The valves are of use in breaking up the veins into segments, each of which is to some extent relieved from the weight of the column of blood above it. When a valve is absent or useless by over-distension of the vein, or rupture of the valve, the segment below loses the relief it would otherwise obtain and it may yield under the extra strain thrown upon it. The *increased supply of blood* to a part, which occurs in inflammation, chronic ulceration, or prolonged muscular action, also raises the intravenous pressure. *Gravity*, too, may play an important part, for although the column of blood in the veins of the lower limb is supported by that in the arteries, yet the pressure of the blood upon the vessels is increased by gravity and is greatest at the lower end of the column. *Occupations* that entail prolonged standing or walking are believed to predispose to varix. This is attributed partly to the influence of gravity, partly to the compression of the deep veins by the contracted muscles forcing more blood into the superficial vessels, and partly to the additional blood supply attending prolonged muscular action.

Age and sex.—Varicose veins are rare in children. They most often develop between the ages of puberty and full manhood, but many cases are not noticed until after thirty years of age. The disease is probably more common in men than women; but, owing to the frequency with which pregnancy increases the fulness of, and the pain in, the veins, women apply for treatment more frequently than men.

The influence of *heredity* is marked. In some cases every member of a family is affected and it is seen in two or three successive generations; and not only so, but the affection will sometimes present the same local characters in several members of a family, the same vein or the same part of a vein being attacked. The disease occurs in the robust and well-developed more often than in the puny and weakly, in those who lead a sedentary occupation, as well as in the active. It attacks all classes and, as we have seen, it comes on most often during the later stages of growth, when the organism is reaching maturity, and not when the vessels are becoming worn out by the strain of life. These facts render it exceedingly probable that the real cause of varix is an inherited tendency to over-development of veins, or perhaps to some defect in the veins. The upright position of man appears to be an important factor, for the disease is vastly more common in the lower limbs than elsewhere; and we may therefore regard varix as the result of the special strain thrown upon the veins of the lower limbs by the upright position of men influencing and transmitted through countless generations.

Constipation has been thought to lead to varix by the loaded colon pressing upon the iliac veins. There is no sufficient evidence of this. Statistics on the point are misleading, for constipation and varix being both of them of great frequency, it is certain that a large number of varicose patients will also exhibit constipation. Most aggravated constipation, extending over years, certainly fails to produce varix in a large number of individuals; and in cases of obstruction, where the colon is most distended and must press most upon the iliac veins—cases of cancer of the rectum, for example—varix does not arise.

It is important to bear in mind that the conditions which reveal varicose veins are by no means necessarily those that induce them. Anything which produces fulness of the venous system not only makes the large veins evident to sight, but the distension of the dilated veins may cause pain. It is this fact which leads patients, and surgeons, too, to attribute too great an ætiological importance to standing or exercise, to pregnancy or constipation. In many cases these are only the conditions which lead to the varix being noticed.

Effects.—It is commonly held that the varicose dilatation of a vein impedes the return of blood along it and leads to chronic congestion and œdema of the parts drained by it, while these in turn induce eczema, ulceration, and chronic induration of the skin and subcutaneous tissue. The frequent association of these latter conditions with varicose veins is a fact, but that they are related as cause and effect is highly improbable. Even in cases of extreme and long-continued varix no one of these phenomena may be observed, and they are all of them very often met with apart from varix. It is also interesting to note that varix in the thigh and varix of the main subcutaneous veins, even when greatly developed, is not associated with these complications nearly so often as varix in the leg and varicosities of the smaller subcutaneous veins. Varix of the spermatic veins certainly does not cause chronic thickening of the testicle; but if any effect at all is produced on this organ, it is at the most a slight softening. Mere dilatation of a vein slows the current of blood along it, but it offers no obstruction to the flow of blood into it from tributaries; on the contrary, it favours it. The lack of efficient valves in varices, which would otherwise tell injuriously, is generally compensated by the extreme tortuosity of the enlarged veins, the numerous and abrupt curves in the channel acting like valves; where a vein is greatly enlarged transversely without these convolutions due to its increased length, the effect upon the circulation is liable to be more marked. Varix is sometimes attended with a thickening and pigmentation of the skin over it: more often, however, the skin is thinned by the constant pressure; and when, as may happen, this is combined with a thinning of the coats of the vein, the vessel is liable to burst externally, either spontaneously, or as a result of strain, or of an injury, or ulcer. The thinning of a vein chiefly occurs in saccular dilatations of its walls.

Symptoms.—An extreme degree of varix may exist without giving rise to any subjective symptom. More usually the patient experiences a sense of fulness, or an aching pain in the part, on standing or after long walking; this pain is quickly relieved by raising the limb. Deep varix may cause cramp-like pains as well as a sense of fulness and tension. The superficial enlarged veins are readily recognised; often their blue colour is visible, and their tortuous outline is plainly seen. By raising the part the large veins are emptied and are hardly visible or palpable; by depressing the part they become fuller. In large varices there is often an impulse on coughing felt as far down the limb as below the knee. In the skin the dilated veins show as blue lines arranged in a stellate manner and these may exist alone, or in combination with, and over, deeper varices. The great dilatations of veins forming venous cysts are recognised by occurring in the course of a vein, by their rounded outline, softness, and compressibility, and by their emptying and filling under the influence of position; they are usually met with at the upper end of the internal saphena vein and have a distinct impulse on coughing, which sometimes leads to their being mistaken for hernia.

Complications. 1. **Hæmorrhage.**—Varices may be wounded, may burst under the influence of strain or long-continued pressure, or may be opened by ulceration; in the majority of cases of so-called "bursting of a vein," the hæmorrhage is due to an ulcer having opened into it. When from any of these causes a varicose vein is opened, the loss of blood is very rapid; not only because the vein is enlarged and does not collapse, but chiefly because the valves being lost the blood flows both from above and below. If the patient continues standing or sitting with the leg hanging down, many pints of blood may be lost, and death ensue in a short time. Happily, this serious hæmorrhage is most easily controlled; the patient should immediately lie flat down and have the affected leg well raised: this will at once arrest the bleeding; a pad should then be fixed over the wounded vein by a bandage carried from the root of the toes to the knee. From ignorance of this simple treatment many a life has been lost, and many patients have been permanently crippled by incurable anæmia.

2. **Thrombosis** is a frequent and a serious complication of varices, much more often met with in the leg, thigh, and anus than in the spermatic veins. The coagulation of the blood may be due to its slow flow over the altered inner coat, or to injury—and the mere prominence of varicose veins exposes them to injury; at the anus the enlarged veins are liable to be bruised by the passage of a large hard motion, or over-distended by straining efforts.

3. **Chronic eczema** is a frequent complication of varicose veins and, as already stated, it and (4) *chronic induration* and (5) *chronic ulcer* of the leg have been commonly regarded as resulting from varix, and the term *varicose ulcer* has been given to an ulcer in the middle of a patch of thickened adherent skin with varicose veins leading from it. The only way in which a varix certainly leads to an ulcer is when it "bursts," and primary healing of the wound is not obtained. The benefit to a chronic ulcer, often following

operation upon an associated varix, is due partly to the rest in the raised position this treatment necessitates, partly to the influence of the incision in draining the part, and partly to the asepsis secured.

Treatment.—In many cases no treatment is required, for no discomfort is caused by the disease. For the majority of other instances, **palliative treatment** only is indicated. This consists in supporting the enlarged veins by a carefully-applied bandage or well-fitting elastic stocking, in improving the patient's general health by overcoming constipation, or by the exhibition of tonics, or digitalis where these are indicated. Prolonged standing and straining efforts should be forbidden; but exercise, short of fatigue, should be taken: patients should not be made chronic invalids on account of varicose veins.

The **curative treatment** consists in obliterating or removing the diseased vessels. It is indicated in certain cases only, particularly when the patient is in good health, not past middle life, where the varix is limited in area, and where it is a source of pain to the patient. A vein may be operated on when it leads up from a chronic ulcer or leads up to a venous cyst. Three modes of radical cure are now chiefly practised.

(1) *Acupressure.*—By passing a hare-lip pin beneath the vein, placing a piece of gum bougie over the vessel, and then throwing a silk thread in a figure-of-8 over the ends of the pin, the walls of the vessel can be compressed, and a certain amount of traumatic phlebitis excited, which may lead to the occlusion of the vessel. One or many pins are employed as may be required: they should be withdrawn in about a week, unless marked irritation is excited earlier, and they may be left longer if the local irritation is very trivial. If in passing the pin the vein is punctured, as shown by the free escape of venous blood, the pin should be withdrawn and passed more deeply. This operation often fails to obliterate the vein. If two pins are introduced close together, and then the vein divided subcutaneously, the result is rendered more certain. This method has long been practised, but it is far inferior to either the ligature or incision, and has nothing to commend it to the surgeon.

(2) *Ligature.*—An incision about three-quarters of an inch long is made down to the vein, and an aneurysm needle is carefully passed round the vessel, threaded, and withdrawn. The ligature may be of aseptic silk or chromic catgut; it may be single or double, the vein then being divided between the two threads. A single suture closes the skin wound. This method is reliable in that it obliterates the vein, but the obliteration is usually very limited in extent. It is to be employed in cases of numerous limited varices, or for the obliteration of a vein leading from an ulcer, or up to a "venous cyst." It is sometimes modified by excising a short length of vein—say up to an inch—between two ligatures. The numerous branches and communications opening into the affected veins explains the limited obliteration produced by the ligature.

(3) *Excision.*—This is the operation which gives the best results and is to be employed in the majority of cases. An incision is made along the varix to be removed, the vein is carefully exposed, and a double ligature is tied at each end, and the vein divided between. The vessel is then raised from its bed, and each branch tied or seized in forceps before it is divided. The skin incision is carefully closed with a fine suture. This operation is rendered at times a little troublesome from the great length of the tortuous veins and the number of branches opening into them, but it gives the most satisfactory results. It has been modified by exposing the varix through an inch-long incision, freeing a short length of it, and then seizing this in torsion forceps, and slowly twisting the vein, so as to draw out a considerable length of the vein. Sometimes this answers admirably, but at other times the divided ends of the veins bleed, and some trouble is given to find and secure the bleeding points.

Of course all operations upon veins should be performed only with thorough aseptic precautions. It is a fact of great interest that excision of varicose veins in well-selected cases is followed by most satisfactory results—by cure. The patient's pain and inability to stand long, to undergo fatigue, or to bear exposure to heat, are entirely removed. But not only so, in most cases there is no fresh development of varicose veins nor any sign of injury from obstruction to the venous return; this shows not only how abundantly free the provision for the return of blood is, but also that varix is not simply the effect of over-distension of veins. In some cases new varices do develop after operation; but this is to be considered as the natural progress of the particular case, for it is often observed without any operative or other treatment. If a case has long been stationary without any new varices appearing, operation will not be followed by such event; but where new varices have recently appeared, the removal of those visible may be followed by the development of others: not, however, as the consequence of the operation.

IV. ANGEIOMATA.

Varieties.—An angeioma is a tumour composed mainly or exclusively of blood-vessels; some of these vessels are newly formed, others are pre-existing ones more or less greatly dilated. The vessels may be arteries, veins, or capillaries, and scattered among them may be enlarged lymphatics. Arterial angeioma is more often called *cirsoid aneurysm;* capillary and venous angeiomata are commonly called *nævi*, or "*mother's marks.*" It is common to find two or more of these varieties associated together, and such cases are then classified in accordance with the predominant character of the tumour. (*See also* Article XXXVII., on Diseases of the Head, Vol. II.)

Arterial varix; cirsoid aneurysm; plexiform angeioma.—These names are applied to various conditions of dilated and elongated arteries. When a single large artery is lengthened, tortuous, and pouched like a varicose vein, it is known as an *arterial varix.* When this condition affects several arteries, forming a tumour composed of a congeries of enlarged arteries, it is called a *cirsoid aneurysm.* When the condition of vascular dilatation has spread from the arteries to the capillaries and veins, it is known as *aneurysm by anastomosis* or *plexiform angeioma.* In different cases the exact condition of the vessels varies considerably.

Seat and causes.—The disease, in any of its varieties, is a rare one. It may be met with in any part of the body, but its most frequent seat is in the scalp, affecting the superficial temporal, posterior auricular, or occipital arteries. It occurs also in the orbit, on the face, on the trunk and limbs, and even in internal organs, such as the liver. Sutton has seen a large one in the perineum; "the corpus spongiosum was surrounded by a number of arteries as large as the coronary branches of the facial, and veins as big as the cephalic." Erichsen observed cases on the side of the chest, nates, and foot, and others involving the hand or the upper limb extensively are recorded. Such cases, however, are very rare, and the disease has been chiefly met with in the scalp.

In some cases it follows an injury, such as a cut, bruise, or burn, and, on this account, has been considered inflammatory in nature. In other cases it has supervened upon a congenital erectile tumour, or venous nævus, developing spontaneously—sometimes at puberty—or as a result of injury. Like venous varix the disease most often commences between puberty and thirty years of age.

Pathological changes.—The arteries are considerably dilated, pouched, and greatly convoluted. Their walls are very thin, chiefly owing to the atrophy of the middle coat. They may lie in parallel convolutions or be arranged most irregularly, and connected with them there may be enlarged and varicose veins. The arteries leading to a cirsoid aneurysm are varicose, and the disease tends to spread, both centrally along the arteries feeding it, and towards the capillaries. The skin covering the angeioma and the other soft tissues may be pigmented, thickened, and spongy, or thinned and the seat of ulcers, which, by extending into the arteries, occasion very dangerous hæmorrhages. Subjacent bone may be grooved, or even perforated, by the enlarged vessels.

Symptoms and course.—*Arterial varix* occurs as a single enlarged tortuous artery, which can be traced in the scalp or other part; the vessel is compressible and the seat of strong pulsation.

Cirsoid aneurysm occurs as an ill-defined pulsating swelling, in which tortuous vessels can sometimes be distinguished. The swelling is soft and compressible; its pulsation is forcible and a distinct thrill is often felt; usually there is a very loud bruit of a rasping, cooing, or musical character, and the patient is sometimes conscious of the bruit. Dilated and tortuous arteries can generally be traced leading to the tumour and giving it an indistinct outline; very enlarged veins may also be traced passing from it. By compression of these arteries the pulsation is lessened, and the thrill may be lost, but as a rule, the arteries feeding the "aneurysm" are so numerous, that digital compression of those felt does not entirely arrest the pulsation. The skin over the swelling is hotter than natural and has a dull bluish colour; it may be thin, or thickened and pigmented, or with an abundant growth of hair.

The tumour may remain stationary and cause little or no inconvenience; in very rare instances it has been known to undergo spontaneous cure. More often it steadily, and even rapidly, enlarges, involving more and more vessels. If the skin over it ulcerates, the bleedings which occur gradually exhaust the patient.

The **diagnosis** is usually very easy. From common *aneurysm* it is distinguished by its position, irregular outline, compressibility, and by the enlargement of the vessels feeding it. The character of the bruit, and the effects of compressing the main artery of the part, differ in the two cases. Cirsoid aneurysm following upon an injury might be mistaken for a *varicose aneurysm*, as the bruit and thrill, and the enlargement of many vessels, are somewhat alike in the two cases; but the tumour formed by a cirsoid aneurysm is less well-defined and its pulsation is not arrested by the compression of a single arterial trunk.

From *aneurysmal varix* a cirsoid aneurysm is distinguished by noting that the tumour is arterial, not venous, that it is connected with several arteries, and that its pulsation is not controlled by compression of a single arterial trunk.

Treatment.—Owing to the ill-defined character of the tumour, its tendency to spread and involve new vessels, and still more to the fact that the aneurysm is fed by many arteries, so that cutting off the supply of blood from one, or even several, sources does not starve it, the treatment of cirsoid aneurysm is beset with special difficulty and often fails altogether. In some cases, after temporary improvement—mistaken for "cure"—the tumour has developed again. Therefore if the aneurysm is not increasing in size, is not ulcerating, and is not causing grave distress to the patient, the surgeon should be content to protect it with a metal or leather covering, if in a part exposed to injury, and should not undertake any operation for it. If, however, the tumour is growing, or the skin over it is ulcerating and threatens to open a vessel, or if hæmorrhage has already occurred, an attempt to cure the disease must be made, and the surgeon must choose one of the following methods, according to the nature of the particular case.

(1) *Excision of the angeioma.*—The operator must be very careful to cut wide of the diseased vessels, and to tie each artery as it is divided. Neglect of either of these precautions may lead to frightful hæmorrhage. This method is applicable to cases of limited extent and fairly circumscribed in character.

(2) *Ligature of the angeioma* is preferred by some to its excision. It is unattended with the risk of hæmorrhage, if the ligatures are carefully passed beyond the tumour. It may be followed by recurrence of the growth and in this respect is less reliable than excision. It

may be used in the same cases as excision and in some where excision cannot be practised.

(3) *Ligature of the feeding vessels* alone has never yet been successful. When combined with division of the soft parts between the ligatured arteries, it has, in a few cases, effected a cure. It generally fails, owing to the presence of arteries entering the aneurysm on its deep surface. It may be employed in cases where digital compression of the arteries to be tied entirely arrests the pulsation in the swelling.

(4) *Ligature of the main artery of the part.*—A cirsoid aneurysm of the scalp, too large and diffuse for excision or ligature, may be treated by simultaneous ligature of the two external carotid arteries, combined, if necessary, with ligature of the frontal and supraorbital branches of the ophthalmic artery. The common carotid artery should not be ligatured for such a case.

(5) *Electrolysis* is strongly recommended by some authorities and has been advocated as "the only justifiable method of treatment." A strong current of from 150 to 200 milliampères is required. The needles passed into the vessels are attached to the positive pole, and a large surface negative electrode is placed in contact with the back or some other indifferent part of the body. The operation generally has to be repeated. An anæsthetic is required.

(6) *Injection of perchloride of iron* has been practised, especially by Continental surgeons, with considerable success. A temporary ligature is thrown around the tumour while the coagulating fluid is injected and until the clot is firm.

(7) *Amputation* has been called for in cases of hæmorrhage from cirsoid aneurysm of a limb.

Nævus.—Nævi are of two kinds, "simple" and "cavernous."

A **simple nævus** is a collection of minute vessels bound together by a small amount of connective tissue. If the vessels are arterioles, the nævus is of a bright red colour, and is called "arterial." If they are venules, it is of a blue colour, and is called "venous." Intermediate tints between the two extremes are met with. Simple nævi are sometimes called "capillary."

A **cavernous nævus** or **erectile tumour**, sometimes called also a *venous nævus*, is formed of a series of intercommunicating spaces lined with endothelium similar to that of veins, embedded in fibrous tissue, and fat. Small arteries open into these spaces, and larger veins carry the blood away. The blood spaces are not surrounded by proper vessel-walls. A thin fibrous capsule often encloses the nævus.

Ætiology.—Nothing is definitely known of the cause of nævus. Many nævi are congenital; many others are first noticed shortly after birth. Others appear to originate about puberty and in some instances their development follows upon an injury.

Distribution and varieties.—*Simple nævi* are chiefly met with in the superficial layers of the skin, and particularly on the scalp, face, back, and chest. They are also found on mucous membranes—the conjunctiva, lip, tongue, and labium. They are often associated with a cavernous nævus in the deeper tissues. If the dilated capillaries form but a very thin layer in the papillary part of the skin, it is commonly called a *port wine stain* and these "stains" often cover a large area of skin, even to the extent of one half of the entire body. Occurring in the form of stellate groups of dilated venules, they are called *phlebiectases;* and these are most commonly met with in the skin of the thigh or leg, and particularly in adults. They occur, however, on the face and other parts, and are sometimes seen in the skin close to a nævus either "simple" or "cavernous."

Cavernous nœvi develop chiefly in and beneath the skin and mucous membranes, and occur most commonly in the scalp, lips, cheeks, on the trunk, and the female genitals. They are also met with in the breast, tongue, liver, rectum, and œsophagus. In the liver they are believed to develop late in life.

Clinical characters.—Nævi are often multiple and, as already stated, the two forms are often associated.

Simple nœvi appear as minute bright red or darker specks in the skin, or as large growths, which project slightly from the surface of the healthy skin, and may be a little uneven on the surface. If of large size, the surface may be felt to be hotter than that of the surrounding skin. The colour may be lessened by pressure, but returns very quickly. "Port wine stains" are of varying hue. Some are only slightly purple; others of a deep purple colour.

Cavernous nœvi form soft, easily compressible tumours, often of irregularly lobulated or tortuous outline, which become full and tense under the influence of crying, coughing, or straining. When of large size they may show a tendency to become pedunculated. Beneath mucous membrane or thin skin they show a purple colour, which is intense when the growth implicates the skin itself. Large veins may often be seen coursing from the tumour and the skin over it may be the seat of a "simple nævus." In some cases the blood spaces or enlarged veins are separated by a considerable amount of healthy skin or other tissue, and these cases pass by insensible degrees into a condition of merely enlarged veins indistinguishable from ordinary varices.

Beyond the fact of disfigurement, nævi rarely occasion any ill effects. If cut they bleed freely, but the hæmorrhage is easily arrested by pressure. They may remain stationary throughout life, or may steadily increase, or, having remained stationary for a time, may suddenly take on an active growth, perhaps from the effects of injury or the stimulus of puberty. Nævi may become more prominent at the menses and they have been known to be the seat of vicarious menstruation; puberty sometimes arrests their growth.

On the other hand, they may undergo regressive changes; the centre becomes gradually paler and this change extending, may remove the entire disease; in other cases the tumour ulcerates and leaves a white cicatrix; this process is attended with danger from hæmorrhage. Or thrombi may form spontaneously and becoming organised, obliterate the blood-spaces in which they occur; in some instances repeated limited thrombosis occurs.

In some instances of cavernous nævus a *blood cyst* develops, owing to the shutting off of a blood-space and its subsequent distension; in other cases a pigmented warty growth has replaced a nævus. A combination of cavernous nævus with lipoma—*nævo-lipoma*—is met with as a congenital tumour.

A simple nævus cannot be mistaken for any other affection. A cavernous nævus, when covered by healthy skin or mucous membrane, is easily recognised by its spongy feel, compressibility, distension under effort, purple colour, and the absence of pulsation.

Treatment.—Nævi undergoing natural cure, and superficial nævi of wide extent, such as "port wine stains," should not be subjected to any treatment. Nævi on parts of the body which are covered by clothing, and which are stationary, may be left for a time and watched; but even if they show no tendency to grow, it is better to remove or destroy them than to leave them to be a possible source of trouble hereafter.

There are many ways of dealing with nævi: they may be excised, strangled with a ligature, burnt with caustics, obliterated by inflammation, or the blood within them coagulated by electrolysis, or the injection of coagulants. Each method has its advantages, and is to be chosen in particular circumstances, and it is a mistake to regard any one treatment as universally adapted for a condition which presents such striking variations as nævus does. The objects to be aimed at are, in the first place, *complete cure*, for if only a minute portion of the nævus is unobliterated, the disease is apt to grow again, and often with considerable vigour; secondly, the *removal or destruction of diseased parts only*, all healthy structures being preserved if possible; thirdly the *minimum of scar*, particularly on exposed parts of the body; and fourthly, *rapidity*, *painlessness*, and *absence of suppuration*. The methods commonly employed are as follows:—

(1) *Excision.*—The surgeon must be careful to cut out the nævus, and not cut into it, and each bleeding vessel must be seized in forceps, or tied at once, as hæmorrhage is very serious in infants, who are the usual subjects of this operation. Owing to the deep colour of the tumour, and the presence in cases of cavernous nævus of a thin capsule, the operation is not attended with any special difficulty. The edges of the wound are carefully approximated with sutures, and primary healing with a linear scar is obtained. This treatment is rapid, practically painless, and by it it is easy to ensure the removal of the entire growth; in a large proportion of cases it is wholly satisfactory, but it is unsuited to very superficial nævi, as it involves then a sacrifice of healthy tissue, and to deep nævi of important parts, such as the eyelid, cheek, and lip, the removal of which would cause serious deformity.

(2) *Ligature.*—Almost any nævus can be strangled by a ligature, and when the sphacelus separates, the wound quickly heals, but

leaves a circular or irregular scar. It is a painful method of treatment, much longer in effecting a cure than excision, is attended with suppuration and all its attendant evils, leaves a large scar, and by its means healthy tissues often have to be sacrificed in order to ensure the enclosure of the entire nævus in the ligature or ligatures. The advantages of the method are its extreme simplicity and the avoidance of all risk of hæmorrhage; the surgeon can do it single-handed, and in many cases without the use of an anæsthetic. Where he can secure proper assistance, ligature is undoubtedly inferior to excision, and should only be practised in unusual circumstances. Care must be taken to include the whole of the nævus in the loop or loops, which should be tied as tightly as possible; strong aseptic silk should be used, and the part be rendered aseptic and dressed with the usual antiseptic precautions, in order to prevent suppuration. In no case should the ligature be made to cut its way through skin, but the thread should be placed subcutaneously, or the skin divided with the knife. This is the method which was formerly most widely practised.

(3) *Electrolysis* is an admirable method of treating any nævi. By passing a mild continuous current of electricity through the nævus, coagulation of blood in the vessels or spaces is secured with sufficient irritant action to lead to their permanent occlusion, and without serious necrosis or ulceration. At the positive pole oxygen is set free and oxidises the electrode and the tissues, and if the current is strong, a certain amount of charring of the tissues occurs. At the negative pole nascent sodium is produced, and at once forms caustic soda and liberates hydrogen; if this action be too intense, a wide and deep slough results. The mildest action is obtained by inserting "positive" needles only, and having the negative electrode applied to the skin near by; if a more intense action is desired, the needles passed into the nævus are connected with both poles of the battery. A good plan is to have all the needles but one "positive." The best current to employ is such as is developed from a battery of 5 to 10 Leclanché cells, and to continue it for 15 to 20 minutes; or a current of 100 milliampères may be passed for 5 minutes, and then the position of the needles changed.

A slow action is to be preferred to a rapid. The operation is painful and necessitates an anæsthetic, but the pain ceases immediately the current is broken. Small nævi may be cured at one "sitting," but for nævi of any size, repeated operations are necessary. Entirely subcutaneous nævi may be cured without leaving any scar; but nævi involving the skin can only be cured by destroying the vascular tissue and replacing it by scar, this scar is, however, very little conspicuous. A certain amount of shock is produced when the current is closed; and in operating upon the head, especially in infants, great care must be taken to turn on the current gradually. Port wine stains are treated by flat metal electrodes connected with the negative pole, which are applied to the discoloured skin only, and a current of 40 to 50 milliampères is used, until a soapy *débris* collects under the plate from the caustic action of the soda set free, and the skin is destroyed for a sufficient depth. Electrolysis is an excellent method of destroying small phlebectasies as well as larger growths.

(4) *Caustics* of many kinds are employed and in certain cases are very good. Nitric acid or acid nitrate of mercury, and the fine point of a Paquelin's cautery are the best to use. They are particularly suitable for very superficial nævi, involving less than the whole thickness of the skin, and for scattered points of nævus.

(5) *Injection of coagulants* is but little practised and should be entirely superseded by electrolysis. Owing to the danger of embolism, a temporary ligature must in every case be placed around the nævus for at least fifteen minutes after the injection; perchloride of iron, a solution of tannin, or pure carbolic acid are the fluids employed, and only a very small quantity of either of them is injected.

(6) *Setons and vaccination* are employed to excite inflammation in the nævus and so to obliterate the blood-spaces, but there is great practical difficulty in so regulating the area of inflammation as to make it exactly coextensive with the nævus, and its severity, so that it may "cure" the nævus without exciting sloughing or deep suppuration. Superficial nævi may be cured by vaccination, but other methods of treatment are to be preferred; deeper nævi have been treated by passing threads soaked in vaccine lymph through them.

(7) *Scarification* has been advocated for the treatment of extensive very superficial nævi such as "port wine stains," but the results of this method have been very unsatisfactory.

XXV. ANEURYSM.

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Classification.

I. Idiopathic or spontaneous aneurysm.
  1. Fusiform.
  2. Sacculated : (*a*) Circumscribed.
    (*b*) Diffused.
      (i) Leaking aneurysm.
      (ii) Ruptured aneurysm.

II. Traumatic aneurysm.
  1. Circumscribed.
  2. Diffused; or ruptured artery.

III. Arterio-venous aneurysm.
  1. Varicose aneurysm.
  2. Aneurysmal varix.

IV. Cirsoid aneurysm. (*See* page 577.)

Definition.—*An aneurysm is a blood-tumour communicating with an artery.* If it occurs as the direct result of an injury to the vessel wall, which severs all the coats more or less completely, it is called a *traumatic aneurysm;* but when as the result of disease having impaired its resiliency and strength, the arterial wall permanently yields under the pressure of the blood, it is called an *idiopathic or spontaneous aneurysm.* These two great classes of aneurysm differ not only in their mode of origin, but to some extent in their nature and in their indications for treatment, and they must therefore be dealt with separately. An abnormal communication between an artery and a vein is known as an *arterio-venous aneurysm;* this may be either traumatic or idiopathic in origin, and its features are so peculiar that it is placed in a separate class. *Cirsoid aneurysm* is an *arterial angeioma,* and is described on page 577 under that heading; it has no pathological affinities with the other forms of aneurysm.

I. IDIOPATHIC OR SPONTANEOUS ANEURYSM.

Ætiology.—A spontaneous or pathological aneurysm is formed when an artery permanently yields under the pressure of the blood within it. Normally, such yielding is prevented partly by the strength of the arterial wall, and partly by its elasticity, which ensures its recoil immediately the force of the ventricle is expended. Healthy arteries have such a reserve of resistance, above all the demands that can be made upon them, that simple increase of blood pressure, from plethora, over-action of the heart, or increased resistance in the capillary circulation, never by itself causes aneurysm. When, however, the strength or the elasticity of an artery is lessened, it may yield under even the normal blood pressure, and still more readily does it yield when the blood pressure is increased. From this it follows that *the immediate cause of every case of spontaneous aneurysm is some condition weakening the arterial wall.* In many cases this is *supplemented by an increase in the blood pressure in the arteries.* These factors must be considered in some detail, and then we must more briefly discuss the *secondary or remote causes of aneurysm.*

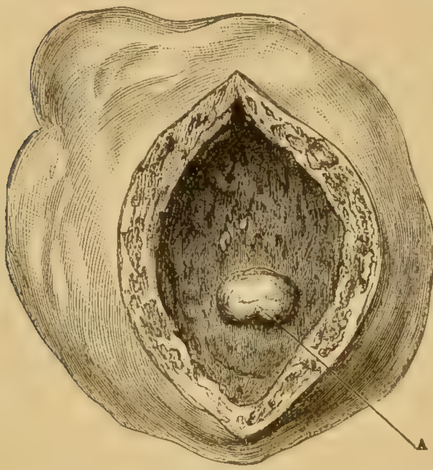

Fig. 166.—Pulmonary Cavity with a small Sacculated Aneurysm (A) projecting into it. (Drawn from a specimen in the Museum of the Hospital for Diseases of the Chest, Brompton.)

Conditions which weaken the arterial wall.—*Atheroma*, with fatty degeneration of the inflammatory products, is the most important of the conditions which weaken arterial walls; although the diseased artery is thickened, it is weaker, and, above all, less elastic than a healthy artery. As a result of the weakening of the vessel, the diseased portion may be dilated by the force of the heart, and the loss of elasticity prevents a recoil from taking place during the cardiac diastole. Calcification preserves arteries from aneurysmal dilatation, although calcareous plates may be found in the walls of aneurysms. (*See* Fig. 168.) Tubular *calcification* may be followed by aneurysm, if under some blow or strain the calcareous middle coat snaps across, and the outer coat of the vessel yields before the blood pressure.

By some authorities *syphilis* has been credited with playing a very important rôle in the production of aneurysm. A form of arteritis, which is undoubtedly due to syphilis, occurs in small arteries, especially in those at the base of the brain, and leads to their thickening and often to their obliteration; and it has been suggested that when the middle coat has been destroyed, and the new tissue in the inner coat absorbed, the artery is so weakened that it yields and forms an aneurysm. This disease is a frequent cause of aneurysm of the cerebral arteries, and it is alleged that a similar disease occurs in the larger arteries, but it is as yet unproved that syphilis leads to a special disease of large arteries, with a weakening of their coats, and therefore a predisposition to aneurysm. In many cases of aneurysm no cause for the condition has been found, except syphilitic infection.

Embolic arteritis.—The impaction of an infective embolus in an artery leads to an inflammation of the vessel, with softening; and in these circumstances an aneurysm may develop. Idiopathic aneurysm in young people is believed to be always due to this cause; the multiple aneurysms which sometimes occur in the course of acute rheumatism and ulcerative endocarditis have the same origin.

Loss of support by absorption of the surrounding tissues has been supposed to be capable of so weakening an artery that it can no longer resist the force of the blood stream. Examples of this are said to be furnished in phthisical lungs, where aneurysms not unfrequently develop on the branches of the pulmonary artery exposed in the walls of cavities (Fig. 166); their rupture is a frequent cause of fatal hæmoptysis. But it is by no means certain that these aneurysms result directly from the loss of support to the vessel. The more probable explanation is that the exposure of the artery leads to an inflammatory change in its walls which weakens the vessel and causes it to yield under the blood pressure. This yielding would be favoured by negative pressure in the lung cavity. A microscopical section of a branch of the pulmonary artery with a minute commencing aneurysm, in the possession of Dr. Percy Kidd, shows distinct thickening of the intima of the vessel, and supports the view that in this, as in every other form of spontaneous aneurysm, disease of the arterial wall precedes the development of the aneurysm.

Under this head we must also mention *injury causing a partial laceration* of an artery. A contusion of an artery, or over-stretching it, as in violent extension of the knee or elbow, may not only snap a calcified middle coat, as mentioned in the previous chapter, but may burst a softened patch of atheroma, or displace a calcareous plate and allow the blood to force its way beneath its edge, or even tear some healthy part of a diseased artery. In either of these ways injury may so weaken an artery as to lead indirectly to the development of an aneurysm.

Conditions increasing the blood-pressure.—The blood-pressure in the arteries may be raised by increased cardiac action or by greater resistance in the capillaries and arterioles, and by plethora; but *the over-action of the heart* is the most important in the ætiology of aneurysm. This over-action is especially excited by sudden, intermittent, and unwonted effort or strain. The heart and blood-vessels accommodate themselves to continuous hard work; but when an unusual effort is made, especially if of the nature of a sudden strain, the blood pressure is materially raised. A like effect is produced by great excitement or violent passion, by alcohol, and by cardiac hypertrophy. *Plethora* is a cause of increased blood pressure; it is one that acts continuously, and it is usually associated with other conditions producing the same result. Its influence is best appreciated by noticing that the opposite condition—anæmia and cachexia—is a great protection against aneurysm, which only occurs under those conditions when an artery has been greatly weakened, as by an infective embolus. The influence of bleeding in arresting the rapid growth of an aneurysm is another proof of the important part plethora may play. The *increased resistance in the arterioles and capillaries*, met with in gout and chronic renal disease, is an important cause of atheroma, by raising the blood-tension in the larger arteries. Like plethora it acts continuously; it also acts intermittently when due to muscular effort, and is one of the factors by which strain leads to aneurysm. Associated with marked weakening of the arteries from disease, the normal blood-pressure is quite sufficient to produce aneurysm, as we see in aneurysm from embolism; but as a great cause of atheroma is increased blood-pressure, this condition, both directly and indirectly, plays a great part in inducing aneurysm. The greater frequency of aneurysm in the aorta than in any other artery is due to the high blood-pressure in, and the great strain thrown upon, this artery.

The secondary causes of aneurysm must be specially noticed.

(1) *Age.*—Aneurysm is most common between the ages of thirty and fifty, at the period when degenerative changes occur in the arteries, when the force of the heart is unimpaired, and when persons are still exposed to strains and injuries. It is very rare in children and adolescents, and in them is probably always due to embolic arteritis. Old age, although associated with advanced arterial disease, is protected from aneurysm by the low blood-pressure due to the feeble action of the heart and the absence of special strain and effort.

(2) *Sex.*—Aneurysm is much more frequent in men than women, the occupations and mode of living of the latter protecting them from the chief causes of the disease. Statistics show that aneurysms generally are thirteen times as common in men as in women; but there are two remarkable exceptions to this rule, for carotid aneurysm is as frequent in women as in men, and dissecting aneurysm is more common in the female sex. No satisfactory explanation of these exceptions has been given. It is worthy of note that the strain of parturition—often very severe and prolonged—does not appear to lead to aneurysm.

(3) *Occupation.*—All occupations which expose to sudden and severe effort, such as those of soldiers and sailors, dispose to aneurysm by the sudden rise of blood-pressure. Laborious occupations as of miners, navvies, and engineers, which throw a constant strain upon the circulation, lead to atheroma and to aneurysm. Those who habitually lead sedentary lives, but occasionally indulge in violent effort such as mountaineering and other forms of active exercise, are prone to aneurysm by the unwonted rise in the blood-pressure such an effort causes. The influence of occupation is well shown by the special frequency of aneurysm in cold and temperate climates,

and particularly in Great Britain and America, and by its occurring more often in the right than the left arm.

(4) *Injury.*—A local injury often precedes the development of an aneurysm; it may act by exciting atheromatous inflammation, by bursting an atheromatous abscess, or by partially rupturing a diseased artery. This sequence is as frequent in internal as in external aneurysm; the concussion of the mediastinum from a blow on the sternum appears to be often the starting-point of an aortic aneurysm.

(5) *Strain* is a very important cause of aneurysm, for it has several injurious effects. It increases the heart's action; it increases the capillary resistance in the muscles; and by both of these effects raises the arterial blood-pressure. It also, in some cases, stretches or compresses an artery, and in this way excites atheroma in it, and it may partially lacerate an already-diseased artery. Strains at the knee are an important cause of popliteal aneurysm.

(6) *Alcoholic indulgence*, like strain, may cause aneurysm in several ways: (*a*) By inducing frequently-repeated cardiac excitement; (*b*) in this way, and also by its direct action, exciting atheroma; (*c*) by setting up chronic renal disease leading to arterio-capillary obstruction; (*d*) by exposing its subjects to injuries and strains.

(7) *Diathesis.*—An *aneurysmal diathesis* has been spoken of as existing in cases of multiple aneurysm. These cases are, however, more easily explained by the consideration that the causes of aneurysm are, to a large extent, general rather than local; and, in view of this fact, it is remarkable how infrequently more than a single aneurysm is met with.

The influence of *syphilis* has already been referred to: some authorities regard it as a very important one; but it has not been proved to play any part in causing external aneurysm. *Gout*, by leading to atheroma, to chronic renal disease, and to spasm of the arterioles, and by being often associated with plethora, certainly predisposes to aneurysm. *Rheumatism* occasionally leads to aneurysm, by causing embolic arteritis; it may also cause acute endarteritis, followed by aneurysm.

Symmetry.—It is interesting to notice that aneurysms are not infrequently symmetrical. Aneurysm of each popliteal artery has often been met with, but such cases as aneurysm on each vertebral artery within the skull—and on no other artery of the body—or of each division of the hepatic artery, occur. This symmetry shows how large a part "constitutional" causes play in the production of aneurysm. The explanation of this symmetry is found—in part, at least—in the following considerations. The arteries on the two sides of the body are in precisely identical anatomical conditions. In many cases the functional activity of the two sides is practically the same—this certainly applies in the case of the vertebral artery, and, in most occupations, to the popliteal artery; the distending force is one and the same in all cases—the force of the heart. These three factors underlie the production of the initial disease of the artery, and its culmination in an aneurysm, in the great majority of cases. Indeed, a purely idiopathic or spontaneous aneurysm must always be symmetrical.

The influence of unequal function is well shown in the much greater frequency of spontaneous aneurysm in the arteries of the right than the left arm. The occurrence of aneurysm in each division of the hepatic artery is probably explained by the occurrence of embolism, a single embolus having been broken into two by impinging upon the septum between the two hepatic arteries.

Development.—When an artery yields to the pressure of the blood within it, the whole circumference of the vessel may be stretched, or a limited patch of the wall of the artery may bulge out. This will chiefly depend upon the extent of artery diseased. In the large arteries atheroma often affects almost uniformly a considerable extent of the vessel; and in them, therefore, a yielding of the entire vessel is most commonly met with. In these cases the artery is dilated transversely and elongated, and a tubular or *fusiform aneurysm* is formed. Uneven distribution of the disease of the artery may cause irregularity in the outline of the aneurysm, and in some cases marked local bulgings occur. When the whole circumference of the artery yields, the three coats of the vessel are found in the sac of the aneurysm. The inner coat is thickened from atheroma, and sometimes contains calcareous plates. The middle coat is spread out, the muscular fibres being separated, and ultimately undergoing fatty degeneration. The outer coat is thickened by the addition of new fibrous tissue. Localised yielding of an artery is due to the weakening of the artery advancing to a much greater degree in one particular spot in the wall. This may be a patch of atheroma that has undergone fatty degeneration; and at first the whole thickness of the artery bulges out, as in fusiform aneurysm. But as it grows the middle coat becomes more and more scanty in its walls, until it disappears, and the internal coat having lost its elasticity, ceases to expand, and the sac comes to be formed by the thickened tunica adventitia only.

In other cases the aneurysm commences in an "atheromatous abscess," which bursts, or an "atheromatous ulcer." The part of the artery thus weakened yields and expands, but in this instance the intima and most of the media are absent from the sac from the beginning, which resembles from the first the later stage of the previously described variety.

As aneurysms grow the original vessel wall is so stretched and thinned that it may ultimately disappear over a large part of the sac; but its place is taken by new fibrous tissue formed by plastic inflammation of the adventitia, and of the sheath of the artery, and so the continuity of the sac is preserved. With yet further enlargement of the aneurysm, the tissues and organs surrounding it, which are at first displaced by it, become incorporated in its wall, and so it comes about that muscles, nerves, other blood-vessels bone, cartilage, fasciæ, skin, and mucous canals may be found matted in the sac of an aneurysm. Such aneurysms have generally a more or less globular outline, and their sac or wall is more or less distinct from the artery from which they spring. They are known as globular or *sacculated aneurysms.*

In some rare cases it happens that the tissues forming the floor and edge of an "atheromatous ulcer" are not so matted together as to resist the force of the blood. When this is the case, the blood, instead of bulging out the floor of the ulcer, finds its way between the coats of the vessel, and separates the intima from the media and adventitia. In this way a diverticulum in the wall of the artery is formed which is called a *dissecting aneurysm.*

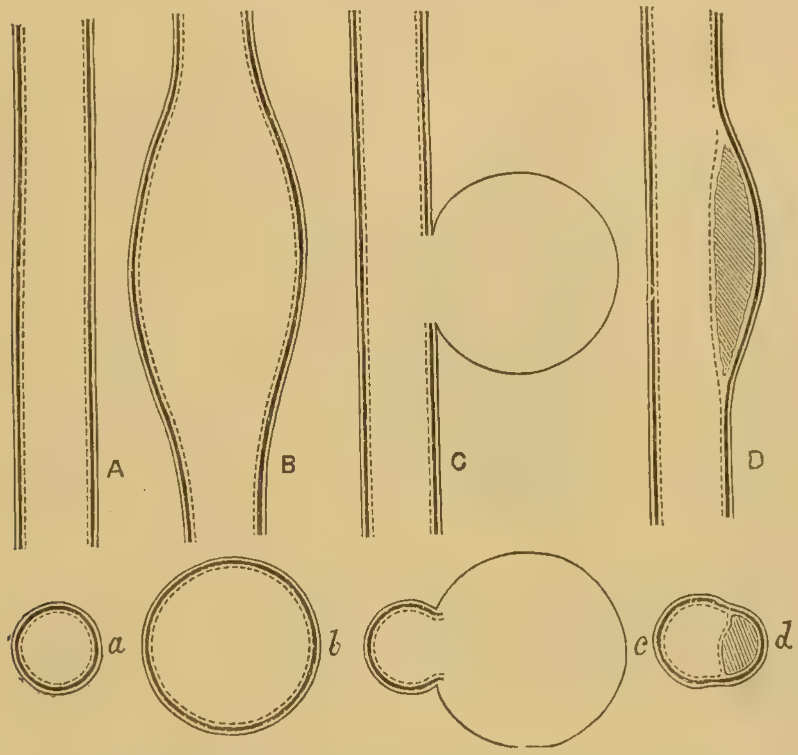

Fig. 167.—Diagrammatic Representation of the Varieties of Aneurysm.

A, A healthy artery; B, a fusiform aneurysm; C, a sacculated aneurysm; D, a dissecting aneurysm; *a, b, c, d,* the same in transverse section.

Varieties.—The difference in the mode of development just described forms the basis of the primary classification of idiopathic aneurysms, and it is important to bear in mind that the distinction between a "fusiform" and a "sacculated" aneurysm in not one of shape merely, but of nature; it is unfortunate that the terms employed do not better express the fundamental difference between them. Sacculated aneurysms are subdivided into two main species—"circumscribed" and "diffused"—according to the continuity or the reverse of the sac of the aneurysm. We therefore arrive at the following classification of *spontaneous aneurysms* (Fig. 167):—

1. Fusiform aneurysm.
2. Sacculated aneurysm
    - (*a*) circumscribed.
    - (*b*) diffused; leaking; ruptured.
3. Dissecting aneurysm.
4. Mixed aneurysm.

A fusiform aneurysm is one formed by the dilatation of the entire circumference of an artery, and it is a dilatation in length as well as breadth; but, above all, its walls (or sac) are formed of all the coats of the artery.

A sacculated aneurysm is formed by the yielding of a part only of the circumference of a vessel; and its sac, except in the earliest stage of a few cases, consists at most of part of the wall of the artery only. So long as the tumour is closed in by a continuous sac, it is called a *circumscribed aneurysm.* When the sac is incomplete, it is called a *diffused aneurysm* or a *ruptured aneurysm,* and this distinction is one of vital importance. Other terms have been used in the classification of aneurysms, especially "true" and "false." A "true" aneurysm is one in which the sac is formed by all the coats of an artery. This is always the case in fusiform aneurysm, but in sacculated aneurysm—to which the term has been generally applied—such a condition is only met with in the early stages of the development of some aneurysms, and when a comparatively large area of the arterial wall has yielded to the pressure of the blood. This condition is incapable of recognition by any clinical features of the aneurysm. Any aneurysm in the sac of which all three coats of the artery are not found has been called "false." These terms have, however, been also used in another sense, for the term "true" has been used to denote an aneurysm in which any part of the coats of the artery can be demonstrated in the whole sac of the aneurysm; and "false" has been used to denote that condition in which a part, at least, of the sac consists wholly of tissues other than the arterial wall. These terms should be given up, for not only have they been employed in different senses, but they connote a difference that cannot be appreciated clinically and that is of no therapeutic importance. Either form of aneurysm, whatever the nature of its sac, is a veritable aneurysm, and to separate off any group and call it "false" is an incorrect and unfortunate use of language.

A dissecting aneurysm is one in which the blood is contained in a space between the coats of the artery. Such a diverticulum may remain as a blind pouch, or it may open into the vessel lower down, or burst through its outer coats, and become a "ruptured or diffused aneurysm."

A mixed aneurysm.—It sometimes happens that the entire circumference of an artery dilates into a fusiform aneurysm, and that at one part of the circumference a sacculated aneurysm is also formed by a localised yielding of weakened tissues. In such cases the disease must be called a *mixed aneurysm.* It is important to denote such cases by a special name, for they are not uncommon, and the addition of a sacculated to a fusiform aneurysm involves all the dangers of the sacculated form of the disease, although it may happen that the less serious fusiform aneurysm forms by far the larger part of the tumour. The special features of each of these forms of the disease must now be separately considered.

1. FUSIFORM ANEURYSM.

Fusiform aneurysm is a dilatation in all directions of the entire circumference of an artery; the vessel is elongated as well as enlarged laterally; the enlargement may be uniform or irregular. This form of the disease is especially met with in the arch of the aorta, but it also occurs in the descending aorta, the iliac, femoral, and, rarely, in the popliteal arteries, and in the innominate, common carotid, subclavian, and axillary arteries—in the largest arteries in fact. Two or more such dilatations may be met with on the same trunk.

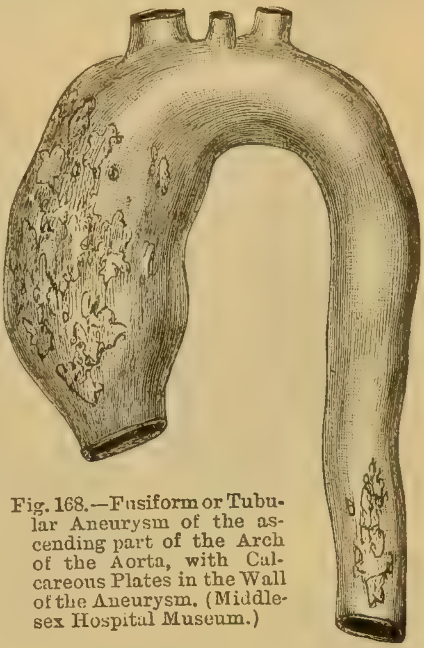

Fig. 168.—Fusiform or Tubular Aneurysm of the ascending part of the Arch of the Aorta, with Calcareous Plates in the Wall of the Aneurysm. (Middlesex Hospital Museum.)

The sac of such an aneurysm is formed of all three coats of the artery, and is, as a rule, thicker than the arterial wall; this fact constitutes the important pathological distinction of fusiform aneurysm, and explains its slow course and its general benign character as compared with sacculated aneurysm. The outer coat of the vessel is thickened, often considerably, with new fibrous tissue; the middle coat is stretched, and the bundles of muscular fibres are thinned and separated, and ultimately undergo fatty degeneration and disappear. The inner coat is always thickened, uneven, and shows well-marked signs of atheroma, calcareous plates are often found in the wall of the aneurysm (Fig. 168). As a rule, the contents of a fusiform aneurysm are solely fluid blood, with, at most, shreds of clot adhering to rough portions of the interior; but extensive clots may be found, and spontaneous cure has been brought about by the total occlusion of the vessel (Fig. 169). The most important change that takes place in a fusiform aneurysm is when some especially diseased part of its wall yields and forms a sacculated aneurysm, for such a sac has a tendency to rapid growth, to exert serious pressure on surrounding parts, and to rupture (Fig. 172). If the disease involves the part of the aorta nearest to the heart, there is a special danger of rupture from failure of the outer coat to undergo thickening and strengthening; this is said to be due to the absence of any fibrous sheath around this part of the artery.

The course of uncomplicated fusiform aneurysm is slow; the disease develops slowly and may long remain stationary and give rise to no serious symptoms. It is often the seat of a very loud rough bruit. When the disease occurs in the arch of the aorta—its most frequent situation—it is often fatal. In this situation the tumour may cause serious pressure upon the very important structures adjacent to it; and from the great pressure of the blood, some weakened part of the sac is very liable to yield and form a sacculated aneurysm, or the first part of the aorta may burst into the pericardium or the auricle, and cause instant death; or, again, the great dilatation of the aorta may affect the orifice and lead to incompetence of its valve, or the great volume of blood in the distended vessel may offer such obstruction to the action of the left ventricle of the heart that sudden syncope occurs. It has also been suggested that in fusiform aneurysm of the first portion of the aorta, the coronary arteries may not be duly filled, if the inelastic aorta does not contract after the action of the left ventricle. It is clear, however, that if the resiliency of the aorta is so entirely lost that blood is not forced into the coronary vessels, the cusps of the aortic valve would not be opened out, and fatal regurgitation would take place. Great dilatation of the root of the aorta may displace the orifices of the coronary arteries to such an extent as to impede the cardiac circulation.

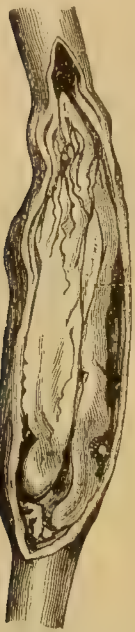
Fig. 169.—Fusiform Aneurysm of Popliteal Artery, entirely filled with Laminated Clot. Spontaneous cure. (Royal College of Surgeons Museum, No. 3246.)

The diagnosis of fusiform aneurysm of external arteries is made by noticing the form and position of the pulsating swelling, and its slow and usually symptomless course. The diagnosis of internal fusiform aneurysm can rarely be made with certainty; the slow course of such an aneurysm, and the absence of grave pressure signs, would be strong evidence in favour of such a diagnosis, but would not be conclusive evidence against a "mixed" aneurysm.

Treatment.—No curative treatment is employed for fusiform aneurysm, for in external arteries it does not cause serious consequences, and in the aorta cure can only take place by total obliteration of the vessel. But palliative treatment should not be neglected, and is of greater value the nearer the aneurysm approaches the heart. It consists of all measures tending to lessen the blood pressure in the aorta, and especially of the avoidance of effort and strain, of avoidance of stimulants of all kinds, including over-eating, and in careful regulation of the secretions.

2. SACCULATED ANEURYSM.

The sac.—A sacculated aneurysm is formed by a dilatation of a part of the circumference of an artery. Where a relatively large portion of the wall of an artery is weakened by disease, and bulges under the pressure of the blood, the sac may at first consist of all three coats of the vessel. In this case, the mouth of the aneurysm will be large, and the sac of small size; in the sac patches of atheroma and calcareous plates may be found, proving without doubt the presence of the inner coat of the artery. In its essential features such an aneurysm resembles a fusiform aneurysm; it is rare, and found only on the larger arteries. If the aneurysm enlarges, the inner coat does not keep pace with it, the middle coat is stretched and atrophied, and the sac then consists of the outer coat of the artery, strengthened on its outside by new fibrous tissue. It would be more correct, however, to say that when in such an aneurysm the inner coats are so diseased that they give way, the aneurysm at once enlarges. The importance of the inner coats of an artery in strengthening the vessel and enabling it to resist the pressure of the blood has been too little recognised. Figs. 154 and 155 show how large a share they form of the wall of even the largest arteries.

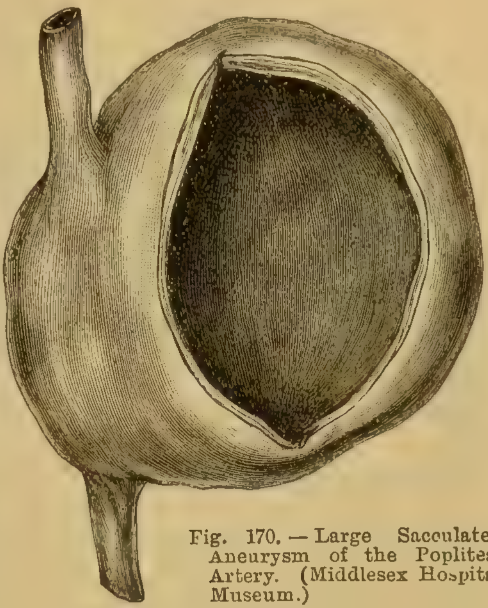

Fig. 170. — Large Sacculated Aneurysm of the Popliteal Artery. (Middlesex Hospital Museum.)

In the great majority of cases of sacculated aneurysm, the sac from the first consists of a protrusion of the outer and part of the middle coat only, the inner coat and the inner part of the middle coat having been destroyed by atheroma. The muscular tissue is thinned out, undergoes fatty degeneration, and quickly disappears. The outer coat grows and is thickened by a new formation of fibrous tissue, produced by chronic inflammation of the sheath of the artery excited by the pressure of the aneurysm. The simplest proof of this growth of the sac of an aneurysm is afforded by the facts that it is usually thicker than the healthy arterial wall, and that it is often so very much larger than the mouth of the sac which corresponds to the portion of the artery from which it took origin (Fig. 170). When blood-clot adheres to the interior of the sac, it quickly becomes organised into fibrous tissue, and strengthens the sac on its inner surface. The sac of such an aneurysm can be distinguished from the sac or wall of any other tumour by the existence of many spaces between the layers of fibres composing it; these spaces are filled with loose connective tissue, and their occurrence is said to be due to the pulsation in the aneurysm.

In a small aneurysm, therefore, the sac consists in part or in whole of the coats of the artery. In a medium-sized aneurysm the sac is formed practically entirely by a new growth of fibrous tissue firmly incorporated with the remains of the outer arterial coat; the new tissue is the product of chronic peri-arteritis, and is sometimes supplemented by the organisation of adherent clot. In very large aneurysms another condition is introduced. As the tumour grows, it presses upon and displaces the surrounding parts; some of these are atrophied, some are flattened out, but it often happens that some of them become embedded in the constantly-forming new fibrous tissue, so that in the sac of a large aneurysm, tendons, fasciæ, ligaments, nerves, vessels, cartilages, and bones may be found matted together by fibrous tissue. *The typical sac, therefore, is a new formation of fibrous tissue*, and it is this fact which explains the special features of the life-history of sacculated aneurysms.

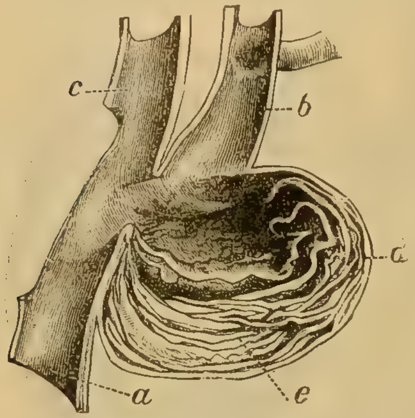

Fig. 171.—Sacculated Aneurysm of Common Carotid Artery.

a, Common carotid artery; *b*, ext. carotid artery; *c*. int. carotid artery; *d*. sac of aneurysm; *e*, laminated clot partly filling sac.

The **outline** of the sac depends chiefly upon three circumstances. The primary tendency is for the aneurysm to assume a globular form, owing to the equality of fluid pressure in all directions. This is modified by the unequal resistance offered by the surrounding structures, as well as by the effect of the direct impact of the blood-stream upon that part of the sac opposite its mouth. But if a clot adheres to part of the sac and becomes organised, it strengthens the sac at this spot, and the chief growth then takes place where the sac is thinner and weaker.

The **contents** of every uncured sacculated aneurysm consist in part at least of ordinary arterial blood, which after death appears as a soft black blood-clot. In nearly all cases, more or less clotting of the blood occurs during life, and these clots play a most important part in the history of an aneurysm. The clots vary greatly in amount, in arrangement and in appearance. They are usually found in concentric layers pressed firmly against the sac, many of the layers being firm and almost colourless, others being softer and more granular, and coloured with varying shades of red and brown. The coagulation results from the adhesion of the ferment-containing corpuscles to the interior of the sac, and their subsequent disintegration, which sets free the ferment and leads to the formation of fibrin. The differences in the clots is explained in two ways: one view is that if this fibrin forms in a rapid blood-stream, the corpuscles are, as it were, whipped out of it, and a clot of nearly pure fibrin is the result; such a clot is called a *white clot*, from its colour, and an *active clot*, from its supposed special active share in the cure of aneurysm, and a *laminated clot* from its occurrence in several imbricated layers (Fig. 171). If, however, the fibrin separates in blood that is nearly or quite stagnant the corpuscles are caught in its meshes, and a *red, passive, or blood* clot results. Between these two extremes, of a rapid blood-stream and stasis, all gradations occur, and therefore clots of various degrees of corpuscular richness and colour result. This has been the usually accepted view. Evidently it rests solely upon surmise, as the process has never been seen, and it is exceedingly improbable that the blood platelets would adhere to the sac at all if they were exposed to the rush of a stream of blood, rapid enough to "whip" the fibrin free from corpuscles. Corroboration of this is found in the absence of such clots in fusiform aneurysms. Another more probable view is that coagulation only occurs in quiet "eddies" and "backwaters" of an aneurysm, and that in all cases the clot when formed is true blood-clot—fibrin and corpuscles—and that it becomes greatly altered in nature and appearance by the pressure to which it is exposed. The pressure flattens out the clot against the sac, and squeezes out the serum, the red corpuscles then disintegrate, and so a "white" fibrinous clot is obtained, and when several clots have been formed and thus pressed together, the "laminated" appearance is produced. A clot not exposed to this pressure and formed shortly before death, presents the usual features of blood-clot.

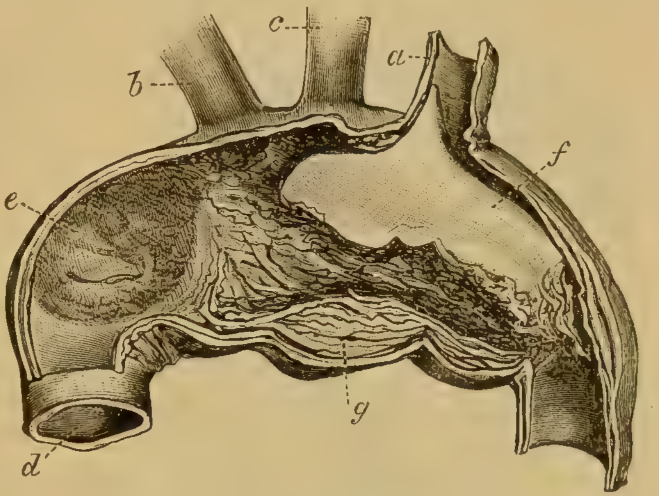

Fig. 172.—Fusiform Aneurysm of Arch of Aorta. (Museum of the Royal College of Surgeons.)

Showing at *e*, a sacculated pouch; *g*, laminated clot: *f*, recent clot extending into *a*, left subclavian artery; *c*, left carotid; *b*, innominate artery.

The layers of clot in an aneurysm are variously arranged: the most typical condition is that in which the oldest clot is next the sac, and those formed later are arranged concentrically within it; this produces a very characteristic laminated appearance. and as each layer is smaller than the layer next outside it, an imbricated appearance is produced. Very often, however, the clots are pressed to one side of the sac, or the blood finds a path between the clots and the sac, and new coagula are deposited there, and then the oldest layers of a laminated clot may be in the centre; the growth of the aneurysm in a new direction is another way in which the order of deposit of clots may be varied. There is one sign by which the relative age of the layers of clot may be determined, and that is their colour; the older a clot the more completely have the corpuscles within it disintegrated, and therefore the whiter it is. The colour of a clot, indeed, is an index of age rather than of the mode of its formation. Old fibrinous clots are very resistant and may continue unaltered possibly for years.

The coagula within an aneurysm are extremely important. In the first place they protect and strengthen the sac; next, as we shall see later, they are the natural means of cure of the aneurysm; and, lastly, by partly filling up the sac they greatly reduce the pressure of the blood on the sac of the aneurysm and on the parts around it. This last is a very valuable function. The pressure on the wall of an aneurysm is in direct proportion to the ratio of its area to the transverse area of the artery from which it springs, and an aneurysm, from this point of view, may be regarded as a living Bramah press, and therefore the mere partial filling of the sac by a solid material—clot—by so much reduces the pressure of the aneurysm. As a result of this, aneurysms containing much clot increase slowly, if at all; and aneurysms without coagula grow very rapidly, and exert enormous pressure upon the sac and the surrounding parts.

The organisation of the clots can only occur when "lymph"—plasma-cells—can pass into them from the living tissue of the sac, and this is prevented by the movement of the clot on the sac with each ventricular systole; if any change takes place by which the clot is protected from this disturbance, then the layer next the sac quickly becomes adherent to it and converted into fibro-cellular tissue. One reason why this does not take place more readily is that the sac of an aneurysm is not, as a rule, lined by the tunica intima, the endothelium of which plays such an active part in the organisation of a thrombus in a vessel. The way in which coagula lead to the cure of the aneurysm will be dealt with farther on.

The effects of aneurysm.—The effects of an aneurysm are partly those caused by the development and growth of a tumour—pressure effects—and partly those due to interference with the arterial circulation.

Pressure effects.—The pressure of an aneurysm upon the parts around it, for the reason already given—that it is a "living Bramah press"—is much greater than that of any other kind of tumour, therefore the pressure effects are often of the highest importance in the course of a case. The first and most constant effect of the pressure is to excite chronic inflammation in the immediately adjacent tissues; this thickens and strengthens the sac on its outer surface, and mats together the tissues. In certain circumstances acute inflammation, with suppuration, may be produced. From their proximity to arteries veins are very often compressed, and more or less obstruction is offered to the venous circulation, leading to cyanosis and œdema of the parts beyond, with dilatation of the smaller veins. A vein may be ultimately obliterated by the pressure or its wall "ulcerated" and a communication opened between it and the aneurysm. (*See* Varicose aneurysm, page 636.) By a similar process an aortic aneurysm may form a communication with the pulmonary artery. Lymphatics are compressed, and pressure upon the thoracic duct leads to wasting and to chylous ascites.

The pressure of an aneurysm often produces great changes in skeletal structures. Nerves are stretched, flattened out, and undergo degeneration; this leads to neuralgic pain, numbness, anæsthesia, and paralysis of muscles. Pressure on a motor nerve rarely, if ever, causes spasm of the muscles supplied by it: pressure on the phrenic is said to cause spasm of the diaphragm: and pressure on the inferior laryngeal nerve is said to cause laryngeal spasm; but it is doubtful how far this occurs. Pressure on a mixed nerve may cause spasm by reflex irritation: in this way pressure on the vagus may excite spasm of the larynx on the opposite side.

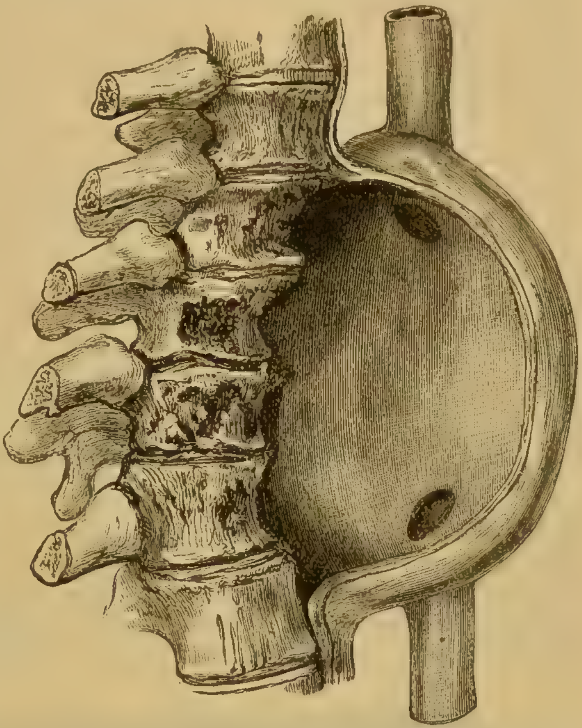

Fig. 173.—Large Sacculated Aneurysm of the Descending Aorta with Absorption of the Bodies of several Vertebræ. The intervertebral discs show no change. (Middlesex Hospital Museum.)

Bones are absorbed, being hollowed out, perforated, and removed without signs of inflammation: this is most often seen in the spine and sternum (Fig. 173). Cartilages, whether costal, intervertebral, or articular, have much greater power of resisting absorption, and may be unaltered when bones and fasciæ have disappeared, but they are flattened out or displaced by large aneurysms. Fasciæ are stretched, thinned, and then blended with the sac. Muscles are displaced, flattened out, and then absorbed; tendons are very resistant, and are displaced, flattened out, and then incorporated in the sac. In certain situations mucous canals, such as the œsophagus, the trachea, or one of the bronchi, are displaced and compressed.

Effects on the circulation. (*a*) *Loss of force.*—The force with which blood is propelled into an aneurysmal sac is so much loss to the pressure with which the blood is propelled onwards, for only a trifling amount is returned in the form of elastic recoil. This loss of force in the circulation beyond it is the most constant and characteristic effect of an aneurysm. As its direct result, the tension in the arteries beyond is reduced, and the nutrition of the tissues supplied by them *pro tanto* affected. But Nature, as it were, responds to the call of the impoverished tissues and imperfectly filled arteries in two ways: (1) by hypertrophy of the left ventricle, and (2) by an enlargement of the collateral arteries above the aneurysm; so that blood is poured into the vessels below, partly through the main trunk and partly through the anastomosing channels. When the condition of the patient prevents compensatory cardiac hypertrophy, and the anastomosing vessels are so diseased that this enlargement cannot occur, the coldness and wasting of the parts beyond an aneurysm may be marked. In many cases of aneurysm the associated cardiac hypertrophy precedes the development of the aneurysm, being chiefly due to widely distributed atheroma and the loss of elasticity in the diseased arteries generally.

(*b*) *Effect on the pulse.*—The pulse-wave travelling along an artery is so interrupted by the presence of an aneurysm that in the artery beyond it the wave is delayed, and smaller and feebler than it otherwise would be. Much attention has been paid to sphygmographic tracing of the pulse in arteries beyond an aneurysm, and the late Dr. Mahomed attributed great diagnostic value to these sphygmograms. In a typical case the tracings show a loss of the impulse and dicrotic waves, and a diminution of the force and rapidity of the tidal wave; this is well seen in the accompanying figure copied from Dr. Mahomed (Fig. 174). The influence of an aneurysm upon the pulse-wave no doubt depends upon the size of the aneurysm and of its mouth, and this explains the variations that are observed in different cases.

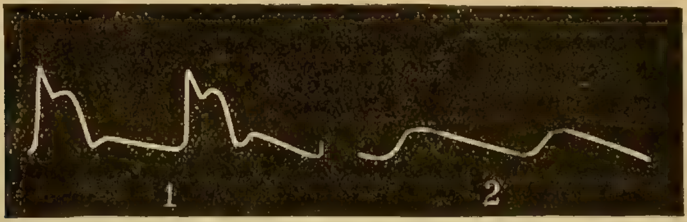

Fig. 174.—Sphygmographic Tracings of the Radial Pulse of a Patient with Aneurysm of the Right Brachial Artery. (Mahomed.)

1, Left radial pulse; 2, right radial pulse.

(c) *Obstruction of an artery* may be caused by an aneurysm in two or three ways: (1) The clot in the aneurysm may extend into and block up the artery, or a portion of it may be detached and carried into the artery as an embolus; (2) the mouth of a branch may be involved in the sac, and first stretched and displaced, and then obliterated; (3) it is stated that the sac of an aneurysm may enlarge in such a way as to compress the vessel from which it springs; but of this there is considerable doubt. The obstruction of the artery will have its usual signs and consequences.

(d) *Syncope* is a not infrequent effect of large aortic aneurysms, as has been already stated (page 594).

Course and terminations.—In every sacculated aneurysm there are two opposing forces and tendencies. The pressure of the blood tends to its progressive enlargement, and this is opposed by the thickening and strengthening of the sac and the coagulation of the blood within it. In some cases these forces are so evenly balanced that an aneurysm remains stationary even for years. As a rule, this balance is not maintained, and the aneurysm either enlarges under the influence of the blood-pressure, or advances towards spontaneous cure; sometimes alternations between one and the other are observed, as the conditions in the aneurysm vary. The main conditions influencing the course of an aneurysm are the strength of the cardiac contractions—habitual and occasional,—plethora, and the obstruction offered by the arterioles, the resistance offered to its growth by the parts around it, the presence or absence of clot within it, and the intensity of the chronic inflammation set up around the sac, on which the continual growth of the sac largely depends. Many other circumstances, such as the nature and importance of the parts pressed upon, affect the clinical course of a case of aneurysm; but we are just now only concerned with the pathological progress of the aneurysm itself.

The cure of an aneurysm is in every case only obtained by obliteration of the artery from which it springs. This statement rests not only upon the authority of Scarpa, who first propounded it, but is borne out by all clinical and pathological experience. So far as the aneurysm itself is concerned, the process of cure consists first in the filling of the sac with coagulum, and secondly in the organisation of this coagulum. We have already studied the mode of formation of clot in an aneurysm. The organisation of the clot is effected in precisely the same way as that of a thrombus in an artery, but the process is less rapid owing to the absence of the endothelium—that is to say, plasma cells pass from the sac into the clot, disintegrate and absorb it, and replace the clot by a vascular fibro-cellular tissue. This process, taking place from the periphery towards the centre, finally reduces the aneurysm to a mass of fibrous tissue, considerably smaller than the original tumour. The first step—the filling of the sac with clot—may and often does occur without the second step—organisation of the clot—and it may be mistaken for "cure," and this mistake explains many disappointments met with in the treatment of aneurysm, and the cases of so-called recurrence of an aneurysm. It is the second part of the process—the organisation of the clot—which is alone really curative, and in the cases just referred to, this essential step fails owing to the forcible stream of blood flowing through the artery. We have seen, in studying the healing of wounded arteries, that in cases of punctured and lateral wounds healing never occurs, unless the wounded artery is occluded, for the stream of blood passing through an artery effectually prevents the organisation of the clot sealing over the wound in its wall. The same holds true of the thrombus in an aneurysm. So long as that is subjected to the impact of the blood propelled through the artery, organisation cannot occur in it. In outlying parts, bays, and recesses of an aneurysm the clot may be so freed from disturbance that organisation can occur in it, and we have seen that in this way the sac may be strengthened from within, and partial cure may even result—*i.e.* certain parts of the aneurysm may be permanently cured. But the entire clot will not be organised, and the local disease cured, unless and until the artery from which it springs is occluded. There is no more striking case of the importance of "rest" in securing "cure" than in the organisation of the clot in an aneurysm, and it is just because this perfect rest can only be secured by the obliteration of the artery that it is essential to the cure of every aneurysm. The obliterated portion of the artery—usually the part between the adjacent branches—is converted into a solid fibrous cord, to which is fixed the button of similar tissue into which the aneurysm is converted.

This fact is of the highest importance. It affords the key to all successful treatment of aneurysm. It should also guard us against mistaking improvement for cure, and it teaches us that the cure of some aneurysms—as of the ascending part of the arch of aorta—is incompatible with life, while cure of some others can only occur at the cost of the gravest disturbance of the circulation and danger of gangrene.

Spontaneous cure of an aneurysm sometimes occurs in circumstances difficult to explain. It is known to be brought about in at least three ways.

(1) *Gradually, by extension of the clot from the aneurysm into the artery.*—The conditions favouring this are: (*a*) a uniform and not too forcible action of the heart; (*b*) the development of collateral vessels, lessening the tension in the artery; (*c*) the development of a second aneurysm higher up in the same arterial trunk, lessening the force of the arterial circulation below it; (*d*) such a disposition of the aneurysm that its sac compresses the artery from which it arises. It is possible, too, that the outline of the sac, the form and size of its mouth, and the existence of disease of the inner coat of the artery may affect this result.

(2) *Suddenly, by embolism.*—A portion of clot may be displaced from the aneurysm, or from one higher up, and washed into the artery, may plug it, and a thrombus then form upon it and extend above the mouth of the sac.

(3) *Plastic arteritis* may be excited by inflammation around the sac of an aneurysm, and seal the artery above and below the tumour. This is a rare event.

Cases of sloughing of an aneurysm without hæmorrhage have occurred. This is, of course, to be carefully distinguished from a "cure" of the aneurysm. The patient recovers, but by a process analogous to amputation. A case of suppuration, with recovery of the patient, has recently been recorded by Oliver, in which the sac of a large subclavian aneurysm, together with a part of the clavicle and part of the brachial plexus, was thrown off in one huge slough, which weighed over a pound; the ends of the subclavian artery securely sealed were visible in the deep wound left.

Growth of the aneurysm.—The continuous growth of the aneurysm may be fatal from its pressure-effects or from syncope, as is often seen in thoracic aneurysm. If not, it ends usually in *rupture*, but occasionally in *suppuration.*

(1) *Rupture.*—An aneurysm may rupture externally through the skin, or internally into a mucous, serous, or synovial cavity, or into the cellular tissue around the sac. Rupture through the skin is finally effected by the separation of a slough of corium, and the hæmorrhage is at once fatal, unless controlled by surgical means, which in these cases are rarely applicable. Rupture into a mucous canal occurs in the form of a small ulcer, which often becomes temporarily plugged with a clot; but as the ulcer enlarges this clot is displaced, and further escape of blood occurs. In such cases, therefore, the hæmorrhage is generally repeated—slight at first, each time more profuse, until a fatal loss occurs. The blood may be poured into the trachea or a bronchus, causing hæmoptysis; or into the gullet or stomach, causing hæmatemesis; or into the bowel, causing melæna.

The rupture into a serous cavity is by a slit or stellate opening, permitting a profuse, and generally at once fatal, hæmorrhage—hæmopericardium, hæmothorax, hæmoperitoneum or intracranial hæmorrhage. Rupture into a joint is of a similar nature, but the hæmorrhage is less serious, owing to the resistance of the capsule.

When the sac ruptures subcutaneously, one of two results may follow. The blood may diffuse itself widely along the planes of cellular tissue of the part, separating and compressing structures, arresting circulation, and eventually causing gangrene, if the patient does not succumb from the acute anæmia. Such a case is well called a "ruptured aneurysm," and is similar in its nature and effects to a "ruptured artery." But the rupture of the sac may be a small one, permitting what may be called a leaking of the blood into the tissues. This blood may coagulate, and a new spurious sac be formed around it, partly by the fasciæ of the part, partly by the products of the inflammation the extravasation has excited. Such a spurious sac is not an effective barrier to the escape of more blood, and usually a further rupture soon ensues. These cases have been grouped together and all called "diffused aneurysms," but the varying extent of the rupture of the sac and the variation in the subsequent amount and force of the escape of blood into the tissues, upon which the symptoms and effects depend, justify a separation of these cases into two classes. Where the sac has ruptured freely it should be called a "ruptured aneurysm," and where the sac has given way to a slight extent only it may well be called a "leaking aneurysm."

(2) *Suppuration.*—The inflammation excited by an aneurysm may become acute and end in suppuration of the tissues adjacent to the sac; this is most often seen where, as in the axilla, the aneurysm is surrounded by loose cellular tissue, and where a slow rupture of the sac has occurred; it may also be excited by prolonged or rough handling of the aneurysm, or by sudden coagulation of the blood in a large sac. The pus thus formed tends to reach the surface, and to burst externally like that of any acute abscess. Two special effects of the suppuration, however, must be noticed: (*a*) As the sac of an aneurysm, like an artery, derives its blood-supply from vessels which reach it on its outer surface, the occurrence of suppuration immediately around it, by destroying these vessels, deprives the sac of all nourishment and it therefore sloughs; this sloughing is the effect and not the cause of the surrounding suppuration. (*b*) The inflammation extends along the sac to the artery and excites arteritis; if this assumes the plastic form it seals the vessel, and when the sac separates the aneurysm is cured; but if it is suppurative such a closure of the vessel does not occur, and either before or after the abscess around the aneurysm has burst, the artery opens into it, and fatal hæmorrhage occurs. This latter is the more frequent event of the two.

Signs and diagnosis.—The characteristic features of an uncomplicated sacculated aneurysm will first be considered, and then those of its varieties and complications. The facts already noticed afford a ready explanation of all the points we shall have to mention, and if the essential features of an aneurysm are kept in mind it is comparatively easy to avoid errors in diagnosis.

(*a*) **Signs and diagnosis of an uncomplicated sacculated aneurysm.**—The primary phenomenon is a *tumour* placed over and fixed to an artery; it varies in shape, but is usually more or less globular. In size it ranges from a small nut to a cocoanut; if placed deeply or containing much clot it is firm in consistence, but if superficial or containing little clot, it is softer, and may even fluctuate.

This tumour *pulsates*, being expanded and rendered more tense with each beat of the heart. The force of the pulsation depends partly upon that of the cardiac contraction, but more upon the proximity of the aneurysm to the heart, the size of the sac, and the amount of clot within it. Ballance and Edmunds have shown by experiment that the effect of the varying blood pressure of the arterial circulation is much greater in an aneurysm than in the artery, each beat of the heart raising the pressure in the aneurysm more than it does in the artery; this accounts for the marked and striking pulsation of aneurysms. It may be more marked in some situations than others, owing to irregularity in the disposition of the clot; but so long as the aneurysm has any cavity into which blood is forced with each systole, the pulsation is of an *expansile character*. If the limb is depressed, the force of the pulsation and the tension of the aneurysm may be noticed to be increased, while if it is raised both are diminished; this is owing to the expansion and contraction of arteries which takes place in dependent and raised limbs respectively.

When the main artery of the part is compressed above the aneurysm, so as to arrest the flow of blood through it, the pulsation in the tumour of course ceases, and the *tumour shrinks and becomes less tense;* by gentle pressure the surgeon may then be able to empty the sac still more. If now the pressure is taken off the artery above, the tumour rapidly fills out again, and in two or three beats resumes its former size and tension. This shrinking of the tumour when the circulation is arrested is due to the elastic recoil of the stretched sac and adjacent tissues, and the degree to which the tumour can be emptied is a measure of the amount of fluid blood it contains. *This sign is the most distinctive feature of aneurysm.* Compression of the main artery just beyond the tumour is said to cause the tumour to become more tense.

In nearly all, but not all, cases a *bruit* is heard over an aneurysm; it is produced by the blood rushing into or out of the aneurysm, and is varied by the position, size, and shape of the mouth, and the disposition of the sac and clot within it. The bruit is most often systolic in time and blowing in character; it is heard equally well all over the tumour, and is not increased by moderate pressure of the stethoscope; occasionally it is diastolic as well as systolic, and it may be harsh or musical in character. The rush of blood into the aneurysm may also cause a *thrill* in the sac with each heart-beat; this thrill is only felt over the tumour. The *pulse* in the artery beyond, when compared with that on the opposite side of the body, is delayed in time and usually is lessened in force; sometimes it is absent, owing to obliteration of the artery. The sphygmogram may be characteristic (Fig. 174). The *heart* is usually hypertrophied, and the *arteries* are often found to be atheromatous.

Various *pressure-signs* may be met with; the *veins* below the aneurysm may be distended and varicose, and the subcutaneous tissue œdematous. *Muscular weakness* and *wasting* are frequently noticed, owing to defective nutrition, and *paralysis* may occur from pressure on nerves; this increases the wasting. Striking examples of the effects of nerve pressure are the dilated pupil, and unilateral abductor palsy of the larynx from pressure on the cervical sympathetic and recurrent laryngeal nerves respectively.

The *pain* of aneurysm may be slight or very severe. It is of two kinds: a constant deep aching, boring, or burning pain in and around the tumour, caused by the peri-aneurysmal inflammation, by the tension of the parts, and especially by absorption of bone; and a sharp lancinating pain referred to the areas of distribution of sensory nerves that may happen to be compressed; tingling, numbness, and, rarely, anæsthesia may be met with.

The diagnosis of such an aneurysm rests upon the determination of two points: *the existence of a circumscribed tumour, and the communication of that tumour with an artery.* The pulsation of a normal, or enlarged, or over-pulsatile, or displaced artery may be mistaken for an aneurysm, and this is especially the case in the condition of "pulsating aorta," frequent in women, and often associated with dyspepsia. To remember that the detection of a distinct tumour is the first and essential step in the diagnosis of an aneurysm will always prevent such an error. The communication of a tumour with an artery is shown by the expansile nature of the pulsation, and especially by the shrinking of the tumour when the artery above is compressed, and by its gradual filling out when that pressure is removed; the blowing character of the bruit and the facts that it is heard equally well all through the tumour, and is not affected by moderate pressure of the stethoscope, are other useful evidences of this condition.

Many other tumours pulsate, either from close proximity to an artery or from extreme vascularity.

A tumour over an artery has a heaving, but never an expansile pulsation, and it is often only noticed in certain parts of the tumour, or in certain positions of the limb; if the tumour is sufficiently mobile, it may be lifted away from the artery, and the pulsation in it at once arrested. When the artery above is compressed, such a tumour does not shrink, and when the pressure is removed and the pulsation returns, no increase of the tension of the tumour results. These facts show that the tumour does not communicate with the artery. Such tumours may produce a bruit by compression of the artery, but it can be distinguished from an aneurysmal bruit. In the first place, it is not blowing in character, but rasping, like that produced by compressing any artery with the end of a stethoscope; then the bruit is often only heard just along the line of the artery, and not over the whole tumour; and lastly, if the end of the stethoscope is so held as to press the tumour against the artery, the bruit is increased in intensity, until the pressure is great enough to occlude the vessel, when, of course, it is lost altogether; pressure on the tumour in any other direction has no such effect. Where the tumour is *cystic*, the signs may be modified in one or two important ways. Thus, if the cyst envelop the artery and its tension is not high, the pulsation may be slightly expansile, owing to the throb of the artery indenting the wall of the cyst over it and displacing a certain quantity of fluid; but this expansion is not to be compared in degree with that in an aneurysm of a similar tension. Again, if the cyst communicates with another cavity, as some cysts near joints communicate with the articular synovial cavity, the tumour can be emptied and then fills out again. But this emptying and refilling can be distinguished from that of aneurysm by noticing three points: (1) that compression of the artery above does not cause shrinking of the tumour, nor return of circulation fill out the tumour if it has been emptied;

(2) that the cyst is emptied by steady gentle pressure equally well whether the artery above is compressed or not, and that it fills out steadily without pulsation, not *per saltum;* (3) the fluid expressed from the cyst may be detected in the joint. By attention to these signs, then, tumours over and receiving pulsation from arteries can be shown not to communicate with the arteries, and, therefore, not to be aneurysms.

Tumours which pulsate from their extreme vascularity, such as some forms of sarcoma and pulsating bronchocele, differ from aneurysms in the pulsation being less markedly expansile, and in the fact that when the main artery supplying the part is compressed and the pulsation stopped, the tumour does not shrink, nor can it be reduced in size by pressure, and when the compression of the artery is removed, the pulsation returns instantly to its former degree—the tumour is not appreciably filled out. If there be a bruit in these pulsating tumours, it is of a soft whiffing character, and is usually more intense in some parts than in others; it is not modified by pressure. A slight thrill may be perceived. In some cases other signs at once establish a diagnosis; for instance, the tumour may be situated quite away from any large artery, as on the side of the knee, or it may be distinctly movable over the neighbouring artery, as a bronchocele, or pulsation may be noticed in part of the tumour only, the rest of it being very firm, or even hard in consistence, or special signs, as of expansion of bone, may be present.

A pulsation and thrill in the veins of the part, and a loud rasping bruit conducted along the vessels, would indicate *communication of the aneurysm with a vein.* (*See* Arterio-venous aneurysm, page 633.)

Enlargement of the tumour, without loss of its distinct outline and with increase in the intensity of pulsation, are the signs by which *growth of the aneurysm,* as distinguished from rupture of the sac, is recognised.

(*b*) **Signs and diagnosis of an aneurysm undergoing cure.**—When an aneurysm is undergoing cure by becoming gradually filled with clot, the tumour becomes smaller and harder, with less marked pulsation; its compressibility, when the circulation is arrested, is diminished, and the bruit and thrill are modified or lost. When the aneurysm is quite full of clot, but the artery still remains patent, the pulsation is heaving and has lost all its expansile character. The tumour is then quite incompressible; and if a bruit is heard over it, it is increased in intensity by gentle pressure. At the same time the local pain and any pressure-signs that may have been present diminish or disappear. The fixity of such a tumour to the artery, and, when known, its previous condition and the general clinical history, serve to distinguish it from a gland or other solid tumour over an artery. When the case passes into one of cure, by the artery becoming obliterated, pulsation disappears, and the tumour shrinks still more, while afterwards the collateral arteries may be felt to be enlarged. When cure takes place more suddenly by embolism, or by rapid coagulation in the artery and the sac, there is often sudden pain in the part, as well as abrupt cessation of the pulsation, bruit, and thrill in the tumour, and a sudden failure in the circulation in the part beyond, shown by coldness, pallor, loss of pulse in the arteries, weakness, and numbness. The tumour then gradually shrinks, and meanwhile the anastomotic vessels enlarge, and the circulation in, and function of, the parts beyond are restored.

(*c*) **Signs and diagnosis of rupture of the sac of an aneurysm.**—The signs of subcutaneous rupture of the sac of an aneurysm are increase in the size of the tumour, with loss of its definite outline—due to the escape of blood around the sac—and diminished or abolished pulsation in the tumour, for the pulsation depends upon the resistance of the sac. If the rupture is small—a mere leak—these are all the signs it presents, and the rapid enlargement of the swelling is distinguished from that caused by growth of the aneurysm by the less clear outline of the swelling, and by the diminished, instead of increased, pulsation. If the extravasation of blood is more extensive, but is still surrounded by a spurious sac, there is noticed a sudden increase in size of the tumour, which presents very little, if any, pulsation, and perhaps both bruit and thrill are lost. The coagulation of the blood in the tissues may make the swelling firmer in places, and the tumour ill-defined. It increases rapidly, and when it reaches the skin, it points, and often fluctuates like an abscess before it bursts. Such a rupture is attended with pain, the circulation in the parts below is considerably interfered with, the pulse in the arteries is very weak or lost, the veins are full, and the cellular tissue is œdematous. The pressure on the nerves makes the parts benumbed and heavy or motionless. When a wide rupture of the sac occurs, and the blood is poured freely into the tissues, the patient usually experiences a sudden acute pain in the part, and he turns cold, pale, and faint. The parts about the aneurysm become very greatly swelled, livid, and cold; all pulsation, bruit, and thrill are lost. The arteries below are pulseless, and the tissues livid, œdematous, and insensitive, and quickly become gangrenous from entire arrest of the circulation. The signs, in fact, are two: those of severe internal hæmorrhage, and the rapid accumulation of blood in the tissues under such pressure that all circulation in the parts is stopped. There is no local heat or other evidence of inflammation. The three degrees of rupture here indicated might be distinguished as "leaking," "diffusion," and "rupture" of an aneurysm.

(*d*) **Signs and diagnosis of suppuration of an aneurysm.**—The inflammatory œdema around the aneurysm causes an increase in the swelling, which obscures the outline of the tumour, and makes the pulsation, bruit, and thrill less apparent. At the same time the part is hot, red, acutely painful, tender, and it pits on pressure. There are also a high temperature, and the other phenomena of acute inflammatory fever. When pus forms, the swelling fluctuates and "points"; and when it is evacuated, chocolate-coloured pus, mixed with fibrinous coagula and sloughs, escapes, and subsequently free arterial hæmorrhage may occur. If, before the abscess bursts, the artery opens into it, there is a sudden increase of the swelling with great increase in the force and superficiality of the pulsation. The signs of suppuration and rupture are closely alike, for both are attended with increased swelling, diminished clearness of outline of the tumour, and lessened pulsation. In rupture, however, the part is cold, there is no fever, and the circulation in the parts beyond the aneurysm is obstructed; in suppuration the part is hot and red, and the patient is feverish, and the circulation beyond may be but little interfered with.

Treatment of aneurysm.—In treating an aneurysm the surgeon should imitate nature in her methods of curing the disease. We have seen that cure is only obtained when the portion of artery from which the aneurysm springs is obliterated. This may be accomplished (1) by lessening or removing the obstacles to the coagulation of the blood within the sac of the aneurysm, and to the extension of the clot into the artery; (2) by exciting coagulation within the sac; or (3) by occluding the artery from which the aneurysm springs. All the numerous methods of treatment are effective only as they lead to one or other of these results. They are conveniently divided into *constitutional* and *local* methods of treatment.

Constitutional treatment.—Constitutional measures are of great value in reducing the tension within an aneurysm, and by this means favouring the coagulation of the blood within it. They are also employed with a view of increasing the plasticity of the blood, and of combating the disease of the arterial wall. In both of these directions their value is much less certain.

To reduce arterial tension two methods are principally relied upon—the lessening of the force of the heart, and the diminution of the total quantity of blood.

(1) *To lessen the force of the heart* the patient must be kept at *perfect rest*—physical, mental, and emotional. He should lie as nearly horizontal as is compatible with comfort, all his wants should be attended to, so that he shall make no movement to help himself. He must not sit up to take food, or empty the bladder and rectum; and in some cases he has been forbidden even to raise a handkerchief to his face. He must not be allowed to transact any business, nor be excited by books, conversation, or other influences. In cases of excited action of the heart some good may be obtained by giving aconite or belladonna internally, and by wearing a belladonna plaster over the præcordia. Opium is often of great service in relieving pain, and in allaying physical and mental restlessness.

(2) *To reduce the quantity of the blood* it is generally sufficient to place the patient on a *dry and restricted diet*, such as the following: 6 oz. of bread, 2 oz. of meat, a little butter, and 6 oz. of milk or water *per diem.* It is better to reduce the diet gradually than to cut it down to this point suddenly, and care must be taken not to distress the patient too much by these restrictions, lest the good

effect of the diet is counteracted by the evil influence of fretfulness and worry. When the patient is plethoric, and the arterial tension very high, repeated saline purges and venesection are useful.

This treatment has sometimes been pushed to an extreme point, and the patient has been so reduced by purgings and repeated bleedings, that he fainted if raised in bed, and the tension of the circulation has been at the minimum consistent with life. Such a course would only be pursued in cases of internal aneurysm, in which no local measures could be adopted; and even in them physicians refuse nowadays to carry their treatment to the point sanctioned by Valsalva and others. But while this extreme measure is to be deprecated, the wise employment of constitutional means to reduce arterial tension should never be omitted or neglected.

To increase the plasticity of the blood has been the second aim of constitutional treatment. It has been thought that by a dry restricted diet, such as that just mentioned, or by a very richly nitrogenous diet, the proportion of fibrin in the blood can be increased, and the natural cure of an aneurysm thereby promoted. Apart from the consideration that so little is as yet definitely known of hæmopoiesis and the means of influencing it, that much uncertainty must be felt on the efficacy of treatment to attain this end, it is important to bear in mind that cure of an aneurysm is never hindered by lack of fibrin in the blood, and that the essential condition for coagulation of the blood is a setting free of the fibrin-ferment from within the corpuscles in which it normally lies. Even supposing that by constitutional means we could cause a breaking-up of these "ferment corpuscles," we should still do more harm than good unless we could ensure that it should only occur in the aneurysm and the artery adjacent—a condition impossible to secure by constitutional means alone.

Iodide of potassium in full physiological doses (ʒij *per diem*) has been much vaunted, but there is great divergence of opinion as to its value. If syphilitic arteritis is the cause of the aneurysm, the iodide is said to exert a beneficial influence upon this disease, arresting its progress and even leading to absorption of some of the inflammatory deposit. But inasmuch as syphilitic arteritis is attended with great thickening of the intima, and only leads to aneurysm when absorption of this new tissue has weakened the artery, it is difficult to appreciate how iodide of potassium can do good by causing the absorption of the syphilitic products. Indeed, when an artery has been so softened that an aneurysm has developed, it is open to question whether any effective repair of the vessel wall can occur. The beneficial effects that have followed the use of iodides in full doses have been probably due to their depressing action on the heart and their power of reducing arterial tension rather than any more direct action upon the diseased vessel. Other drugs, especially acetate of lead, and perchloride of iron, have been employed in aneurysm; but nothing definite is known of their value.

In conclusion, then, we may sum up by saying that constitutional treatment—when not pushed to an extreme barely compatible with life—is very valuable in reducing the arterial tension; but that its beneficial effect upon the composition of the blood and upon the disease of the artery is open to doubt.

Local treatment.—The simplest local treatment is to raise the part, if a limb, and to bandage it with gentle pressure over the aneurysm. By these means the arteries of the part are made to contract, the tension in the aneurysm is lowered, the sac is supported, and the resiliency of it and of the surrounding tissues is supplemented. Combined with judicious constitutional measures this is all that is required to cure some cases, and unless there are special conditions of urgency, these means may be carefully tried in all appropriate cases before adopting more active local measures. These are many, and we will consider them in the following order:—

1. Treatment by compression.
2. Treatment by ligature.
3. Excision of the aneurysm.
4. The introduction of coagulating agents.
5. Amputation.

1. Treatment by compression—

(*a*) Digital compression.
(*b*) Instrumental compression.
(*c*) Flexion.
(*d*) Esmarch's bandage.
(*e*) Temporary ligature.

By these various forms of compression a temporary diminution or arrest of the circulation through the diseased artery and the aneurysm can be obtained without, it is hoped, any permanent block in the arterial system elsewhere, and (with one exception) without the risks inherent to a cutting operation involving blood-vessels. Before the introduction and perfecting of the aseptic treatment of wounds, compression occupied a much more important place than it does to-day: asepsis and the ligature of arteries without rupture of their inner tunics have abolished the perils which formerly attended the operation of ligature, and which were avoided by the use of compression. We shall see that compression and ligature are closely alike in the processes by which they lead to the cure of an aneurysm, that there is no difference in the principle of the two methods, and that the choice between them is determined by secondary considerations.

(*a*) *Digital compression* is the best means of temporarily controlling the flow of blood through an artery. In order that the pressure of the thumb shall be easily efficient, it is necessary for the artery to be placed not too deeply, and to be well supported by bone. This greatly limits the positions in which digital pressure can be successfully employed, and its applicability is further reduced by the fact that relays of assistants are required to carry it out. It is employed as follows: the part, if hairy, is carefully shaved and well dusted with boric acid powder or French chalk. A spot where the artery to be compressed is most superficial is chosen, and the thumb is placed upon it with just sufficient force to stop all pulsation in the aneurysm; it is not necessary completely to occlude the artery, and still less is it right to use such pressure as will light up inflammation or cause sloughing. The hand of even the strongest man quickly wearies unless the muscles are relieved by resting upon the thumb a weight of about 6 lbs.—a conical bag of shot is the best weight to use—then the pressure may be kept up for 20 to 30 minutes. Three assistants should be in attendance together: one compressing the artery, one with his hand on the aneurysm to regulate the amount of compression and to note its effect, and the third resting and ready to relieve the compressor. When the change of hands is made care must be taken to have the artery controlled by the fresh man before the weary one raises his thumb; and this, not only to protect the aneurysm from a sudden return of pulsation in it, but to secure that the pressure shall not be made upon exactly the same portion of skin and artery. The arteries that can be compressed digitally for the treatment of aneurysm are the common femoral, the common carotid, the subclavian, the axillary, and the brachial.

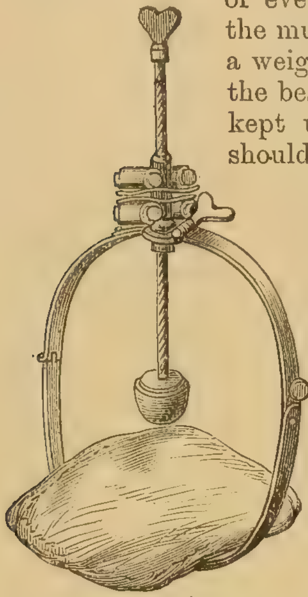
Fig. 175.—Carte's Artery Compressor for the Femoral Artery.

The advantages of digital over other forms of compression are, that it is *less painful* than instrumental; *more exact*, for, in many cases, the pressure can be brought to bear upon the artery without involving the companion vein or nerves; the pressure can be *better regulated* both as to position, direction, and amount by the finger than by an insentient compressor, and the skin of the thumb is less liable to gall that of the patient than any other substance is. Digital pressure can also be employed in two situations at least, where instrumental pressure cannot—to the common carotid and subclavian arteries. On the other hand there are many situations where it cannot be used, and in all cases it is troublesome to carry out owing to the relays of assistants required.

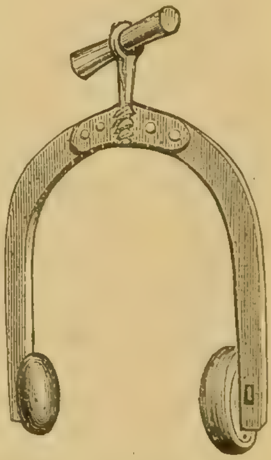
Fig. 176.—Signoroni's Tourniquet.

(*b*) *Instrumental compression* is carried out by various forms of tourniquets and artery compressors, which replace the thumb more or less efficiently; the best are a conical bag of shot and Carte's compressor (Fig. 175), in which the force is elastic, and the direction can be well regulated. Signoroni's tourniquet (Fig. 176) and Lister's aorta compressor (Fig. 177) are also sometimes of use. In using any form of instrumental compression, great care must be taken to preserve the skin from galling and sloughing, and, if possible, to compress the artery only (not the vein) with just enough and not too much force, and so to fit on the compressor that it will not shift or allow any alteration of the direction of pressure; the pressure is best made on two parts of the vessel alternately, using two instruments, one of which is adjusted before the other is raised. To effect all this requires the constant attention of a skilled assistant, and it should not be left to the patient to carry out the treatment. Instrumental compression is more painful than digital, and more difficult to carry out without accident or temporary remission; it can be employed in some situations where no other form of pressure is available—*e.g.* the aorta.

The facility with which either form of compression can be carried out is greatly affected by the disposition of the patient; the pressure is painful, and the entire repose of the patient, which must be rigidly enforced, is irksome; sedatives and narcotics are often necessary, but with a restless, irritable patient, successful compression of an artery is exceedingly difficult to carry out, and may be impossible.

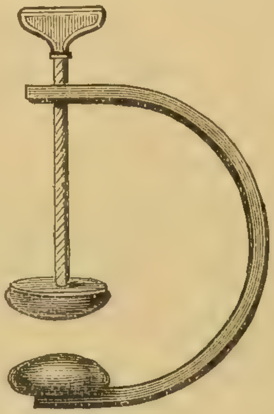
Fig. 177.—Lister's Aorta Compressor.

The *effects of compression of the artery* on the proximal side of an aneurysm, whether digital or instrumental, vary considerably. They may be nil, and even when the compression has been long continued, its removal may be followed by the return of pulsation in the sac just in its former intensity, and no benefit is obtained. In other cases it is found that the aneurysm *gradually* becomes firmer and smaller, and the pulsation less marked and less expansile, until it ceases altogether, and the solid pulseless aneurysm wastes to a fibrous knot on a permanently obliterated vessel. When this is the case, successive acts of coagulation have occurred as a result of the diminution of the tension in the aneurysm, and each clot as formed has been flattened out against the sac, forming "laminated clot," until at last the sac has been filled, and the clot has then extended into and plugged the artery. The clot is formed by successive acts of coagulation, each one being represented by a lamina, and being due to some specially favouring condition of the circulation. Evidence of this can be found in the remains of white corpuscles found between the various laminæ. The clot is not, therefore, a gradual growth of coagulum. Further evidence of this lies in the fact that the pulsation always finally disappears suddenly. As soon as the pulsation is wholly heaving in character, showing that the sac is filled, the pulsation no longer gradually diminishes in force, but ceases suddenly, if at all, from a clot filling the artery.

In other cases—and these form a large proportion of those that are successful—it is noticed that the aneurysm within a short time *suddenly* becomes solid and pulseless, and subsequently undergoes the shrinking and hardening indicative of cure. Here a single act of coagulation has filled both aneurysm and artery with clot, which subsequently organises. These different effects upon the aneurysm are due in part to the varying degrees in which the pressure employed controls or arrests the circulation in the aneurysm, and partly also to the varying "tendency of the blood to coagulate," including in this term possibly varying conditions of the sac and its lining, and of the ferment-containing corpuscles. Compression of the main artery may merely reduce the pressure of the blood in the artery beyond, the blood still flowing into and out of the aneurysm, but with little force, and in a continuous non-pulsatile stream, having either flowed on through the partly-closed artery or through collateral vessels. On the other hand, it may arrest the arterial circulation so effectively that the blood in the aneurysm is stagnant. When the varying degrees of pressure applied, and the varying number and size of anastomosing channels through which the blood can find its way are borne in mind, it becomes at once clear that the effect upon the circulation of compression of a main artery must vary.

But an aneurysm that has become partially or wholly solid does not always become cured. It has often happened that shortly after such a change has been observed, the sac has been found of its former consistence, and pulsating as forcibly as ever. This is because the clot has been disintegrated by the force of the blood and carried away, partly in the form of minute embolisms. The clotting of the blood is only the preliminary step to cure, which depends solely upon the organisation of the thrombus.

Two other effects of arterial compression must be noticed. If it is long-continued or often repeated, the collateral vessels enlarge and permanently take an important share in the blood supply of the part. As a result of this, the blood pressure in the aneurysmal artery is permanently lessened, and the effect of occlusion of the main artery is then less marked upon the whole part, including the aneurysm. It follows from this—and it is most important to bear it well in mind—that the beneficial effect of compression is greatest when first employed, and steadily diminishes with long continuance or frequent repetition, and that its long use lessens the prospect of cure from subsequent ligature of the artery. If long continued and too violent, periarteritis, arteritis, or phlebitis may be excited, and in one case at least a communication has been known to form between the artery and vein—aneurysmal varix—at the point of pressure.

The *mode of practising compression* varies. It may be continuous or interrupted, or digital compression may alternate with the use of a compressor, or the amount of compression may be diminished to allow the patient to sleep. Continuous compression is to be preferred in all cases where the patient can bear it, and the requisite arrangements can be made. Opium may be given to allay pain and restlessness, and to secure sleep; and when considerable pressure has to be employed, especially in controlling the abdominal arteries, the patient may be kept under the influence of an anæsthetic while the compression is applied. In any case in which compression is employed, care should be taken to choose the most suitable form, and to try it thoroughly; if it fails, it should not be frequently repeated, nor continued long. If it succeeds in procuring a clot, great care must be taken to guard this from disintegration, by keeping the part at rest with moderate compression of the artery for two or three days. When compression fails, ligature may often be successfully practised, but it influences the result of the operation in several ways. The ligature should never be placed, if possible, exactly where an artery has been long compressed, as the parts may be found matted together, and the risk of injury to the vein is thereby increased. The enlargement of the collaterals diminishes the risk of gangrene after ligature, but, as we have seen, it lessens the prospect of cure of the aneurysm.

(*c*) *Flexion.*—Aneurysms in the ham or at the bend of the elbow may often be rapidly cured by fully flexing the joint for several hours. By this means the sac is compressed, and the artery is occluded partly by the pressure of the sac, partly by the bend in its course. In these circumstances the blood may coagulate *en masse.* If the flexion is less acute, the flow of blood through the aneurysm may be so moderated that successive acts of coagulation occur, and the sac is gradually filled with laminated clot. This treatment has the merits of simplicity, safety, and rapidity, and it is attended with a minimum of discomfort. It is especially adapted for small slowly-growing aneurysms, and should never be employed when the tumour is of large size, rapidly-growing, or threatening to become diffused. The limb should be evenly bandaged up to the joint, and then fully flexed, and fixed either by a bandage, a strap and buckle, or heavy sand-bags. Frequent examination of the part should be made, and as soon as the sac is found to be consolidated, it is to be protected from the full force of the blood, either by moderate flexion, or by some form of compression of the artery higher up.

(*d*) *Esmarch's elastic bandage* (Reid's treatment) may be used to secure stasis of the blood in the aneurysm and adjacent artery, in the hope that the stagnant blood may coagulate, and the thrombus subsequently organise. It is only applicable to aneurysms below the groin and axilla. The elastic bandage should be carried firmly from the fingers or toes, nearly up to the tumour, and then, missing the tumour altogether, continued firmly round the limb above for a short distance. Sometimes the bandage is carried lightly over the aneurysm, so as slightly to compress the sac, and perhaps also empty the companion vein. The bandage should be left on for an hour and a-half—not more—and the pain subdued by morphia or chloroform; and if, on its removal, the blood is found to have coagulated within the sac, the clot must be protected for two or three days by moderate compression of the main artery. This treatment differs from all other forms of treatment of aneurysms, in that for the time it entirely arrests the circulation in the part. The advantages of this method are its simplicity and rapidity. Its disadvantages are that it is so painful that it is usually necessary to administer an anæsthetic; it often fails; it may modify the general blood pressure to a serious extent, and, by causing rupture or thrombosis of the arteries around the sac, it may interfere with the anastomotic circulation, and cause gangrene; and it may rupture the sac. It is ill-adapted for patients with disease of the heart or internal aneurysm. It does not lessen the prospect of cure by subsequent ligature.

(*e*) *Temporary ligature.*—This is really a form of compression, for it differs from all other forms of ligature in not aiming at a permanent occlusion of the artery operated on. It may be employed in the form of *acupressure*, a long stout curved needle being passed well beneath both artery and vein, and a pad fastened over the artery by thread tied round the ends of the needle. A much better plan is to expose the artery in the usual way for ligature, pass a *wire ligature* beneath it, and bring the ends of the wire out through the skin on each side of the wound, about an inch apart. A piece of cork can then be placed over the artery, and the ends of the wire twisted over it sufficiently tight to impede, but not to arrest, the flow of blood through it. Other forms of ligature may be used. These methods are perhaps ingenious, but they have nothing else to commend them. They are attended with a certain risk of embolism from a clot forming above the ligature and being afterwards carried on, they may cause permanent occlusion of the artery from the contusion of its walls, and they are attended with the risks common to all cutting operations. An aseptic ligature, properly applied, is distinctly to be preferred.

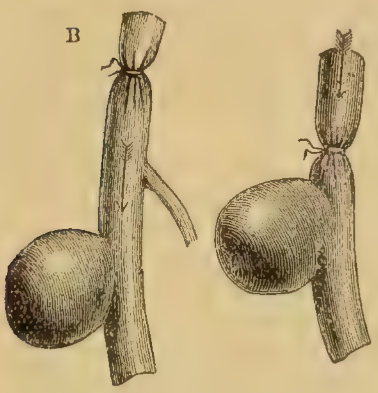

Fig. 178.—Ligature of Arteries for Aneurysm.

A, Anel's operation; B, Hunter's operation.

2. **Treatment by ligature.**

The proximal ligature cures by causing a permanent occlusion in the artery on the cardiac side of the aneurysm, and such a diminution of the tension in the sac, and slowing of the flow through it, that clotting occurs, and eventually spreads into and permanently blocks the artery. The ligature may be applied quite close to the sac, as was first done by Anel, in 1710, for a traumatic aneurysm of the brachial artery at the bend of the elbow (Fig. 178, A); or it may be done at a distance above the aneurysm, as was first done by Hunter in 1785, who tied the femoral artery (and vein) in the middle of the thigh for a popliteal aneurysm (Fig. 178, B). Since that historic date the Hunterian operation for aneurysm has been attended with greater success than any other, and its details must therefore be carefully considered.

As compared with Anel's method, Hunter's operation claimed three conspicuous advantages —(1) It was *easier* to cut down upon and tie a normal artery than to expose a vessel close to the sac of an aneurysm which might have seriously displaced parts, and even overlapped the part of the vessel sought; (2) it was free from the danger of *injury to the sac*, either by the surgeon's knife or from inflammation around it. In pre-antiseptic days, when nearly every wound suppurated, this was a very grave danger. An incision which exposed the sac was very liable to be followed by suppuration around the sac, which then sloughed. With our present knowledge and means of preventing infection of wounds this danger pertaining to Anel's method has disappeared. (3) The third advantage claimed was that the artery at a distance from the aneurysm was more likely to be healthy and safely tied than at a spot close to the sac. There is no anatomical proof of this statement. Arteries close above aneurysms are not found to be more diseased than at some spot nearer the heart chosen at random. Further, with the use of aseptic animal ligatures, especially if so tied as not to sever the inner coats, even diseased arteries may be safely ligatured. To-day, therefore, the advantages that a Hunterian operation presents over Anel's are reduced to this—that in many cases it is easier of performance, and that it does not expose the sac to the risk of injury. (*See also* page 519.)

The Hunterian operation. (1) *Effects.* —As a rule, branches come off from the artery between the seat of operation and the aneurysm, and when the ligature is tied, the anastomosing vessels enlarge, and pour blood through these branches into the main artery below the obstruction. This blood flows on in a gentle, usually pulseless, stream past the mouth of the sac, and into it, if still patent. This flow becomes more and more free as the anastomosing

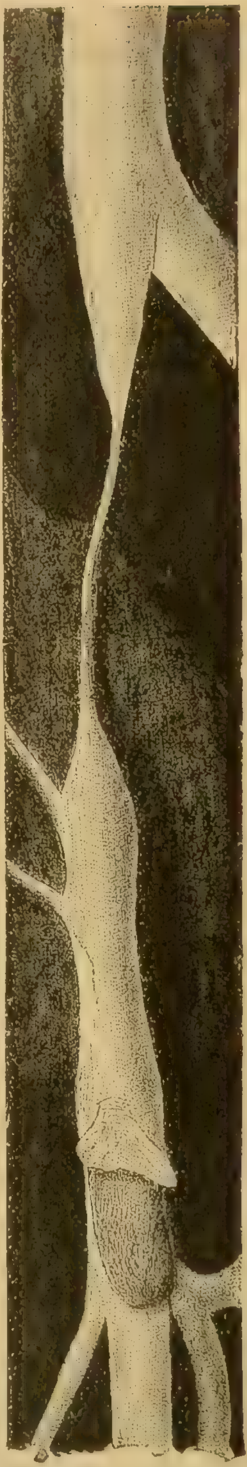

Fig. 179.—Showing the Result of Ligature of the External Iliac Artery, for Aneurysm of the lower end of the Common Femoral Artery. A considerable length of the iliac artery is obliterated. (Ballance and Edmunds.)

vessels enlarge, and by it the limb below the ligature is nourished. The force of the circulation being thus removed, the blood in the aneurysm coagulates, and the clot extends into the artery, and causes a second block in the main blood channel. This thrombus organises, and so leads to the cure of the aneurysm. What we have seen above in considering the effects of compression (page 613) as to the gradual or sudden occurrence of this complete coagulation is equally true when the artery is tied. In some cases—the majority—all the blood in the aneurysm at once clots, and often that in the artery, too ; but in other cases the coagulation is partial, and repeated until the cure is completed. This second block in the main artery may necessitate the enlargement of a second set of anastomosing channels, to carry the blood from the patent part of the artery between the ligature and the aneurysm into the vessels below. The vitality of the parts below the aneurysm thus comes to depend upon the sufficiency of a double set of anastomosing arteries, and it is exposed to great peril. It is for this reason that gangrene is more likely to occur when an artery is ligatured for aneurysm than for any other cause.

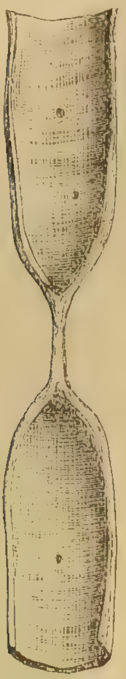

Fig. 180.—An Artery that had been tied in its Continuity, showing Obliteration of the Vessel for half an inch only.

In some cases—as, for example, in ligature of the lower part of the common carotid artery for an aneurysm at the bifurcation of this trunk —no branches intervene between the ligature and the sac. Then, when the thread is tied, the onward flow of blood into the artery and aneurysm is entirely arrested, and only a regurgitant flow from the vessels beyond the aneurysm occurs. The cure is brought about in the same way as before, by the clotting of the blood in the sac of the aneurysm now that it is freed from the force of the circulation. In these cases the clot fills the whole length of the artery from the ligature to the aneurysm, and all this part of the vessel is permanently obliterated. There is no reason to think that this clot results from a gradual growth of that formed on the distal side of the ligature. Rather it is that the whole mass of stagnant blood coagulates. Only one set of anastomoses are enlarged, from the vessels on the cardiac side of the ligature to those on the distal side of the aneurysm. Even when there are branches between the ligature and the aneurysm, clotting and permanent occlusion of the main vessel sometimes occur in the whole length of the vessel. Thus, in a case of Hunter's, in which he ligatured the superficial femoral artery for a popliteal aneurysm, the artery was found to be permanently obliterated from the origin of the profunda femoris to just above the bifurcation of the popliteal trunk ; but this result is the exception, for Savory has shown that of seventeen cases cf ligature of the femoral for popliteal aneurysm the vessel was pervious between the ligature and the cured aneurysm in fourteen. In some cases the length of artery obliterated at the seat of ligature is very small, and this is well illustrated by the specimen in St. George's Hospital Museum, from which Fig. 180 is taken.

(2) *Failure to cure the aneurysm* by proximal ligature may be due to two or three causes. Very rarely it is found that the sac remains filled out with fluid blood, no coagulation occurring, and it may even enlarge without pulsation from the blood regurgitating into the artery. More often the clotting that occurs is limited to the aneurysm, and does not extend into the artery; or, if it does, the arterial thrombus is disintegrated, and disappears leaving the vessel patent. Whenever this is so, the aneurysm is not cured even if it become solid and undergo considerable shrinkage; enlargement may at any time occur. Thus Mr. Scott, of Bath, has recently recorded the case of a man whose femoral artery was tied for a popliteal aneurysm in 1883, the tumour became much smaller, and was said not to pulsate. Eight years later it began to increase in size, and in 1894 a large aneurysm was excised from the ham, and the popliteal artery was found to be patent. Here it is clear that the first operation failed to cure the case, because the artery was not occluded, and the shrunken aneurysm afterwards yielded to the pressure of the blood.

This "recurrent pulsation," as it is called, or failure in the cure of the aneurysm, may depend upon some special relation of the mouth of the sac to the artery; but its more probable and better known cause is a too forcible stream through the artery. This may result in some cases from failure of the ligature to occlude the artery, and this may arise either from the first knot in the thread slipping before the second is tied, or from premature softening and absorption of an animal ligature allowing the artery to open out, or from the vascularisation of the scar in the artery being so free as to open up the vessel again. All these may be included under the head of failure of the ligature to occlude permanently the tied artery.

Another equally important cause is *too great freedom of the anastomotic circulation*, so that the blood is carried in a rapid and forcible stream into the artery below the ligature. Hence any circumstance which leads to a previous enlargement of the collaterals, such as long-continued trials of compression, diminishes *pro tanto* the prospect of cure by the Hunterian ligature. That this is the explanation of most cases (at any rate) of "recurrent pulsation" is shown by the readiness with which these cases are cured by compression or by religature of the artery lower down. Recurrent pulsation is more frequent with the modern aseptic absorbable ligatures than with others: this is in part due, no doubt, to the failure to occlude the artery that may attend their use; possibly also the smaller extent of the clot which forms on the distal side of such a ligature than of a septic non-absorbable one has something to do with this result. As with compression, so with ligature; the more delayed the cure of the aneurysm, the less likely is it to be brought about at all. As a curative agent the ligature is most potent immediately after its application when the disturbance of the circulation at the seat of the aneurysm is at its maximum.

(3) *The accidents that may occur after the Hunterian operation*, as distinguished from mere failure to cure the aneurysm, are (*a*) *secondary hæmorrhage* at the seat of ligature, (*b*) *gangrene,* and (*c*) *inflammation and suppuration around the sac.* For the causes and treatment of secondary hæmorrhage from an artery tied in continuity, the reader is referred to page 541. Gangrene after ligature of an artery is also dealt with elsewhere. (*See* page 534.) We may here repeat, however, that the gangrene is generally of the "moist" variety, for venous obstruction is a marked element in the case. When an aneurysm has been the cause of great venous obstruction, ligature of the main artery of the part, by greatly diminishing the *vis a tergo*, is very liable to lead to moist gangrene, extending as high as the aneurysm. "Dry" gangrene is the result of failure to establish a sufficient anastomotic circulation, or of thrombosis in a diseased artery ; it may be limited to a small area, or involve the whole limb up to the aneurysm. Inflammation around the sac is a much rarer occurrence ; it has occurred most often in the groin and axilla, where the tumours are not well supported, and particularly in the case of a large sac or where the aneurysm is becoming diffused. The causes that have been assigned are the handling to which the tumour has been subjected before and after the operation, and the presence of large masses of fibrin in the sac. The sudden solidification of a large aneurysmal sac in the midst of soft loose tissues is probably an important element in the production of local gangrene from pressure and thus of suppurative inflammation.

(4) *Symptoms after ligature.*—At the moment of tightening the ligature the aneurysm shrinks and ceases to pulsate. In the best cases it quickly becomes solid, and then gradually diminishes in size, without any return of pulsation in it. Very often, in eighteen to thirty hours, when the anastomosing vessels are fully dilated, and the limb is warm, a faint pulsation, or a trembling sensation, or a distinct throbbing is detected in the aneurysm ; this may last for hours, or even days, and then pass off, or it may increase and become permanent. These differences depend upon the extent and rapidity of the clotting ; if the whole aneurysm and adjacent artery become at once thrombosed, no pulsation is felt ; if the sac is filled and the artery patent, pulsation is felt, which varies in intensity with the force of the heart and the freedom of the anastomotic circulation.

The limb is at first cold, benumbed, and the arteries below the ligature are all pulseless ; if all goes well, sensation returns, the part becomes warmer, and a pulse can be felt in the arteries, and some of the enlarged collaterals may also be felt. If moist gangrene comes on the part remains cold, swells, becomes mottled, vesicles form on it, sensation and the power of motion are entirely lost, and with this the usual constitutional symptoms of moist gangrene develop. If dry gangrene ensues, the bloodless part remains cold and insensible, shrivels, dries, and mummifies.

(5) *Treatment after ligature of the artery.*—Immediately after ligature the limb, covered up with a thick layer of cotton wool, should be slightly raised and supported evenly on pillows. Hot bottles may be placed in the bed, but not in contact with the limb; the aneurysm should not be examined or disturbed. When the re-establishment of the circulation and the cure of the aneurysm are accomplished, these precautions may be gradually and carefully dispensed with. Should pulsation be noticed after a short time, the surgeon must have patience, as in great probability it will pass off But if it persists, or even increases, and it is evident that the operation has *failed to cure* the aneurysm, compression of the main artery with gentle pressure upon the tumour should be tried, and if the aneurysm is in the ham, flexion is very likely to succeed. If this fails, the artery should be tied nearer the sac, if possible, in such a place that no large branch shall intervene between the second ligature and the sac; should this fail or be impracticable, the aneurysm should be excised. If the sac remains full of fluid blood, but pulseless, it should be excised.

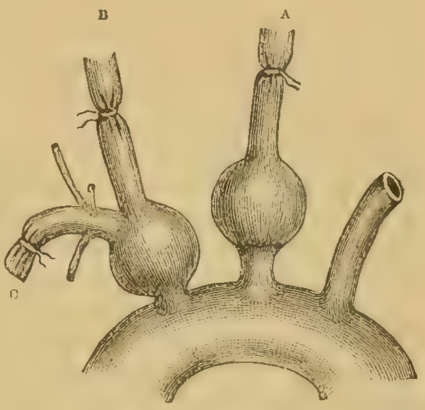

Fig. 181.—Diagram illustrating the Mode of applying Distal Ligature for Aneurysms at the Root of the Neck.

The ligature on left common carotid artery, A, is Brasdor's operation; ligature of right carotid and subclavian arteries, B and C, for innominate aneurysm is Wardrop's operation.

If moist gangrene supervenes amputation above the aneurysm should be performed at once; but if the gangrene be "dry" a line of demarcation should be awaited, and time given for the full re-establishment of the circulation before any operation is done, and then the surgeon should content himself with either removing the dead part, or only so much more as will permit of the formation of a good stump. If the ligature fails by slipping or premature softening and absorption, the wound should be opened up, and a second one applied immediately above the position of the first.

If secondary hæmorrhage occurs, religature of the artery should be practised if simple pressure fails to arrest it, and if that does not succeed, amputation above the aneurysm at the level of the ligature must be performed. (*See also* page 548.)

When the sac suppurates, the best treatment to pursue is open to question. When suppuration is only threatening, free antiseptic incision may succeed in cutting short the inflammation. When the mischief is more advanced, the choice lies between excision of the sac and amputation; where hæmorrhage has already occurred, where the aneurysm is large or deeply-placed, or the inflammation widely-extending, amputation is certainly indicated.

The distal ligature.—This is employed in two ways. *Brasdor's operation* consists in tying the diseased artery beyond the aneurysm, so as to arrest entirely the circulation through and past the sac: *e.g.* ligature of the common carotid artery close to its bifurcation for aneurysm of the root of the artery (Fig. 181, A). *Wardrop's operation* is a modification of this, inasmuch as one or more of the branches coming off beyond the aneurysm are tied, but the whole stream through the aneurysmal artery is not stopped: thus, in a case of innominate aneurysm, the common carotid and third part of the right subclavian arteries may be tied, still leaving the stream of blood passing from the innominate trunk into the branches of the first part of the subclavian artery (Fig. 181, B).

The distal ligature resembles in its mode of action the plugging of an artery by an embolus washed out of an aneurysm. It succeeds in curing an aneurysm partly by the clotting spreading back from the seat of ligature to the aneurysm, and partly by the lessened tension and stasis in the aneurysm caused by the diversion of the blood stream, allowing the natural tendency of the blood in the aneurysm to coagulate, to have full play. When the common carotid artery is tied near its bifurcation for an aneurysm lower down, not only may the clot formed at the seat of ligature extend down along the artery as far as the aneurysm, but this clotting is favoured by the fact that such a diversion of the blood stream into the subclavian artery of the same side occurs, that the tension in the carotid is diminished.

In Wardrop's operation these influences are less powerful in their action, and it is therefore less frequently successful than in Brasdor's.

In some cases, after a temporary improvement, the sac has rapidly enlarged after distal ligature, and the operation has appeared to do harm; suppurative inflammation around the sac may also occur. The distal ligature is never used where the proximal can be applied; it has been chiefly employed on the arteries of the neck for aneurysm of the aorta and of the arteries at the root of the neck. When dealing with these special aneurysms we shall have to discuss its value more in detail.

3. **Excision of the aneurysm.**—This operation is called also the "old" operation for aneurysm, the "operation of Antyllus," and the method of "double ligature," but the name we have chosen is the one that best describes it. It may be performed in two ways. The circulation in the part being controlled by a tourniquet, one method is for the surgeon to make a free incision down to the outer surface of the aneurysmal sac, and then carefully to separate all the structures from this sac, taking great pains not to injure veins and nerves. The artery or arteries attached to the sac are then carefully ligatured in turn; when all are tied they are cut on the aneurysm side of the ligatures—or they may be tied with double ligatures and divided between, one by one—and the tumour is lifted away. By the other method, the sac is laid freely open, all blood and clot within it turned out, and each end of the artery entering it and any branches passing out from it are tied. Where possible, the first method is

the best to employ. The second method, which is the "old" operation, may be one of the most formidable operations in surgery, and, up to the days of Hunter, many bold surgeons preferred to amputate the limb rather than expose their patient to the enormous primary and secondary risks from hæmorrhage, diffuse cellulitis, and gangrene. Percivall Pott spoke of the event of the operation as "always fatal," and Hunter had not a single successful case.

The experience of a century ago is not a sure guide to us to-day in any operation. With anæsthesia to secure perfect stillness of the patient, and to free the surgeon from all need to hurry; with improved means of hæmostasis, enabling us to operate without loss of blood, where formerly continuous bleeding not only exhausted the patient, but obscured the field of operation; and with asepsis, which abolishes inflammation, suppuration, blood-poisoning, and secondary hæmorrhage, it follows that we must try to form a new estimate of this operation. Viewed from our present standpoint, then, the operation has two defects: (*a*) it may be, and often is, very difficult of execution; and (*b*) it exposes the patient to danger from injury of important structures. The risks from wound infection and from hæmorrhage can generally be avoided; it has the merit which belongs to no other treatment of aneurysm—that if the operation is successfully carried out it certainly "cures" the aneurysm. Largely owing to the great success of other treatment, this operation is rarely employed, but where proximal ligature fails to cure, it should always be chosen in preference to amputation. Unfortunately it is just in those cases in which the ligature is least applicable and least successful that excision cannot be employed. Several successful cases of excision of aneurysms have recently been recorded, and it is probable that this mode of treatment will be employed more frequently in the future than in the century following Hunter's memorable operation.

4. **The introduction of coagulating agents.**—Manipulation of the sac; acupuncture; galvano-puncture; injection of coagulants; introduction of foreign bodies.

Manipulation was suggested by Sir W. Fergusson. His object was to displace the clot lining the sac into the mouth of the artery. To employ it properly the artery should be compressed on the cardiac side of the aneurysm; the sac is then inverted by the thumbs until its contents are felt to be displaced; it is hoped that the artery may be occluded, and the tumour cured by this means. The treatment has been occasionally successful, but it has been attended with such grave accidents that now it is abandoned. Employed in aneurysms at the root of the neck, there is great danger from fatal or very serious embolism; in any situation it may lead to rupture of the sac or to inflammation around it. The manipulation of an aneurysm involved in its surgical examination has not unfrequently led to its rapid solidification, and such an "accident" is always welcome; but, as an intentional mode of treatment, manipulation is not now resorted to.

Acupuncture.—By simply passing needles into the sac and leaving them for some days, coagulation has been set up in aneurysms which had resisted other means of cure. Dr. Macewen, of Glasgow, has employed the method in a more precise manner, and has unquestionably obtained admirable results. He passes his needles in such a way that by the throbbing of the aneurysm its sac is scratched against their ends. He asserts that by this scratching of the sac a white clot is produced, and he lays great stress upon the importance of this "white clot," which he says resembles the clot which can be watched to form in the capillaries of a frog's web when it is injured. It is evident that to pass needles in the way directed must be an exceedingly delicate operation, for if done carelessly the sac might be torn quite through. It is open to doubt whether a white clot, composed of fibrin and disintegrated corpuscles only, is formed, or that if it were, its presence would be of greater value than ordinary blood clot. The treatment should only be employed when compression, ligature, and excision are alike impracticable.

Galvano-puncture is employed as follows: two or more fine steel needles, carefully insulated to within one-third of an inch of their points, are introduced into the aneurysm about an inch apart, having the whole of their bare points within the sac. One needle is connected with the negative pole, and the other or others with the positive pole of a constant current battery; or all the needles may be connected with the positive pole, and the negative pole placed in contact with the skin close by. A current of low intensity, but high tension, such as is obtained from several small cells—a battery of ten or twelve Leclanché cells will do—is used, and should be continued until a decided effect is produced, such as diminished pulsation or hardening; the needles are then withdrawn and the punctures sealed with collodion. When successful, a firm clot is formed around the positive pole or poles, consisting of fibrin and coagulated albumen precipitated by the dissolved iron of the needle; this clot is acid in reaction and oxygen gas is given off at this pole. A soft, frothy, spumous, alkaline clot is formed at the negative pole. The operation usually requires to be repeated. The evolution of gas may be so abundant as even to give a resonant percussion note. Galvano-puncture has, up to the present, had the greatest amount of success among the methods now under consideration.

Injection of coagulants, such as perchloride of iron, has been practised. It is necessary that the artery should be compressed above and below the aneurysm during, and for some time after, the injection, lest the fluid pass into the general circulation and serious thrombosis and embolism result. The best of all coagulants is Wooldridge's ferment. Owing to the difficulty of guarding against the coagulant escaping from the aneurysm, the scope of this method is very limited. Langenbeck suggested the injection of a solution of ergotin around the sac, with a view of exciting contraction of the muscular fibres in its wall; but this treatment lacks both theoretical and practical sanction.

Introduction of foreign bodies (Moore's operation).—Foreign bodies have been passed into the sac of an aneurysm to excite coagulation of the blood, and to assist in filling the sac with solid matter; iron wire, catgut, and horsehair have been employed. The best material to use is fine steel wire coiled small and rendered aseptic by prolonged immersion in liquor potassæ. This can most conveniently be passed into the sac through a Southey's cannula. The success attending this operation has been exceedingly small, and the method is not regarded with favour. Cure and improvement have been reported from it, but in the great majority of cases failure has attended its use; in one case extensive sloughing of the sac followed its employment. If used at all, only a small quantity of wire should be introduced at any one time.

The introduction of coagulants is the least satisfactory of all the methods of treating aneurysm. In the first place it can only be carried out by means which involve more or less injury to the sac; if any clot is formed it may be carried away as an embolus and do harm, or the coagulating agent may itself escape into the artery. For these reasons it is most safely employed where the aneurysm can be completely controlled by compression of the artery above and below, and this is just the condition which renders the aneurysm suitable for treatment by compression, ligature, or excision. As a curative agent, therefore, this method has a very limited value. In cases of internal aneurysm, for which other surgical measures are unavailing, it may be cautiously used with the object of producing a certain amount of clot in the sac, and thereby to strengthen it and prevent its enlargement; in cases of aortic aneurysm, acupuncture and galvano-puncture have sometimes succeeded in accomplishing this amount of good.

5. **Amputation.**—Amputation is sometimes necessary for various reasons and to accomplish different purposes. Thus, in the first place, it is to be employed to remove a disease which threatens life, and which cannot be satisfactorily treated in any other way. Before the time of Hunter, amputation was often employed for popliteal or femoral aneurysm, being preferred to the risks attending the disease or any other then known treatment. Now, amputation is rarely necessary for this purpose; it may be needed in cases of inflamed and suppurating or sloughing aneurysms.

It is required, in the second place, in certain accidents attending the treatment of aneurysm—gangrene and secondary hæmorrhage. Thirdly, in very rare cases, amputation of the limb below an aneurysm may be employed to cure the aneurysm; for example, amputation through the shoulder has been performed for aneurysm of the subclavian artery. The cure is effected partly by the spreading of clot in the artery from the stump to the aneurysm, and in great part by the lessened tension in the artery caused by the reduction in the stream of blood passing through it.

The choice of treatment.—Having reviewed the various methods that may be employed, it remains now to consider the principles that should guide the surgeon in choosing the course to follow.

(1) **In external aneurysm,** rest with elevation of the part and moderate pressure with a bandage should first be tried. If not quickly successful, the most suitable form of compression for the particular case should be given a careful trial; if success is not attained, the attempt should not be prolonged, but proximal ligature should be performed. The line of conduct to be pursued, when ligature fails or is attended with accident, has already been stated (page 621).

This may be taken as the general routine plan to be followed, which may have to be modified under certain special conditions, some of which are stated below in the comparison between compression and ligature, and others are as follow:—

(*a*) If the aneurysm is rapidly enlarging, or if blood is leaking from the sac into the tissues, as shown by enlargement of the tumour with diminution of the pulsation in it, the proximal ligature should be practised without any delay.

(*b*) If the sac of the aneurysm has given way, but the blood is not widely extravasated, being still bound in by a spurious sac, the patient should be subjected to operation at once. The vessels having been emptied, and controlled by Esmarch's bandage and tourniquet, an attempt may be made to excise the aneurysm, turning out all extravasated blood, and, of course, ligaturing every divided artery. Failing this, amputation must be performed above the aneurysm.

(*c*) In cases of subcutaneous rupture of the aneurysm immediate amputation above the aneurysm is the only possible resource.

(*d*) Where inflammation around the sac has occurred, the artery should be at once tied on the proximal side. This relieves the tension, and lessens the blood supply to the inflamed part. If this does not check the inflammation, a free incision should be made down to the outer surface of the sac to set free the inflammatory and infective products. The patient must then be watched continuously, and at the first sign of hæmorrhage the artery controlled by a tourniquet. An attempt should then be made to excise the sac. If this fails, the limb must be amputated above the tumour.

(*e*) Where suppuration has already occurred, the pus must be liberated at once by a free incision and thorough antiseptic irrigation of the inflamed tissues practised. The patient must be watched, and hæmorrhage dealt with as in the last case.

(*f*) In varicose aneurysm the arteries and veins communicating with the sac must all be tied close to the sac.

(*g*) In aneurysm close to the trunk, where the proximal ligature cannot be employed, the choice lies between distal ligature, excision of the sac, and amputation, and they should be chosen in that order. The complete distal ligature should be preferred to the partial. It should be performed as near the disease as is compatible with the integrity of the sac, and if it fail, excision may be practised if the other circumstances are very favourable. Galvano-puncture or, better, Macewen's acupuncture may be employed in such a case, and as a last resource there is amputation.

(2) **In internal aneurysm.**—Careful constitutional treatment should be given a patient trial, and if the growth of the aneurysm is arrested, the surgeon should be content; but if the disease advances, two courses are open—(*a*) the distal ligature, and (*b*) the introduction of coagulating agents. Coagulants can only be used when the aneurysm is near the surface. They could not be employed, for instance, in an aneurysm of the aorta which did not extend beyond the thorax. In many of these cases the distal ligature seems to act empirically. If compression of an artery beyond the sac markedly lessens the pulsation in the aneurysm, undoubtedly the surgeon should tie that artery; or if, by the distal ligature, the circulation through the aneurysmal artery is of necessity greatly modified, the operation is indicated. As a rule, if coagulants are used in these cases, improvement rather than cure is to be anticipated.

In aneurysms in any situation the value of opium in relieving pain, and of venesection in lessening excessive arterial tension, must not be lost sight of.

On the choice of compression or ligature.—Much stress was formerly laid on a correct appreciation of the relative merits of compression and ligature in the treatment of aneurysm; but the introduction of aseptic surgery and absorbable ligatures has narrowed the grounds of choice to a very limited field, and thereby deprived this subject of much of its importance. Compression and the proximal ligature lead to the cure of the aneurysm by the same means—the sudden and complete, or the partial and repeated, coagulation of the blood in the sac and artery. Both alike may be rendered free from the risk of wound infection, and of secondary hæmorrhage.

It may be claimed for compression that it can, as a rule, be carried out without the use of an anæsthetic, and without a dreaded "operation"; that it is, by its very nature, free from the risks of wound infection and secondary hæmorrhage; and that this freedom does not depend, as in the case of ligature, upon certain precautions taken by fallible men. It can be applied so as to partially occlude the main artery, and thus to cause less embarrassment to the general and the local circulation than the complete occlusion by a ligature may do. It also does not cause any permanent obliteration in the artery; and if the aneurysm is cured, and its artery blocked, the local blood supply is only impeded by one instead of two blocks in the main arterial channel. On the other hand, it may be urged against compression that it is troublesome to manage; tedious, if not very painful, to the patient; and on the whole, less constantly successful than the ligature. The ligature, as now employed, is the most successful of all methods of treatment for aneurysm, and by the use of anæsthetics it is attended with a minimum of suffering and inconvenience. In certain positions compression of an artery is impossible, or is attended with great difficulty and danger—*e.g.* in the abdomen and the root of the neck.

Compression and ligature on the distal side of an aneurysm are attended with the same amount of risk, and the ligature has the greater chance of leading to cure, as the clot formed about the ligature may extend into the aneurysm.

The conclusions we draw from these considerations are as follow:—

(*a*) Where there is reason to fear that the sudden obliteration of a large artery may embarrass the heart too much, cause the rupture or enlargement of an internal aneurysm, or produce gangrene of a limb, moderated compression is to be preferred.

(*b*) Where the artery affected is known to be seriously diseased, compression is to be chosen.

(*c*) Where, in other cases, the patient is of an irritable or sensitive nature, and intolerant of restraint, or where the aneurysm is acute, of large size, rapidly-growing, and full of fluid blood, and it is therefore necessary to obtain a marked effect and a rapid result, the ligature is to be recommended.

(*d*) Distal ligature is always to be preferred to distal compression.

(*e*) Where compression of any particular artery is attended with special difficulty or danger, its ligature is to be preferred

(*f*) In the absence of any of these special conditions, compression should first be tried, the effect being as closely as possible approximated to that of ligature. If it is not quickly successful, ligature should be practised.

On the choice of the site of the ligature.—In all cases where it can be safely employed the proximal ligature is to be preferred to the distal, because it more certainly arrests the circulation through the sac, and takes off "the force of the circulation," or diminishes the tension in the aneurysm.

The proximal ligature may be applied close to the sac, or at some distance above it. The practice of Hunter, Scarpa, and the other great surgeons of the past century has been to choose a part of the artery well above the aneurysm, and, as a rule, to have a branch or branches intervening between the two. A procedure sanctioned by such authorities cannot be lightly set aside, but the following reasons may be urged in favour of ligature nearer the sac:—

(*a*) It is now known that it is neither necessary nor desirable for cure of the aneurysm that a stream of blood should enter the artery below the ligature, and flow gently past the sac.

(*b*) It is now known that the artery is not progressively more healthy above the aneurysm. It is as likely to be found healthy near to as far above the sac.

(*c*) Even if the artery is not quite sound where the surgeon exposes it, he can now apply a ligature in such a way as not to involve any evil consequences from the arterial disease.

(*d*) As a rule, the ligature at some distance above the aneurysm results in the formation of two permanent blocks in the main artery, with a patent portion between, and the nutrition of the limb depends upon the establishment of a double series of anastomosing channels.

(*e*) The stream of blood pouring into the artery between the ligature and the sac may prevent cure of the aneurysm.

These reasons have led surgeons lately to ligature the popliteal artery for popliteal aneurysm, and the results have been extremely good. In fact, the best site for the application of a proximal ligature is as near to the aneurysm as it can be applied without risk of damage to the sac. The greater ease and convenience with which arteries can be exposed at some situations than others, of course, must influence the choice of operation.

The distal ligature should by choice be placed close beyond the aneurysm, that there may be the greater chance of clotting spreading back into the sac, and also because in that situation it most powerfully affects the circulation in the aneurysm. Where a choice of arteries beyond the aneurysm presents itself, if all are not tied, those should be selected whose compression produces the most marked effect upon the particular aneurysm in question.

3. Dissecting Aneurysm.

This form of aneurysm is more frequent in women than in men, and in cachectic than in robust persons. It occurs most often in the aorta, and may extend down the iliacs and as far down as the femoral artery, or up into the neck as far as the bifurcation of the common carotid trunk. After extending a certain distance in the wall of the artery, the blood may burst through a softened patch of intima into the lumen of the vessel again, or it may burst externally through the outer coats and become "diffused"; on the other hand, the passage of the blood may be arrested, and the aneurysm exist only as a blind pouch or diverticulum in the coats of the artery; by projecting inwards it may then greatly narrow the artery and obstruct the passage of blood through it.

Dissecting aneurysm is very rarely *diagnosed* during life. At the moment of its formation a sudden severe pain shooting along the course of the artery is experienced, and then there is noticed enfeeblement of the pulse in the arteries beyond, and there may be syncope. There is no known *treatment* for this condition.

II. TRAUMATIC ANEURYSM.

Definition.—A *traumatic aneurysm* is a "blood tumour communicating with an artery, produced by an injury dividing all the coats of the artery." The large share that strain and other injuries take in the production of spontaneous or idiopathic aneurysm makes it important to lay stress upon the latter clause of this definition, and it is by bearing this in mind that the ætiology of traumatic aneurysm becomes at once clear.

Causes. (1) *A subcutaneous wound or rupture of an artery.*—

Such a wound may be produced in fractures by a fragment of bone tearing the artery; the ruptures are caused by contusions, strains, and sometimes in dislocations, either by the displacement of the bone or by the surgeon's efforts in reducing it.

(2) *An oblique or valvular wound involving an artery.*—These are most often stab-wounds, or wounds with broken glass. The wound may be so oblique that the blood from the first collects around the artery instead of all escaping externally. But more often the dressing that is put on seals up the outer wound or closes the track from the wounded vessel outwards, without occluding the vessel, and the blood which escapes from it accumulates around the artery.

(3) *Punctured and other incomplete wounds of arteries.*—We have seen (page 500) that a wound of an artery is only soundly and securely healed when the lumen of the vessel is obliterated; if the current through the artery continues, it either prevents the formation of clot, or displaces any clot that has sealed the wound, or prevents its organisation into a firm unyielding cicatrix. We have seen that the primary hæmorrhage from such an injury may be arrested spontaneously or by surgical means, and yet unless permanent occlusion of the artery is obtained, its healing does not take place, and an aneurysm forms. In an incomplete division of an artery this obliteration can only be certainly obtained by completing the division or by double ligature. Hence it is that the *nature* of the injury of the artery is a cause, in this instance, of traumatic aneurysm.

(4) *An artificial valvular wound of an artery due to the improper application of pressure.*—Many cases of deep wounds of arteries can be so treated as to lead to the formation of an aneurysm. Whenever healing of the overlying soft structures occurs without cicatrisation of a divided artery, this result must follow. So, even in a case of *complete* division of a vessel, if the soft parts are approximated, and such a dressing applied that they heal up under it, while the pressure is only sufficient to limit but not to arrest the flow of blood from the artery beneath, when the external wound heals an aneurysm is found beneath the scar. The reader is referred to what we have already said on this point in speaking of the treatment of wounded arteries by pressure (page 514).

Varieties.—The varieties of traumatic aneurysm—including the arterio-venous, which we deal with separately—depend entirely upon the freedom with which the blood escapes from the wounded artery. If the internal hæmorrhage is rapid and profuse, and the blood finds its way along the planes of cellular tissue unhindered by firm clot or tense resistant fasciæ, forming a huge extravasation, the condition is better spoken of as a *ruptured artery;* such a condition is only met with in subcutaneous rupture of an artery. More commonly the blood, as it escapes from the artery and infiltrates the soft tissues, forms a cavity round the artery, which is imperfectly limited by clotted blood—which becomes firm and fibrinous or "laminated" by the pressure to which it is subjected—and by the tension of the stretched and displaced fasciæ and muscles. Such a condition is known as a *diffused traumatic aneurysm.* In many other cases the escape of blood is smaller, slower, and later after the primary injury. Around the clots that form a spurious sac is developed by the inflammation excited by the extravasated blood, and by the plastic processes of the healing of the primary wound. This sac grows and yields under the pressure of the blood, and sometimes it bursts, and the condition comes to resemble that just described, or even that of a ruptured artery. The firmness of the sac, and the amount of clot which forms within it, to strengthen it and lessen the pressure upon it, are very important elements in determining the progress of the case. When healing of the wound has advanced considerably before the external pressure is relaxed, so as to allow the blood to escape from the wounded artery, a firmer sac forms around the clot than when the aneurysm begins to form earlier. The presence of tense and strong fasciæ near the artery also affects the strength of the sac formed. These aneurysms, which are limited by a distinct sac, are called *circumscribed traumatic aneurysms.* In no case of traumatic aneurysm does the arterial wall take any part in forming the sac.

Course and terminations.—A **circumscribed traumatic aneurysm,** if the sac is firm and strong, may undergo spontaneous cure in the same manner as a spontaneous aneurysm. But owing to the imperfect formation of the sac, there is a great danger of the aneurysm enlarging, and of the sac yielding, either to form a diffused aneurysm, or so freely as to lead to that widespread extravasation, best called a ruptured artery.

A **diffused traumatic aneurysm,** owing to the absence of any resistant sac, tends to grow rapidly, and it ends in one of three ways: (1) It may reach the surface and burst externally, causing severe hæmorrhage, which may be fatal at once, or after repeated recurrences. (2) It may become widely diffused in the part, the effusion of blood not being limited even by clot—*ruptured artery.* (3) Acute suppurative inflammation may be set up round the collection of blood, and when the abscess bursts, blood-clot and pus in great quantity are discharged, and then profuse hæmorrhage occurs, unless, by a rare chance, the artery has been sealed by plastic arteritis.

A **ruptured artery.**—The extravasated blood, unlimited in any way, either ruptures externally, or the tension in the part is sc great that the venous circulation is entirely arrested, and moist gangrene of the part beyond results. Fatal syncope may occur from the subcutaneous hæmorrhage.

Symptoms.—A **circumscribed traumatic aneurysm** has all the local characters of a spontaneous sacculated aneurysm; there is in addition a history of a recent injury, and a scar or unhealed wound points to the nature of the case.

A **diffused traumatic aneurysm** varies somewhat in its characters, according to the rapidity of its formation and growth, and the amount of clot around the fluid blood. In all cases it is a rapidly-formed and a rapidly-growing swelling; its outline is ill-defined; its consistence varies with the tension of the parts, and particularly with the proportion of clot; usually it is tense and firm; the skin over it becomes stretched and blue, or stained with extravasated blood. There may be a distinct expansile pulsation in the swelling, but more often the pulsation is very indistinct, or even only a slight vibratory tremor. A bruit is generally to be detected at some part, at any rate, of the swelling, and occasionally there is a thrill. The pulse in the arteries beyond may be lost altogether, or only weaker than on the opposite side. If the main artery of the limb is the one injured, and if the injury divides it nearly or wholly across, the pulse in the vessels below is lost; but if the aneurysm is formed in connection with a branch of the main artery, or from a small wound of the main artery, the pulse beyond is not lost, only weakened, unless the pressure of the aneurysm is sufficient to arrest the arterial circulation altogether. The pressure on the veins causes lividity and coldness of the limb, and œdema, and this and the pressure on the nerves of the part cause a sense of weight and numbness. There is generally intense local pain, and this is sometimes markedly relieved by compressing the main artery above the swelling. The lividity, œdema, and numbness may pass into moist gangrene.

The greater the want of defined outline to the tumour, the more rapidly it grows, and the more marked the effect upon the circulation in the limb below, the greater the warrant for regarding this case as one of **ruptured artery** rather than of diffused aneurysm.

The parts around the aneurysm may become inflamed, the skin being bright red and hot, and the superficial tissue œdematous, or an abscess may give rise to a "pointing" fluctuating swelling.

The **diagnosis** is usually made by recognising the rapid appearance of such a swelling soon after an injury or wound near a large artery, or the sudden enlargement of a smaller firm pulsating swelling under a cicatrix—the diffusion of a circumscribed traumatic aneurysm. But considerable difficulty may be found in distinguishing this form of aneurysm from an acute abscess, and the difficulty becomes increased by the fact that the two conditions may be associated together. The diagnosis will be arrived at by paying careful attention to these points: (1) the exact details of the history of the case, noticing the precise order of events and their relation to the injury; (2) the signs of the swelling communicating with an artery—the pulsation, bruit, thrill; and (3) the interference with the circulation beyond, especially the loss of pulse, but also the œdema and numbness, for the more marked these are the less the probability of the swelling being entirely inflammatory, and the greater the probability that it is aneurysmal.

The circumscribed aneurysms are chiefly met with in the hand and foot, and the scalp; the diffused in the ham and axilla.

Treatment.—(1) A *circumscribed traumatic aneurysm* may undergo cure by the simple treatment of rest, combined with elevation of the limb and gentle compression of the part by a bandage. Where these measures fail the same treatment may be pursued as in the case of spontaneous aneurysm in the same situation. Thus, compression of the artery leading to the tumour may be tried, and if this fails the proximal ligature or the excision of the tumour, with the ligature of the artery above and below, is to be resorted to. When these tumours are small and easily accessible, their excision is the better course to pursue; if deeply placed, and this operation presents any special difficulty, the Hunterian ligature is to be preferred.

Traumatic aneurysm complicating fracture is generally cured by the compression of the splints and bandages applied for the broken bone; if not, the main artery above should be compressed or tied. In these cases the union of the fracture is generally delayed.

(2) *Diffused traumatic aneurysm.*—Where the tumour is enlarging or gangrene threatens, the artery must be tied where wounded without any delay; the internal hæmorrhage being treated exactly as if it were primary external bleeding. This operation may be one of extreme difficulty and danger from hæmorrhage. If the circulation can be controlled by a tourniquet or pressure above, this should be done, and a free incision rapidly made into the swelling, and the blood and coagula quickly turned out. The wounded artery is sought, cleaned, and a ligature is carefully applied above and below the wound. Difficulty is especially met with where the main artery cannot be thus controlled; and if a free incision were made into the tumour the patient would probably die at once from the hæmorrhage that would occur. To prevent this, an incision about one inch and a half is made, and into this two fingers of the left hand are passed and feel for the open mouth of the artery; this is firmly pressed against a bone to prevent any further escape of blood. A probe-pointed bistoury is now passed along the fingers and the wound enlarged sufficiently to lay the entire tumour open. The ends of the artery are then cleaned and tied. Easy as this operation is to describe, in practice it may be attended with the extremest difficulty.

If the tumour is stationary, or extending but slowly, and there are no signs of threatened gangrene, the surgeon may wait in the hope that a sac will form around the clot and then the Hunterian ligature may be applied.

If moist gangrene occurs, amputation at the level of the wounded artery is the only available resource.

Wherever there is a limiting sac, proximal compression and ligature may succeed, but in all other circumstances the double ligature of the artery above and below the wound is the only appropriate treatment.

III. ARTERIO-VENOUS ANEURYSM.

Definition.—An abnormal communication between an artery and a vein constitutes an arterio-venous aneurysm. When the two vessels are adherent together, and the blood passes directly from the artery into the vein, the forcible impact of the blood distends the vein, and the pulsation in this varix makes it resemble an aneurysm, and so it is called an *aneurysmal varix.* But if an aneurysmal sac is developed between the two vessels, opening on the one side into the artery, and on the other into the vein, the condition is known as a *varicose aneurysm.* This difference is a very important one, and is well expressed by these names, the one condition being in essence a varix with only a resemblance to an aneurysm, while the other is, in all important particulars, a sacculated aneurysm with all the dangers and therapeutic indications of aneurysm, to which a peculiar kind and degree of varix is superadded.

While most often traumatic in origin, arterio-venous aneurysm is sometimes idiopathic, and therefore it is dealt with in a separate section of this article.

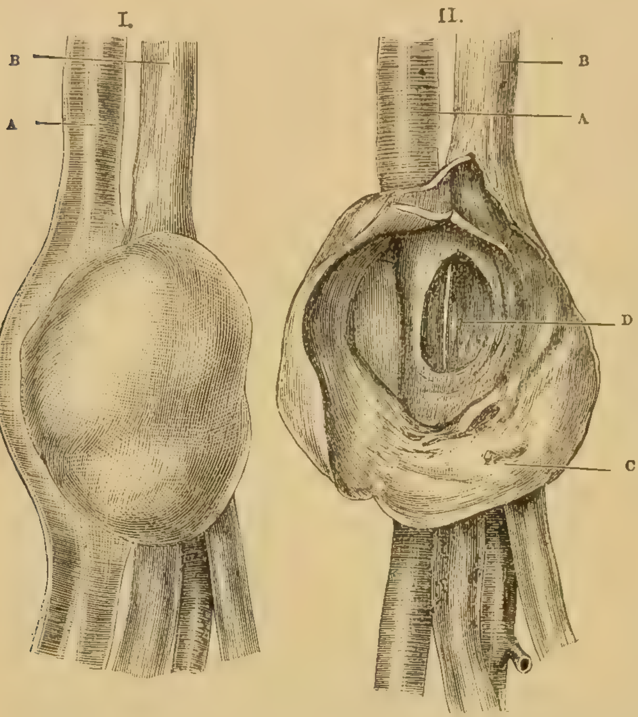

Fig. 182.—Mr. Pemberton's case of Aneurysmal Varix of the Common Femoral Vein. (From *Med. Clin. Soc. Trans.*)
I. The Varix seen from the outside. II. The Varix laid open, showing the rounded opening into the artery.
A, Common femoral artery; B, common femoral vein; C, the varix laid open; D, the communication between artery and vein.

Aneurysmal varix. **Causes.**—A direct communication between an artery and a vein is nearly always traumatic in origin; very occasionally it is idiopathic or congenital. The common cause is a simultaneous punctured wound of an artery and vein, as in careless phlebotomy, stabs, or gunshot wounds. The edges of the adjacent wounds in the vessels adhere, and then when the pressure with which these injuries are treated is removed, the blood passes freely from the artery into the vein. In a stab it may happen that there is a single wound in each vessel from the knife passing, as it were, between them; more commonly, however, it is a lancet which transfixes the vein and punctures the artery beneath it, the superficial vein-wound heals, and the deep one adheres to that in the artery. A remarkable case is on record in which the median-basilic vein, one of the humeral veins, and the brachial artery were simultaneously wounded; the result was that a communication formed between the artery and its companion vein, and another between the two wounded veins, so that blood passed from the artery first into the humeral vein, and then on into the median-basilic vein.

In the days when bleeding was very commonly practised, this condition was much more frequent than it is now. A remarkable instance of the idiopathic origin of aneurysmal varix has been recorded by Pemberton, in which a communication formed between the common femoral artery and vein some time after prolonged compression of the artery for aneurysm (Fig. 182).

Aneurysmal varix, whilst most common at the elbow, has been met with in the skull, the neck, the axilla, the abdomen, and the thigh. A few years ago a patient with aneurysmal varix in Hunter's canal, resulting from a stab wound in the thigh, was in Middlesex Hospital under Mr. Lawson.

Pathological changes.—Whatever the original shape of the aperture between the two vessels, it soon becomes rounded, smooth, and thickened. The *artery* for some distance above is dilated and its walls are thinned; below the communication it is sometimes narrowed, sometimes dilated; the dilatation is said not to affect the branches of the artery in any case. The *vein* opposite the communication is dilated into a globular or fusiform pouch with thickened walls. The venous dilatation—varix—extends along the vein in both directions and into its branches; below the communication the enlarged veins are tortuous and pouched, but above it they are simply enlarged. The interior of the vein may show atheromatous changes.

The pressure in the artery being much greater than in the vein, arterial blood is forced *per saltum* into the vein and this causes three effects: (1) The vein opposite the aperture becomes dilated and thickened; (2) the increased quantity of blood returned to the heart from the vein causes the enlargement of the vein on the cardiac side; (3) the return of blood entering the vein below the aperture is obstructed by the forcible flow passing from the artery, and this leads to the dilatation of the veins below the communication.

Signs and diagnosis.—The varix forms a soft, compressible, often *ill-defined tumour* in the course of a vein, into which dilated veins can be traced, both from above and below. This varix is the seat of a forcible expansile *pulsation*, a continuous very marked vibratory *thrill*, and a very loud *bruit*. The bruit is a continuous rasping, purring, hissing, or rushing sound, sometimes compared to the noise of distant machinery in motion, or to the sound of a fly in a paper box. It is usually of great intensity and may be audible even to the patient and to bystanders. The pulsation, bruit, and thrill are all conducted for some distance along the distended veins, but with gradually diminishing intensity, and the bruit and thrill lose their continuous character at a little distance from the abnormal communication, and become interrupted, and synchronous with the pulse. If the limb be raised the *tumour shrinks*, and the pulsation becomes less marked; if the artery leading to it is compressed, the tumour not only loses its pulsation, but shrinks and disappears. When the limb is depressed the *tumour becomes very full*, and the pulsation in it is more marked. The dilated condition of the artery above the varix is sometimes quite apparent. The *condition of the limb* below varies in different cases, probably with the freedom of the communication with the artery, and the extent to which the circulation is interfered with. It may be cold and œdematous, with a feeble pulse, or it may be hotter and hypertrophied; obstinate ulcers with a tendency to bleed sometimes occur. The patient may suffer *acute pain* in the tumour, or experience only a sense of numbness and weakness in the limb below. It is often noticed that the symptoms increase up to a certain point, and then remain stationary. A scar over the swelling generally points to the nature of the case.

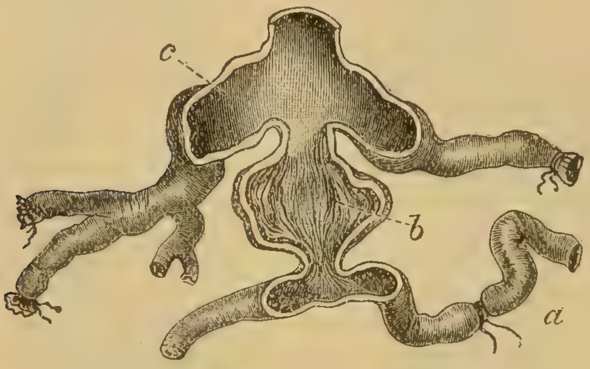

Fig. 183.—Varicose Aneurysm at Elbow. (After Erichsen.) *a*, Ulnar artery; *b*, aneurysm partly filled with clot; *c*, dilatation of median basilic vein.

Treatment.—In many cases all that is required is to apply some form of external support to the part—a bandage or an elastic stocking—for the condition remains stationary and causes very trifling inconvenience. If the varix steadily increases, or the patient suffers serious inconvenience from the pain or the effects of the disturbance in the circulation, the artery should be ligatured above and below its communication with the vein.

In a case of aneurysmal varix in Hunter's canal Mr. Lawson tied the artery above and below the communication with the vein, and the patient made a rapid and complete recovery. In the days when this affection was more common such an operation was fraught with dangers from which it can now be guarded, and therefore it was rarely resorted to. Now that the dangers can be avoided the operation is more readily and more often recommended.

Varicose aneurysm. **Causes.**—The most frequent cause of an aneurysm with a communication with a vein (Fig. 183) is the simultaneous injury of an artery and vein—usually a stab, puncture, gunshot, or phlebotomy wound. In place of the wounds in the two vessels being sealed up, or cohering together, a circumscribed traumatic aneurysm develops between them, which communicates with both. An aneurysmal varix may slowly develop into a varicose aneurysm if the tissue uniting the two vessels yields. But such an aneurysm may develop spontaneously by an ordinary idiopathic aneurysm first compressing and then opening into a neighbouring vein. This has been known to occur in the thorax, abdomen, and groin. (*See* Fig. 184.)

Pathology.—The only difference between this condition and a circumscribed traumatic aneurysm arises from the fact that the sac has an additional opening or openings into it from a vein or veins. The relative position of the arterial and venous openings into the sac varies much. A case is recorded in which two veins communicated with the sac of an aneurysm, so that on laying it open four venous openings were detected. A still more interesting case is one in which the deep wound in a transfixed vein cohered with that in an artery; but the superficial wound in the vein did not heal up, and the blood escaping from it formed an aneurysmal tumour. The blood flowed from the artery into the vein, and then on into the sac of the aneurysm.

The veins of the part undergo the same changes as are seen in aneurysmal varix. Traumatic varicose aneurysms are usually of small size, and their sacs may contain very little, if any, fibrin, owing to the direct passage of blood through them. The sac may steadily enlarge and rupture externally or subcutaneously. Spontaneous cure is very rare. Occasionally clot forms in the aneurysm, so as to shut off the communication with the vein, and the case is reduced then to one of simple traumatic aneurysm. In internal aneurysms—as of the aorta—the communication with a vein or the pulmonary artery may greatly add to the gravity of the case; but in external aneurysm the communication with a vein gives rise to very characteristic symptoms, but does not influence the course of the disease to any important extent.

Signs and diagnosis.—The signs of the communication between artery and vein are the same as in aneurysmal varix—distension and pulsation of the veins, with a very intense bruit and thrill conducted for some distance along the dilated veins. In addition, there are the signs of an aneurysm. Thus it is sometimes possible to distinguish two tumours, or two parts of one tumour—one, the dilated vein, soft and compressible, and the other, the aneurysm, firmer.

The diagnosis is rendered clearer if, when the artery above is compressed, the dilated vein collapses, leaving the aneurysm plainly felt as a firm tumour. In addition to the continuous loud bruit, a soft-blowing systolic aneurysmal bruit can sometimes be distinguished; and it may be discovered, on careful examination, that pressure on a certain spot stops the loud rasping bruit and the thrill, without arresting the pulsation in the aneurysm or the true aneurysmal bruit. All these special signs point to the existence of an aneurysm, in addition to the arterio-venous communication. In other cases the existence of an aneurysm is known, and the fact that it has formed a communication with a vein is shown by the marked venous dilatation, not merely below the tumour—which might be caused by pressure only—but over and above it, by the very marked thrill and pulsation in the veins, and by the peculiar character and intensity of the bruit that is developed.

Treatment.—Varicose aneurysm should, whenever possible, be submitted to active treatment for the cure of the aneurysm. To leave it alone, or to be content with merely protecting the part by a supporting bandage, is to expose the patient to all the dangers of an untreated aneurysm. The most successful form of compression has been digital pressure of the artery on the proximal side, combined with local pressure over the communication between the aneurysm and the vein. Wherever it can be carried out, this plan should be tried. If it fails, or if the case is urgent from rapid extension of the aneurysm, the sac should be excised and all the vessels communicating with it carefully tied. To do this, the limb should be rendered bloodless by Esmarch's bandage and a tourniquet. The dilated vein is first exposed and a double ligature placed upon it. The sac of the aneurysm is then carefully cleared and traced down to the artery with which it communicates; a double ligature is placed on this vessel and the sac removed. The Hunterian operation has succeeded in these cases, but the "old" operation is to be preferred wherever it can be carried out satisfactorily.

ANEURYSMS OF PARTICULAR ARTERIES.

Having considered the pathological and clinical features of aneurysms in general, without regard to locality, we must now study them from a local point of view, and try to notice the special features of aneurysms as they occur in connection with particular arteries.

Aneurysm of the thoracic aorta.—The aorta is the most frequent seat of aneurysm, and the disease is met with in every part of the vessel, but most often in the arch, particularly on the right side of the ascending portion, and least often in the abdominal aorta. All the forms of spontaneous aneurysm are met with in the aorta. Fusiform aneurysm is very common, and the enlarged artery may reach a great size. Sacculated aneurysm occurs without any dilatation of the entire circumference of the artery, but more often as a pouch or sac projecting from a fusiform aneurysm; and it is these cases that attain the greatest size, and also, as a rule, give rise to the most marked symptoms. A sacculated aneurysm close above the valves is a very dangerous form, owing to its great liability to rupture into the pericardium or heart. Dissecting aneurysm occurs more often in the aorta than elsewhere, but is so rare as to be a pathological curiosity.

The signs of aortic aneurysm vary considerably, for the tumour may involve any part of the vessel, and grow in any direction, and the symptoms and whole course of the disease largely depend upon this factor. The whole artery is so deeply placed that an aneurysm of it may form no obvious tumour, and although the vessel is in relation with many most important parts, it may be so placed as to avoid injurious pressure on any of them. It therefore happens that an aortic aneurysm may be present without giving rise to any symptoms by which a diagnosis can be arrived at, and may even run its course to fatal rupture without its presence having been suspected.

The *tumour* reveals its presence by its pressure effects in the chest, by an abnormal area of dulness to percussion, and by localised swelling. The swelling is found most often on the right side of the sternum, at first displacing forwards the structures of the chest wall, but often absorbing the bone, and forming a tumour superficial to the sternum. In connection with the transverse part of the arch the tumour may rise up into the neck at the suprasternal notch. Aneurysm of the descending aorta projects—if at all—posteriorly in the thoracic region, on the left of the spine. *Pulsation* may be felt over an aneurysm in the chest before any bulging of the wall has occurred, and is a very important sign. *Bruits* of various kinds are heard over aortic aneurysms. They may be systolic, diastolic, or both, soft or harsh, blowing, musical or rasping, and there may be an entire absence of all bruit. In some cases the heart-sounds are heard with marked intensity all over the aneurysm; and in the absence of the more definite signs, this may be an important sign of aneurysmal dilatation of the first part of the arch. In aneurysms of the descending aorta the bruits are heard at the back, especially on the left side of the spine. In some cases a marked *thrill* is present in the tumour. *The pulse* may afford valuable indications, or none at all. It may be lost in certain arteries—as either carotid or subclavian—from the aneurysm compressing the origin of these vessels. In other cases there is a marked difference in the fulness and force of the pulse in the arteries on the proximal and distal side of the aneurysm, and the sphygmograph may make this effect very plain.

The *pressure effects* of aortic aneurysm vary with the position, size, and rapidity of growth of the tumour. The most constant is *pain*, which may be of at least three kinds: a sense of weight or constriction, or a dull aching pain at the seat of the aneurysm; a deep boring or burning pain when the aneurysm is pressing upon bone and causing its absorption; and intercostal neuralgia, a lancinating pain referred to the area of distribution of compressed spinal nerves. *Displacement of the heart* is a very common effect of a thoracic aneurysm: usually the heart is pushed downwards, and to the left; but an aneurysm of the descending aorta may push it forwards. *Pressure on the pulmonary artery* may lead to serious dyspnœa and cyanosis from imperfect aëration of the blood, and to dropsy and the other results of obstruction to the emptying of the right heart. *Pressure on the great systemic veins*—the vena cava or either innominate vein—causes cyanosis, venous distension and œdema of the areas drained by them. *Pressure on the pulmonary veins* causes general cyanosis, dyspnœa and the signs of pulmonary congestion. The *trachea*, either *bronchus* or the *lung*, may be compressed and cause grave dyspnœa and interference with the function of one or both lungs. Pressure on the *œsophagus* causes dysphagia, more or less complete, and compression of the *thoracic duct* leads to rapid emaciation and chylous ascites. Pressure upon the left *inferior laryngeal nerve* as it courses round the aorta quickly causes abductor paralysis, the vocal cord is drawn into the position of phonation by the unopposed adductors and the voice is unaffected. With increased pressure the adductor muscles are paralysed, the cord is motionless in the cadaveric position, and the voice is hoarse. Pressure on the *vagus nerve* may cause reflex spasm of the opposite vocal cord, grave disturbance of the heart's action, or even its arrest in diastole, and dyspnœa. Pressure upon the phrenic nerve paralyses the diaphragm on the same side. An aneurysm eroding the spine and compressing the *spinal cord* or *nerves* causes pain, anæsthesia, and paralysis. In the case of aneurysm of the transverse part of the arch, if the patient throws his head back and the surgeon seizes his larynx and gently draws it up, he will be conscious of a sensation of pulling downwards of the trachea with each beat of the heart. This has been called the *tracheal tug:* it indicates that the body pressing upon the trachea in the chest is moved with each beat of the heart.

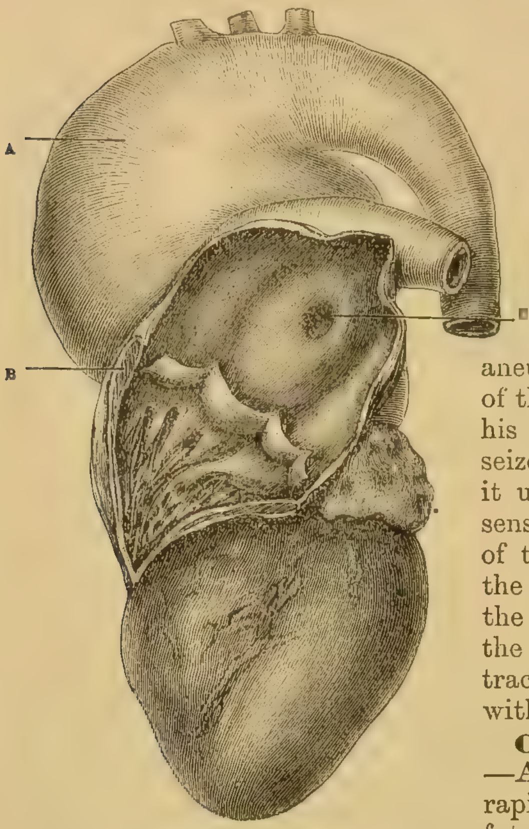

Fig. 184.—Aneurysm of the Arch of the Aorta communicating with the Pulmonary Artery. (From a specimen in the Brompton Hospital Museum.)

A, Aneurysm; B, pulmonary artery laid open; C, communication between aneurysm and pulmonary artery.

Course and terminations.—Aortic aneurysm may run a rapid course and be quickly fatal, or its course may be slow and insidious and extend over years. The progress depends first upon the nature of the aneurysm—sacculated being much more rapid and dangerous than fusiform; then upon its position and direction of growth—aneurysms within the pericardium and those on the concavity of the arch are the most serious; upon the size and disposition of the mouth of the aneurysm, the force of the heart, and upon the habits, occupation, and temperament of the patient. Patients have been known to live for some years with aneurysms projecting through their chest walls.

An aortic aneurysm never becomes truly cured: but great and lasting improvement may take place either spontaneously or as the result of treatment, from the deposit of clot within the sac and its partial organisation; but as the artery from which the aneurysm springs does not become obliterated, such an aneurysm, however solid, is not really cured, and subsequent growth may and often does occur. Aortic aneurysm causes death in many ways. The sac may rupture into the pericardium, heart, pleura, mediastinum, trachea, bronchus, œsophagus, spinal canal, or externally. Fatal syncope may occur from obstruction to a coronary artery, or from the weight of blood above the aortic valves. Death may be due to interference with the circulation by pressure upon or communication with the pulmonary artery (Fig. 184), or pressure on the pulmonary or systemic veins; or to the respiratory difficulty caused by pressure on the trachea, bronchus or lung, laryngeal paralysis, bronchitis, and pneumonia. Cerebral embolism is yet another cause of death. In many cases aortic aneurysm is complicated with valvular disease of the heart, and this is another factor in the fatal termination of such cases. Great dilatation of the ascending portion of the aortic arch often leads to incompetence of the aortic valve.

Treatment.—The treatment of aortic aneurysm is palliative only: it cannot, so far as our knowledge goes at present, be curative; but yet it may be so far successful that the patients may be able to resume active work for a few years. The constitutional treatment for aneurysm described on page 609 should be carried out with the utmost care and attention to detail. Absolute rest should be enjoined, the heart's action, if excited, should be calmed, and a carefully-regulated diet prescribed. Large doses of iodide of potassium may be useful and, in cases of syphilis, should always be given. Under this treatment, continued for some time and then most cautiously relaxed, great improvement may be obtained; but as the aneurysm in every case still exists, such patients should carefully avoid effort, strain, excitement, and all conditions tending to raise the arterial tension.

Of surgical measures, electrolysis and acupuncture have been the most successful. They can only be employed when the aneurysm is pressing against or bulging through the surface, and it is in sacculated aneurysms with small orifices and without large branches opening out of the sac that the best results have been obtained. In employing electrolysis it is best to use steel needles connected with the positive pole only, a large negative electrode being placed on the back. A current gradually increased up to 40 or 50 milliampères is to be employed for 20 to 30 minutes, and repeated if necessary at intervals of seven to ten days. If acupuncture is employed Macewen's method should be followed, the number of needles used and the frequency with which they are introduced varying with the nature of the case. By either of these plans a clot may be formed which may arrest the growth of the aneurysm and avert immediate danger and relieve pain. But the clot may become organised and greatly strengthen the sac, and in some very favourable cases a pouch of the aneurysm may in this way be obliterated.

The introduction of wire into the sac has also been practised, but not with such success as to justify its use. It has also been suggested that after introducing a coil of wire into the aneurysm, it should be connected with the positive pole of a battery; in this way the effect of the wire would be supplemented by the coagulating effect of the electricity and of the chloride of iron it produces.

The distal ligature has also been practised, and with occasional success: thus one carotid artery—particularly the left—or the carotid and subclavian artery, have been tied. These operations are themselves attended with considerable risk; they have sometimes been followed by aggravation of the aneurysm, and there is neither any satisfactory explanation of the rationale of the successful cases, nor guide as to when the operation has a prospect of success. For these reasons the distal ligature has not won the general acceptance of surgeons.

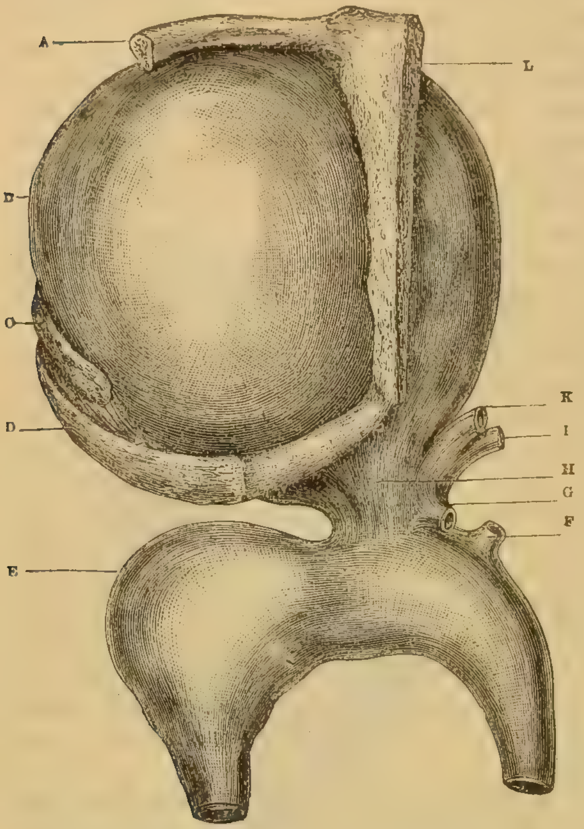

Fig. 185.—Large Aneurysm of the Innominate Artery, which grew high up in the Neck and caused great Displacement of the Sternum, Clavicle and First Rib. The arch of the aorta is the seat of two fusiform dilatations. (Museum, Middlesex Hospital.)

A, Clavicle; B, aneurysm; C, first rib; D, second rib; E, arch of aorta; F, left subclavian artery; G, right subclavian artery; H, innominate artery; I, left carotid artery; K, right carotid artery; L, sternum.

Abdominal aneurysm.—Aneurysm may occur in any part of the abdominal aorta, but it has been most often met with at its bifurcation, or near the origin of the cœliac axis. The mesenteric, hepatic, and splenic arteries may also be the seat of aneurysm. These aneurysms, like others, may remain small, or become filled with coagulum; but they may attain an enormous size, and may rupture into the peritoneum, subperitoneal tissue, stomach, intestine, or pleura.

Symptoms.—These aneurysms may cause local discomfort merely, but if they erode the spine, the pain is severe—an aching, boring, or burning pain in the loins—while pressure on the lumbar plexus may cause pain in the scrotum or front of the thigh. A strong subjective sense of epigastric pulsation may be present;

pressure upon the bowel may cause signs of obstruction; jaundice, vomiting, and albuminuria are other symptoms that may be observed; serious compression of the inferior vena cava very rarely occurs.

The **diagnosis** may be a matter of considerable difficulty and the complete relaxation of the abdominal muscles by means of an anæsthetic is a great help in arriving at it. *A pulsating aorta* is often observed in anæmic women, who at the same time suffer from dyspepsia. This condition is distinguished from aneurysm by noticing the absence of all tumour, that the pulsation can be traced along the line of the artery, that there is no aneurysmal bruit, and that when the patient lies on her hands and knees, with the abdominal walls relaxed, the hand laid gently over the part does not detect any pulsation. Fæcal accumulations over the aorta, and malignant growths of the pylorus, pancreas, or spine are other conditions that may simulate aneurysm.

Treatment.—In all cases a prolonged and careful trial of constitutional measures only should be made; this alone may be successful. If it fails, and the aneurysm is so placed that the pad of a tourniquet can be insinuated above it and over the aorta, proximal compression of this artery continued for several hours, and with the patient under the influence of an anæsthetic, should be tried; this treatment has been fairly successful. Distal compression of the abdominal aorta has not been practised with success.

Aneurysm of the innominate artery.—Innominate aneurysm is always spontaneous; it may be either fusiform or sacculated, and may affect any part or the whole of the artery; aneurysm of the origin of the artery is always associated with dilatation of the arch of the aorta.

Symptoms.—The tumour may not project at all and merely give rise to dulness over the sternum and beneath the inner end of the clavicle; projection of the aneurysm is usually first observed behind the sternal origin of the sterno-mastoid muscle, and as the tumour grows it displaces this muscle, or the sternum and clavicle, or the trachea. As the tumour increases in size it may grow upwards into the neck over the carotid artery, where it has been known to reach as high as the cricoid cartilage, or backwards and inwards towards the trachea and spine, or outwards along the subclavian artery (Fig. 185). Pouches of the sac may extend in several or all of these directions, but growth outwards is most common. On auscultation over the tumour no bruit may be heard, but the heart's sounds may be very plainly audible.

The special symptoms of the aneurysm are alteration of the pulse in the radial and branches of the external carotid artery on the right side, dyspnœa, stridor, and alteration of the voice, dysphagia, cyanosis, and œdema of the right hand and arm, and of the right side of the face, head, and neck; the œdema may extend to the left side of the face also. The patient often experiences pain down the right arm, and weakness in it, from pressure upon the brachial plexus, and shooting pains in the head and neck from irritation of the superficial cervical plexus. Compression of the sympathetic trunk may lead to dilatation of the arteries of the same side of the head and face, with abundant sweating, and contracted pupil. Spontaneous cure has been noted in one case only; the disease generally proves fatal, death being due to asphyxia from pressure, or to rupture of the aneurysm, either externally or into the trachea, œsophagus, or lung.

Treatment.—Careful constitutional treatment should first be well tried, and digital compression of the right common carotid artery may be combined with it. If these means fail, simultaneous ligature of the right common carotid and subclavian arteries should be practised. It does not suffice to tie one of the trunks, for the free current of blood still flowing through the parent vessel prevents cure of the aneurysm; even when both are tied, the flow of blood to the branches of the first part of the subclavian artery may be sufficient to prevent occlusion of the artery and cure of the aneurysm.

Should the size of the aneurysm prevent the ligature of one or both of these arteries, or the operation fail to cure, electrolysis and acupuncture are the only other means at the disposal of the surgeon.

Diagnosis of aneurysms at the root of the neck.—A tumour at the root of the neck having been recognised as an aneurysm, it is often a matter of great difficulty to determine exactly what artery, or part of artery, it is connected with. Attention to the following points, will, however, generally lead to a correct diagnosis.

(1) **Apparent origin.**—An *aortic aneurysm* is often detected within the chest before it appears at the root of the neck, and generally extends farther into the mediastinum than an innominate aneurysm; its first appearance in the neck may exactly resemble that of an innominate or carotid aneurysm; an aneurysm of the second part of the arch may project immediately above the episternal notch. An *innominate aneurysm* appears first behind and above the inner end of the right clavicle, and fills up the hollow between the two heads of the sterno-mastoid muscle. A *carotid aneurysm* bulges forwards beneath the inner head of the sterno-mastoid. A *subclavian aneurysm* appears first in the posterior triangle, or beneath the outer head of the sterno-mastoid muscle.

(2) **Direction of growth.**—An *aortic aneurysm* may extend in any direction, especially within the chest; the detection of a considerable tumour within the thorax renders the diagnosis clear. An *innominate aneurysm* may be of a globular shape and project forwards, or it may extend along either of its branches. A *carotid aneurysm* grows up the neck along the sterno-mastoid, while a *subclavian aneurysm* usually extends horizontally along the upper border of the clavicle.

(3) **Limits.**—No lower limit can ever be felt to an *aortic* or *innominate* aneurysm, but the finger can often be dipped down below a *carotid* aneurysm, or to the inner side of a *subclavian* tumour.

(4) **Pressure effects.**—The pressure signs are very similar in

the different cases. Pressure on the left recurrent laryngeal nerve would distinguish an aortic aneurysm from one situated on the right vessels ; pressure on the right nerve in a like manner excludes an aortic aneurysm. Pressure on the left innominate vein indicates aortic aneurysm rather than innominate ; compression of the internal jugular or subclavian vein only, points to carotid or subclavian aneurysm. A " tracheal tug " indicates an aneurysm of the aorta.

(5) The **pulse** in the branches of the great arteries is, perhaps, the most useful sign of all. Where the pulse in the two radial arteries is exactly similar, it shows that the aneurysm is aortic, either on the proximal side of the innominate artery or the distal side of the left subclavian : the sphygmograph may show the dicrotic wave and percussion stroke lost. If, however, the left radial pulse is " aneurysmal " and the right is normal, it points to an aneurysm either of the transverse part of the arch beyond the origin of the innominate trunk, or of the left subclavian artery, and the condition of the pulse in the branches of the left carotid artery will determine this point. When the right radial and carotid pulses are aneurysmal, and the left are normal, it indicates an innominate aneurysm ; where, of these, only one is aneurysmal, it shows an aneurysm of the corresponding branch. It must be carefully remembered that the pulse may be lost in an artery by the sac of an aneurysm of a neighbouring trunk compressing it, or occluding it by displacement or by a plug of clot. The special aneurysmal pulse, and not the loss of pulsation, is the guide to diagnosis.

Aneurysms of the carotid arteries.—Aneurysm of the *common carotid artery* is nearly always "spontaneous" in origin, for wounds of the artery are generally fatal—those that are recovered from are frequently associated with wound of the jugular vein. It occurs more commonly in women than any other external aneurysm. The favourite seat of the aneurysm is at the bifurcation of the trunk and the next most frequent spot is at the origin of the right artery : the thoracic portion of the left artery appears to be exempt.

Symptoms.—Commencing as a small tumour, the aneurysm may grow very slowly, and remain unaltered for years, or it may more quickly attain a large size and fill up the neck from the chin to the sternum. In addition to the usual signs of aneurysm, there may be great dyspnœa with frequent spasmodic cough, which may end in asphyxia, hoarseness and loss of voice, dysphagia, *tinnitus aurum*, dimness or loss of sight, contracted pupil, giddiness, stupor, hyperæsthesia of the scalp, and a sense of pulsation referred to the whole of the same side of the head. These symptoms are the result of pressure upon the larynx, trachea, pharynx, œsophagus, recurrent larnygeal and sympathetic nerves, or of interference with the circulation in the brain. Spontaneous cure has been known to occur; more often the aneurysm ruptures externally or into the trachea, larynx, or œsophagus, with rapidly fatal hæmorrhage. Cerebral embolism is another danger.

The **diagnosis** of carotid aneurysm often presents great difficulties, and many mistakes have been made. *Tumours of the thyroid gland* are distinguished by rising and falling with the trachea during deglutition, by their fixedness on the inner side, often by the implication of the isthmus, and by their mobility over the carotid artery. *Lymphatic glandular tumours* are globular, ovoid, or lobulated in outline, often multiple, and generally incompressible and freely movable away from the artery. When the artery passes through the middle of the tumour it may be somewhat compressible, and it is not movable over the vessel; in such a case the outline of the swelling, its history, the presence of other enlarged glands, and of some obvious cause for the swelling—one or all of these--clear up the diagnosis.

A *cyst* under the sterno-mastoid muscle may pulsate from its close proximity to the artery, and if the cyst is flaccid and folded round the artery, there may be slight expansile pulsation in it. The history of the case, the irreducibility of the tumour, the fact that compression of the carotid artery below the tumour does not affect its size or tension, distinguish it from aneurysm. In some cases when the sterno-mastoid muscle is relaxed, the cyst can be lifted away from the artery and then its pulsation is lost.

Abscess over the artery is distinguished by the usual signs of inflammation, by obvious fluctuation, and by the irreducibility of the swelling when the artery below is compressed; the special features of the aneurysmal pulsation and bruit are also absent. When the artery has opened into an abscess cavity, the diagnosis is more difficult; the signs of inflammation and the absence of a clearly defined outline of the swelling, as well as of many of the other leading signs of aneurysm, are the chief points to be noted. *Varix* of the internal jugular vein is a soft compressible swelling, which shrinks during deep inspiration and becomes fuller when expiratory efforts are made; the pulsation in it is transmitted and is not truly expansile. *Other solid tumours*, fatty or sarcomatous, must be distinguished by their outline, mode of growth, mobility upon the artery, fixity in some cases to other structures, and by their incompressibility and the absence of the other signs of aneurysm.

Treatment.—Whenever practicable, digital compression below the aneurysm should be tried, as it has been attended with a good deal of success; if possible, the artery should be controlled by pressure against the transverse process of the sixth cervical vertebra without pressure upon the vagus nerve. Ligature of the artery, either below or above the aneurysm, is the other chief means of cure; the proximal ligature is to be preferred in all cases where there is room to apply the thread between the tumour and the sternum. This operation is attended with risk. Its chief danger is cerebral anæmia followed by white softening, or by the passive congestion which may follow the ligature of an artery in any situation. An abscess may develop in the anæmic area. Immediately on tightening the ligature there may be syncope, dimness of sight, tinnitus, and giddiness; and if softening occurs, hemiplegia ensues, and there may be convulsions, and death often results.

Pulmonary congestion is another frequent complication and it is liable to run on to hypostatic pneumonia. The interference with the circulation in the brain and medulla caused by the operation is the probable explanation of this occurrence. It is noteworthy that the effects of sudden closure of the carotid artery are much more grave than when the obstruction is gradual. A patient has even lived for some time after gradual obstruction of both carotid and both vertebral arteries. Simultaneous ligature of both carotids is always fatal from cerebral anæmia and coma, but if an interval of two or three weeks is left between the ligature of the two vessels, the danger of cerebral disease is not greater than if one only is tied.

Suppuration around the sac of the aneurysm may follow ligature of the artery, either from the sudden solidification of the tumour, or, more probably, from wound infection. The pus should be evacuated by an early free incision, and if hæmorrhage occurs the sac must be laid open and the ends of the artery tied. Manipulation or the introduction of foreign bodies should never be practised in carotid aneurysm on account of the great danger of cerebral embolism.

Aneurysm of the internal carotid artery in the neck is rare; it has a great tendency to bulge into the pharynx and even to simulate an abscess in the tonsil. It should be treated by digital compression or ligature of the common carotid artery.

Aneurysm of the external carotid artery may, by pressure upon the hypoglossal nerve, cause paralysis of the same side of the tongue. If digital compression of the common carotid artery is not successful, a ligature may be placed upon the external carotid artery below the aneurysm, and only if the tumour arises too low for this to be possible should the common carotid artery be tied. Recurrent pulsation is very apt to occur, owing to the free anastomoses of the many branches arising close together from the artery. For this, if rest and careful direct pressure fail, the branches communicating with the aneurysm should be tied. If the tumour is small, or of moderate size, its excision may be undertaken at first, instead of the proximal ligature.

Aneurysmal varix in the neck is usually the result of sabre cuts and stabs, in which the weapon has passed through a vein into an artery. A case is recorded, however, in which a communication between the common carotid artery and internal jugular vein arose spontaneously. The vessels involved may be, in order of frequency: (1) The common carotid artery and the internal jugular vein; (2) the internal carotid artery and the internal jugular vein; (3) the common carotid artery and the subclavian vein; and (4) the external carotid artery and the internal jugular vein.

Immediately after the injury there is a great effusion of blood in the cellular tissue of the whole side of the neck, and this may be so great as to threaten asphyxia; when this is absorbed, the characteristic symptoms of the aneurysmal varix manifest themselves. The patients are liable to headache, giddiness, and other signs of cerebral congestion, and also to palpitation, which it is believed may arise from the presence of arterial blood in the right side of the heart. The bruit may be so loud as even to interfere with the patient's sleep. Only palliative treatment has been adopted for these cases; when the common or internal carotid artery is implicated, operation is not, as a rule, indicated; if the symptoms are very marked and annoying, a ligature may be placed on the artery above and below the communication with the vein; but if in any case the surgeon can satisfy himself that the artery involved is the external carotid, this artery should be tied above and below the communication with the vein. The cerebral congestion, when severe, may be relieved by leeches.

A case of *varicose aneurysm of the internal carotid artery* close to the skull has been recorded; the appropriate treatment for such a condition is ligature of the artery lower down in the neck.

Traumatic aneurysm of the temporal artery may result from accidental wounds, or after arteriotomy. The best treatment is excision of the aneurysm.

Intracranial aneurysm.—The miliary aneurysms on the arteries in the substance of the brain, which are always to be found in cases of cerebral hæmorrhage, are met with almost exclusively in the latter half of life, and may be regarded as the result of arterial degeneration. The aneurysms which are met with in the arteries at the base of the brain are less frequent, but to the surgeon more important, and the following remarks apply exclusively to them.

These aneurysms are nearly equally frequent in each decade of life after the first to the seventh, and after that become very rare. They appear to be due most often to syphilis or embolism, rarely to injury or atheroma. Syphilis is known to affect the arteries of the brain more often than other arteries; the association of intracranial aneurysm with valvular disease of the heart is also very marked. The arteries most often affected are the middle cerebral, the basilar, and the internal carotid. The aneurysms are generally the size of a pea, but may be as large as a hazel nut, a grape, or even a hen's egg. Both fusiform and sacculated aneurysms are met with; a variable amount of clot may be found in the sac, and spontaneous cure has been known to occur. Rupture of the aneurysm is the most frequent termination, and the resulting hæmorrhage into the membranes, brain substance, or cerebral ventricles, causes sudden or speedy death; occasionally the rupture is small and the surrounding parts resistant, and the hæmorrhage is slow and its effects are gradually produced.

Symptoms.—In many cases no symptoms have been observed prior to those of the fatal rupture. The most constant symptoms are headache and giddiness; paralysis may be induced by pressure upon the nerves at the base of the brain, or the brain itself, and by carefully noting the paralysis the exact position of the tumour may be determined; optic neuritis has been especially observed in connection with aneurysm of the internal carotid artery. In some cases a bruit is audible to the patient, or to the observer, and the place of greatest intensity, and the effects upon the bruit, of compression of the carotid artery in the neck may enable the surgeon to determine on which series of vessels the aneurysm is placed; aneurysm of the basilar artery has been known to give a bruit audible just behind the mastoid process; the bruit of an aneurysm of the internal carotid is arrested by compression of the vessel in the neck. In some cases of spontaneous aneurysm the patient gives a history of a sudden pain, or a sense as of something bursting in his head at the outset of the disease.

Treatment.—If the aneurysm be diagnosed, and cannot be exactly localised, the only treatment that can be pursued is constitutional—the avoidance of all causes of increased arterial tension, and, especially in cases of syphilis, large doses of iodide of potassium. If the aneurysm be known to be on the internal carotid or middle cerebral artery, the internal carotid artery should be ligatured in the neck. For an aneurysm of the basilar artery, one or both vertebral arteries might be tied.

Intra-orbital aneurysm.—*Pulsating exophthalmos* is probably a better name for the series of cases we are now to consider, for they have been grouped together on account of their clinical resemblance, and not their pathological identity.

The **symptoms** of these cases are more or less marked proptosis, with swelling of the eyelids, congestion and œdema of the conjunctiva, and a well-marked pulsation in the orbit. Often there is a distinct thrill to be felt, particularly near the inner canthus, and on listening over the brow or temple a loud musical or whizzing bruit is audible; the bruit is heard also by the patient. There may be ocular paralysis, dilated pupil, steamy cornea, and opacity of the media, causing almost entire blindness; if the fundus can be seen, the retinal veins are noticed to be full and often pulsating, and the outline of the disc is blurred.

Pathology.—The conditions that have been shown by careful dissection to give rise to these symptoms are varied. They include aneurysm of the ophthalmic artery within or behind the orbit, rupture of a carotid aneurysm into the cavernous sinus, aneurysmal varix—the carotid artery communicating with the cavernous sinus—dilatation of the carotid artery in the petrous bone, thrombosis of the cavernous sinus, and compression of the ophthalmic vein by inflammatory products. It is believed that cirsoid aneurysm may occur in the orbit as a congenital affection. Many of the cases are traumatic in origin, arising from severe injuries to the head causing fracture about the cavernous sinus, or from punctured wounds of the orbit. In the idiopathic cases a sudden sense of something bursting or giving way behind the eyeball may usher in the symptoms.

Course and diagnosis.—The rapidity with which the symptoms develop varies in different cases ; in some they quickly run on to pronounced exophthalmos and blindness, in others the course is slow, and the symptoms may remain stationary for years, or even abate spontaneously.

The pulsation, the thrill, the loud bruit, and the effects of compression of the common carotid artery distinguish this condition from a pulsating sarcoma in the orbit, the only other liable to be confounded with it. An exact diagnosis of the cause of a given case of "orbital aneurysm" is often difficult and may be impossible. If congenital in origin it may safely be regarded as a cirsoid aneurysm ; if traumatic in origin the cause is most probably a rent of the carotid artery, where it lies in the cavernous sinus—aneurysmal varix. In the idiopathic cases attention should be paid to the history and to any concomitant affection. A history of a sensation as of a sudden snap behind the eye before any proptosis or pulsation was noticed would justify the diagnosis of aneurysm. The existence of suppuration in the ear, nose, face, or about the base of the skull would support the diagnosis of thrombosis, and if the affection spreads to the other orbit, is attended with fever of a remittent type, or if rigors and secondary abscesses occur, this diagnosis is established. Cardiac disease or general arterial degeneration would suggest aneurysm.

Treatment.—Prolonged rest in bed with avoidance of all cardiac stimulants in many cases leads to a gradual or more abrupt cure, and should always be carefully tried before resorting to other measures. In addition, an ice-bag may be applied externally and iodide of potassium given internally. If, in spite of this treatment, the symptoms persist, the internal carotid artery should be tied in the neck, if there is reason to believe that the condition is an aneurysm or an aneurysmal varix ; in such cases this operation has been very successful. For cirsoid aneurysm electrolysis may be used before, or after, resorting to ligature of the carotid. Electrolysis has been successfully adopted in some other cases. For infective thrombosis of the sinus no surgical measures are of any avail.

Aneurysm of the subclavian artery.—Subclavian aneurysm is much more common in men than women, and on the right than the left side, and this is believed to be due to the influence upon the artery of free and forcible movement of the shoulder and of injuries to the part. Although strain and over-use of the right arm play an important part in causing spontaneous aneurysm, true traumatic aneurysm from wound of the artery has not been met with, partly because the vessel is so well protected from direct injury, and partly because the hæmorrhage from a wounded subclavian artery is quickly fatal. There are, however, at least two recorded cases of *aneurysmal varix* from stabs.

Symptoms.—An aneurysm may arise from any part of the artery, except the first part on the left side, but is most common in the third part of the artery. It usually forms a small tumour about the size of a hen's egg in the clavicular triangle, but it may be much larger, and bulge forwards the sterno-mastoid muscle ; in other cases it grows downwards and backwards towards the pleura, which then becomes thickened and adherent to the lung, and the lung may be incorporated in the sac of the aneurysm. The radial pulse on the same side is weakened and delayed ; there is often œdema of the arm, and usually distension of the external jugular vein, and the venous obstruction may even cause gangrene of the arm. From pressure upon the brachial plexus the patient may suffer from pain down the arm, numbness, or muscular weakness, and pressure on the phrenic nerve may cause spasm or paralysis of the diaphragm. The aneurysm may remain stationary or progress but slowly; in many cases spontaneous cure has been accomplished; in other cases steady enlargement ending in rupture of the sac occurs. The condition most likely to be mistaken for subclavian aneurysm is that in which the artery is raised over a cervical rib; a little care will at once guard against this error.

Treatment.—The surgical treatment of subclavian aneurysm is very unsatisfactory. In all cases rest and constitutional treatment should be well and fully tried, and to these means, careful direct pressure upon the sac may be added. If these measures fail, the question of treatment is one of great gravity. Where there is room on the proximal side of the aneurysm for compression of the artery that should be first employed, and if it fails the artery may be ligatured in its second or the commencement of its third part. Very often this is impossible. Ligature of the first part of the artery has never been successful and should not be practised. Ligature of the innominate artery has been recovered from in only two instances out of eighteen operations; but with the practice of aseptic surgery, and the use of a ligature so applied as not to lacerate any of the arterial wall, better results may be hoped for. In any case, however, this operation must be attended with grave danger, owing to the depth and important relations of the artery. Distal ligature of the artery has never succeeded, but combined with amputation at the shoulder joint, may be successful. By removing the limb, the amount of blood passing through the subclavian artery is greatly diminished, and the pressure in the artery is lowered; and this fact, together with the proliferation of the intima that results from it, greatly favours the consolidation of the aneurysm and obliteration of the artery. The other means that have been employed are manipulation of the sac and electrolysis, each of which has attained a limited success. The matter may be summed up by saying that where active surgical measures are called for, if possible a ligature should be placed on the cardiac side of the aneurysm, but if from any cause this is impossible, or imperils the sac, or fails, the arm should be amputated at the shoulder joint, the vessel being ligatured as high up as practicable, as the first step of the operation.

Aneurysm of the vertebral artery in the neck is always traumatic in origin. The artery may be tied just before it enters the transverse process of the sixth cervical vertebra, or the sac may be laid open and the artery closed by very careful and firm plugging, or electrolysis may be employed.

Aneurysm of the axillary artery.—Aneurysm may arise from any part of the axillary artery; it is more common in men than women, and on the right than the left side. Traumatic aneurysm from stabs and wounds, or from injury attending dislocation of the shoulder and its reduction, is not infrequent. Injury also plays an important part in the production of spontaneous axillary aneurysm, owing to the very free movement of the arm at the shoulder joint, and the frequency of injuries to the shoulder.

Symptoms.—Owing to the laxity of the surrounding tissues, an axillary aneurysm grows rapidly and may attain a large size. Most often it projects forwards between the clavicle and the pectoralis major muscle; it may grow up under the clavicle into the clavicular triangle; the strong axillary aponeurosis retards its downward progress. The tumour, if large, interferes with the free movement of the arm, especially with abduction; the head may be held inclined to the same side and the outer end of the clavicle may be pushed up. By pressure upon the axillary vein it causes blueness and œdema of the hand, fore-arm, and arm, and afterwards of the chest-wall; there is often severe lancinating pain down the inner side of the arm to the elbow, and weakness, numbness, or even paralysis may result from the pressure on the brachial plexus. The aneurysm may burst into the shoulder joint or erode the humerus, or extend in between the ribs and displace the lung. From the obstruction of the artery, the brachial or radial pulse may be lost. Owing to the great size of the sac and the looseness of its surroundings, it is peculiarly liable to inflammation; when the venous obstruction is very great, gangrene may ensue. *Varicose aneurysm* has been met with in the axilla, but only rarely.

Treatment.—Cases of spontaneous cure are extremely rare. The surgeon should first of all treat the disease by digital compression of the subclavian artery just above the clavicle, where it lies upon the first rib; and, if necessary, the patient may be placed under an anæsthetic while continuous pressure is maintained. At the same time, gentle direct compression of the sac to compensate for the absence of the tense tissue which usually surrounds an aneurysm may be of service. Where the aneurysm has so spread up into the neck, or has so raised the clavicle that digital compression of the subclavian artery is impracticable, Esmarch's bandage may be applied to the limb up to the sac for one to two hours, with a view to leading to coagulation of the blood. Should these means fail, the subclavian artery should be tied in the third part of its course, or in the second part if the vessel is encroached upon by the tumour, or is found very diseased. This operation may be rendered very difficult by the proximity of the sac or the displacement of the clavicle. Secondary hæmorrhage may occur after this operation, and should be treated by a carefully-applied compress; gangrene is rare, owing to the freedom of the anastomotic circulation. But two special dangers attend the operation, viz. inflammation of the aneurysm and intrathoracic inflammation. The latter is the more frequent cause of death; it may take the form of pleurisy or pneumonia, or of cellulitis in the anterior mediastinum, with secondary pericarditis. The causes of these complications are wound of the pleura at the time of the operation, implication of the pleura in the sac of the aneurysm, rupture of an inflamed aneurysm into the pleura or lung, injury to the phrenic nerve, and the extension of septic inflammation along the cellular tissue between the scalene muscles, which is continuous with that in the mediastinum. Where the aneurysm has been situated low down, it has been recommended to tie the first part of the axillary artery, but it is better to tie the subclavian trunk. In the case of a very large axillary aneurysm it has been advised to tie the subclavian artery, and then at once amputate at the shoulder joint. For sacculated *traumatic aneurysm*, digital compression of the subclavian artery, with careful pressure on the tumour, should be tried first, and that failing, the third part of the artery should be tied. If the sac is very thin and threatens to rupture, it would be better to lay it open and tie all the arteries opening into it. *Varicose aneurysm* must be treated by ligaturing the artery above and below the sac.

Inflamed axillary aneurysm.—The inflammation may arise spontaneously or after ligature of the artery above; in the latter case the inflammation may spread from the wound to the sac, or it may result from the sudden solidification of a large quantity of blood. The condition is recognised by the onset of pyrexia, with increasing swelling of the tumour, local heat, redness, and pain; then fluctuation may be detected, an abscess burst, and the escape of discoloured pus and coagula be followed by free arterial bleeding. The abscess may, however, burst into the pleura, or into a bronchus, and the pus be coughed up. If suppuration occur after ligature of the subclavian artery, an early incision should be made into the fluctuating part, and, if bleeding occur, an attempt should be made to tie the bleeding vessel, and failing this, the limb should be amputated. When occurring spontaneously, amputation is usually indicated; but if the aneurysm is small, and the sac firm, the subclavian artery may be first tied.

Recurrent pulsation.—If ligature above the sac fail to cure the aneurysm, carefully-adjusted direct pressure, as by a sand-bag, should be tried. If that fails, the surgeon has to choose between employing a direct coagulant, such as electrolysis or acupuncture, laying open the sac and tying all vessels communicating with it, and disarticulation of the limb. Laying open the sac has been successful, but the milder means should be first tried, and if the "old" operation of Antyllus is undertaken, the surgeon must be prepared to amputate if he is unable to complete it. *Diffuse aneurysm*, or aneurysm with threatened *gangrene* of the arm, can only be treated by amputation at the shoulder joint.

Brachial, radial, and ulnar aneurysm.—Aneurysms are rare below the axilla, except those at the bend of the elbow, due to traumatism. Not only are the majority of such aneurysms traumatic in origin, but there is no other part of the body where so many traumatic aneurysms have been met with. Nearly all the spontaneous aneurysms are associated with cardiac disease and embolism. At the bend of the elbow an aneurysm generally grows upwards along the brachial vessels, being limited by the biceps on the outer side, and by the internal intermuscular system internally; it may extend as a flattened sac under the biceps.

Symptoms.—Owing to the proximity of the median nerve, pain is often felt along the palmar surface of the thumb, forefinger, middle finger, and the outer side of the ring finger. If the aneurysm attains a great size, the flexor and pronator muscles of the fore-arm may be paralysed. The anastomotic circulation is so free in the upper limb that it imperils the success of the ligature, owing to the rapidity and freedom with which blood returns to the sac; it also renders the danger of secondary hæmorrhage greater than in the leg, while the danger of gangrene is much less. Excision of the aneurysm is therefore more widely practised here than elsewhere.

Treatment.—*Aneurysms at the elbow*, whether traumatic or spontaneous, should first of all be treated with compression of the artery above the tumour; if that fails to cure, the choice lies between the Hunterian ligature and excision of the sac; owing to the superficial position of the artery, and the ease with which we can control all the vessels of the upper limb by a tourniquet, excision of the aneurysm is often to be preferred.

Varicose aneurysm at the elbow may be treated with Esmarch's bandage, and if that fails, by laying open the sac and carefully tying every vessel opening into it. The only treatment necessary for an *aneurysmal varix* is some form of external support, such as an elastic armlet.

Aneurysms of the fore-arm are to be treated first by direct and indirect compression; full flexion of the elbow is the best means of compressing the brachial trunk. Esmarch's bandage may also be employed. If these means fail, when the aneurysm is deep beneath the flexors of the fore-arm the brachial artery should be tied low down in the arm; but when the aneurysm is superficial in the lower half of the fore-arm it should be carefully excised.

Aneurysms of the hand are not common. Compression of the radial and ulnar arteries at the wrist, or of the brachial artery by acute flexion of the elbow, together with direct pressure over the sac, should first be tried. When that fails, excision of the aneurysm, or the Hunterian ligature, must be performed. A few years ago the writer had a case of traumatic aneurysm of the radial artery immediately before its entry into the palm; it was successfully dealt with by excision of the sac and double ligature of the radial artery. For aneurysms deep in the palm, ligature of the brachial artery is the best treatment. Small traumatic aneurysms of the *digital arteries* are sometimes met with; they should be excised.

Inguinal aneurysm.—Aneurysms of the external iliac and common femoral artery are most conveniently discussed together, for they are most common at Poupart's ligament, and then often grow both up into the abdomen and down into the thigh, the sac presenting two lobes with a constriction opposite the fold of the groin.

Symptoms.—Inguinal aneurysm is commonly of slow growth and may be long unobserved, but it may form a very large tumour in the iliac fossa; the part in the thigh expands less rapidly than that in the belly, owing to the support it receives from the strong fascia lata. By pressure on the femoral and internal saphena veins, the tumour causes œdema and lividity of the lower limb, and pain along the front of the thigh, or down to the inner side of the knee and instep, may be caused by irritation of the genito-crural or anterior crural nerve. The natural termination of inguinal aneurysm is external rupture and death from hæmorrhage.

Diagnosis.—Great care is often required in deciding the nature of a tumour in the groin; and when any special difficulty is met with, an examination should be made under an anæsthetic, so that the abdominal muscles may be completely relaxed. The conditions which simulate aneurysm are abscess, pulsating tumours, and enlarged glands; in several instances, inguinal aneurysms have been opened in mistake for abscesses. The directions already given will enable the surgeon to make a correct diagnosis. An aneurysm of the aorta, even in its thoracic part, may extend down to Poupart's ligament, and then form an external swelling, at first sight like that of an inguinal aneurysm.

Treatment.—Constitutional treatment should always be carefully tried before resorting to surgical measures. Where it fails, proximal compression or ligature, or galvano-puncture may be resorted to. Proximal compression of the lower end of the aorta by Lister's compressor, the patient being under an anæsthetic, has been successful, and should be tried in all cases where there is no special bar to it, such as great size of the aneurysm or obesity of the patient. Distal compression of the common femoral artery and gentle direct compression of the sac may be combined with it. Where the aneurysm is low down in the groin, ligature of the external or common iliac artery—by preference the former—has been very successful. But if the aneurysm is too high for this operation, the case presents great difficulties; ligature of the aorta has never been recovered from, and distal ligature has never been successful in leading to the cure of an inguinal aneurysm. In these cases galvano-puncture or acupuncture may be employed. Excision of the sac can only be practised in special circumstances, where the aneurysm is low in position, small in size, or is the seat of recurrent pulsation; in other cases it is difficult of execution, and very dangerous from hæmorrhage. The sudden consolidation of an inguinal aneurysm is liable to be followed by suppuration around the sac, owing to the looseness of the cellular tissue in which it lies. Wounds of the iliac artery are usually fatal, but a single case of *inguinal varicose aneurysm* has been recorded.

Aneurysm in the buttock.—An aneurysm in the buttock may spring from either the gluteal or sciatic artery, and is about as often traumatic as spontaneous in origin. Gluteal aneurysm is quite four times as frequent as sciatic.

Symptoms.—The tumour is buried deep in the buttock, and unless large, may escape notice for a long time; if connected with the sciatic artery it may grow into the pelvis. At first the tumour is small, firm, deep, and fixed, and closely simulates a pulsating tumour of bone; later in its course it may attain a great size and come to resemble an abscess, and to render certain the diagnosis between abscess and aneurysm a fine exploring needle should be passed into the swelling, when, if it is an uncured aneurysm, bright red blood will escape. The chief symptoms of these aneurysms is limitation of movement at the hip, pain in the part, and pain from pressure upon the great sciatic nerve. When small, the height of the swelling on the buttock distinguishes a gluteal from a sciatic aneurysm, but when of large size it may be impossible to distinguish one from the other; the early onset of pain along the sciatic nerve, and the extension of the tumour within the pelvis, are points in favour of its being connected with the sciatic artery. Spontaneous cure has been known to occur, but the general termination of these cases is death from external hæmorrhage.

Treatment.—Entire rest and the usual constitutional treatment should be given a fair trial. Aneurysm in the buttock is well adapted for galvano-puncture, and the employment of other direct coagulating agents; there is no danger of wounding any important structure in operating, and if portions of clot are washed into the branches of the artery, the embolism is without special danger. At the same time, the tumour is particularly badly situated for the employment of other means. The distal ligature cannot be practised; it is very rarely possible to place a ligature on the diseased artery between the pelvis and the aneurysmal sac; excision of the tumour is difficult, and there is danger of primary and secondary hæmorrhage, but it has been successful in five cases. Ligature of the internal iliac artery also is available, and has been successful, and with the improved method of reaching this artery through the peritoneum, instead of behind it, this operation is to be recommended.

Aneurysm of the femoral artery.—Spontaneous aneurysm of the femoral artery is not very common, as in the upper part of its course the artery is well supported by its strong fibrous sheath, and in Hunter's canal it is surrounded by muscles as well as covered over by a strong aponeurosis. It is more frequent in Scarpa's triangle than in Hunter's canal, and is an affection limited to the male sex. It may be fusiform, but it is more commonly sacculated in nature.

In Scarpa's triangle it is usually globular in form and often projects through the saphenous opening; but in Hunter's canal the pressure of the surrounding parts causes the tumour to assume a more flattened outline.

Aneurysm of the *profunda femoris* artery occasionally occurs. It is distinguished from femoral aneurysm, which it closely simulates, by the fact that the pulse in the popliteal and tibial arteries is the same on the two sides, and that the unaltered femoral artery can be traced pulsating over the side of the tumour in the upper part of the thigh.

Traumatic aneurysms in connection with the superficial or deep femoral artery are met with, and they may be circumscribed or "diffused," and in the latter case the swelling attains a great size. *Varicose aneurysm* has been met with at the groin, and *aneurysmal varix* both at the groin and in Hunter's canal.

Spontaneous cure of femoral aneurysm sometimes occurs, but the more frequent natural termination of the disease in this situation is rupture externally and death from hæmorrhage.

Treatment.—Rest and constitutional measures, combined with slight direct pressure, may prove successful. The other surgical measures that may be employed in cases of spontaneous aneurysm are compression by Esmarch's bandage, digital or instrumental compression of the common femoral artery, and the proximal ligature. Either the external iliac, common femoral, or superficial femoral artery may be tied. If there is room above the aneurysm the ligature should be placed upon the superficial femoral trunk. Ligature of the common femoral artery has been discarded by many surgeons on account of the danger of hæmorrhage, owing to the proximity to the ligature of one or more of the branches of the artery, and also because the anastomotic circulation is less free when this artery is ligatured than when either the external iliac or the superficial femoral artery is tied. With an aseptic ligature so applied as not to sever any of the arterial coats, the danger of hæmorrhage can be obviated, and the operation has found more favour of late. Ligature of the external iliac artery is, however, preferred by some surgeons; it is liable to fail on account of the extreme freedom of the anastomotic circulation. In a case where this happened, Mr. Rose excised the aneurysm with complete success, and this example should be followed in similar cases. Aneurysm of the *profunda femoris* artery is to be treated by compression or ligature of the common femoral trunk.

Circumscribed traumatic aneurysm is treated like spontaneous aneurysm, but the *diffused* variety can only be dealt with by cutting into the swelling, turning out the clot, and applying a ligature above and below the mouth of the sac.

For *varicose aneurysm* of the groin, the external iliac artery has been tied, but without success, and it would be better in any such case to tie the artery above and below the opening into the sac. Ligature of both the common femoral artery and vein exposes the patient to great risk of gangrene. *Aneurysmal varix* in the groin should be treated by elastic support; in a case of aneurysmal varix in Hunter's canal the writer saw the femoral artery tied above and below the opening into the vein, with entire success.

Popliteal aneurysm.—With the exception of the aorta, aneurysm occurs more frequently on the popliteal than any other artery. The affection is often symmetrical, the two tumours being noticed simultaneously, or the second appearing after the cure of the first. The frequency of popliteal aneurysm is due to several causes:—(1) The contact of the artery with the bone. (2) The alteration of the length and calibre of the vessel occurring in the frequent and rapid movements of the knee-joint. (3) The want of support of the artery; all the other main arteries of the lower limb are well supported by muscles or fasciæ, the popliteal alone is surrounded by loose cellular tissue and fat. (4) The termination of the artery in small arteries which lie deep among the muscles; in the mouth of these branches an embolus may be caught and prove the starting-point of an aneurysm.

Symptoms.—Both fusiform and sacculated aneurysms are met with in the ham, and the latter may spring from either the front or the back of the artery. When springing from the back the sac often attains a considerable size, compresses the popliteal vein and nerve, and tends to rupture. Sacculated aneurysm of the front of the artery is usually of small size; it is liable to erode the femur or tibia, and to cause effusion into the knee joint, or even to rupture into the articulation. Popliteal aneurysm may spread up along the artery into Hunter's canal, or down into the leg and rupture beneath the calf muscles, or it may burst subcutaneously. Sometimes the patient can state exactly when the tumour developed, but more often its origin and early progress are insidious. Often the first symptom is pain about the knee or down the leg into the ankle, together with stiffness of the knee, and the condition is frequently mistaken for rheumatism; if there is effusion into the knee joint, still further support is lent to this error. Pressure upon the internal popliteal nerve causes pain shooting down to the toes, cramps, muscular weakness, and paralysis. Pressure upon the popliteal vein causes cyanosis of the limb below, with distension of the superficial veins and œdema. Erosion of the bones or of the ligament of Winslow is attended with constant aching or hot burning pain in the joint itself, with great stiffness of the joint and pain on attempting to move it.

When a popliteal aneurysm opens into the knee joint, the synovial cavity becomes suddenly distended, the part is hot to the hand, and if the common femoral artery is compressed, the swelling of the knee yields a little to gentle compression. In any case of doubt, the introduction of a grooved needle or fine trocar will demonstrate the nature of the fluid in the joint.

Popliteal aneurysm must be diagnosed from abscess, bursal cyst, pulsating sarcoma, and a solid tumour over the fascia. The signs by which the diagnosis can be arrived at have been already mentioned.

An *aneurysmal varix* of the popliteal artery and veins has been several times observed.

Treatment.—Spontaneous cure may occur, and in some cases all that is required is to place the patient at rest in bed, with the affected limb raised and lightly bandaged, and to order a suitable diet. Should this simple treatment fail, and the tumour is small and firm, flexion should be tried, or Esmarch's bandage may be employed.

Digital or instrumental compression of the femoral artery has been successful in a large number of cases; if the aneurysm does not quickly consolidate under this treatment, it should not be prolonged for the reasons already given, but the artery should be tied.

Ligature on the proximal side of the aneurysm is a very successful operation; it maybe applied to the superficial femoral artery at the apex of Scarpa's triangle, or in Hunter's canal, or to the popliteal artery just below the opening in the adductor magnus muscle. Of these situations, the one that has been most favoured by surgeons is the apex of Scarpa's triangle, partly because the artery is more superficial and more readily accessible there, partly because it is far removed from the seat of disease, and the artery is probably healthy. But the great length of artery between the ligature and the aneurysm is really a disadvantage, for when the operation is successful, it causes either a double obliteration of the main artery—at the seat of ligature and at the aneurysm—or the obliteration of the entire length of artery, and in either case it causes an unnecessary interference with the circulation in the thigh; also failure to cure the aneurysm —or so-called "recurrent pulsation"—is frequent. For these reasons, and also because the artery close above an aneurysm is not so often diseased as to render its ligature perilous, ligature of the highest part of the popliteal artery has been recommended of late, and in practice this has been very successful. The operation is not difficult, with modern methods it is not dangerous, and in its effect upon the aneurysm it is more certain than ligature of the femoral vessel. Hunter, in his first three cases of ligature of the femoral artery for popliteal aneurysm, included the vein with the artery, but in his fourth case he tied the artery only, and since then surgeons have taken special pains not to wound or include the companion vein.

Where the aneurysm is growing rapidly, and especially if with rapid enlargement there is a loss of clear outline and forcible pulsation, showing that the sac is leaking, the artery above should be tied without any delay, and without trial of any other method.

If after ligature of the femoral artery pulsation recurs in the aneurysm, that is to say, if the operation has failed to cure the aneurysm, digital compression at the groin, with direct pressure over the tumour, is usually successful; if not, the surgeon must either tie the artery lower down, in Hunter's canal or in the ham, or must excise the aneurysm.

For secondary hæmorrhage after the ligature the wound may be carefully plugged or, better, opened up, and the artery tied above and below the bleeding point, great care being taken not to rupture the inner coats of the artery, and to leave the wound aseptic; on the appearance of a few drops of bright blood in the discharge from the wound—the precursor of "secondary hæmorrhage"—the femoral artery should be controlled in the groin by compressors or assistants' fingers, and if this prevents freer hæmorrhage, the control should be maintained until the wound is consolidated. If this treatment is unsuccessful, amputation through the thigh is necessary; ligature of the artery at a higher level either fails to arrest the bleeding or it causes gangrene of the limb.

If *moist gangrene* follows the ligature, the limb should be at once amputated above the knee without waiting for any "line of demarcation"; but if the gangrene is *dry*, the separation may be left to nature, the mummified part being meanwhile swathed in iodoform wool.

When the aneurysm is threatening to become diffused, or is "*leaking*" into the tissues, the artery above should be at once ligatured, and with great hope of success. But if the aneurysm has really *ruptured* into the cellular tissue, or externally, amputation through the lower end of the femur is the only resource. If the sac has ruptured into the knee joint, the artery above should be tied, in the hope that the aneurysm may be consolidated and the blood absorbed from the joint; but if the tumour has caused extensive absorption of the femur and disorganisation of the joint, amputation should be at once practised.

For *inflammation of the sac*, ligature of the artery above with appropriate local treatment is indicated; if suppuration occurs, a free incision should be made to liberate the pus as soon as its presence is detected; if, upon this, hæmorrhage occurs, the surgeon should attempt to excise the sac and secure the artery beyond the area of inflammation, and failing in this, he must amputate the limb.

Gangrene of the leg and foot may occur as the direct result of the aneurysm, either from venous obstruction, embolism, or arterial thrombosis. If from venous obstruction it will be *moist* in variety, and involve the whole limb up to the knee; the only treatment that can be employed is immediate amputation above the aneurysm. If the gangrene is arterial and *dry*, and it involves all the foot and part or the whole of the leg, amputation is likewise indicated; but if it is limited in area, involving the toes only perhaps, these mummified parts may be left to separate, and the aneurysm treated on ordinary principles.

Aneurysm in the leg and foot.—Aneurysm is rare below the ham, and in the leg and foot is usually traumatic in origin, resulting from gunshot wounds, stabs, punctures, and fractures.

When complicating fracture of the leg, the firm application of the splints employed for this injury is often sufficient to cure the aneurysm. In other cases, *circumscribed aneurysm* in the leg must be treated by compression of the femoral artery or ligature of the popliteal trunk. If the aneurysm is *ruptured*, an attempt may be made to tie the artery above and below the sac, and failing this, amputation must be performed.

For *aneurysm at the ankle or in the foot*, the "old operation" of excision of the sac and ligature of each artery communicating with it, is the best course to pursue. Compression or ligature of the artery above is very liable to fail, owing to the very free anastomoses between the arteries of the foot.

XXVI. INJURIES AND DISEASES OF LYMPHATICS.

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Wounds of lymphatic vessels.—Except in very rare instances, the lymphatics of the extremities show little of the results of injury, although in any extensive laceration their trunks must of necessity be severed. The lymph that exudes from the divided vessels is not to be distinguished from the blood and serum which are poured out in much larger quantity, the numerous valves prevent any considerable reflux, and the divided ends are quickly closed by the collapse of the tissues, aided also by the coagulation of the lymph itself as it issues from the vessels.

Lymphatic fistulæ have sometimes developed on the surface as a sequel to the opening or removal of lymphatic glands, and very large quantities of lymph have been discharged, and death, it is said, has followed from exhaustion.

The most important lesion of the lymphatic vessels is **section** or **rupture of the thoracic duct,** which, though so deeply placed and so well protected throughout its course, has been divided by stabs and gunshot wounds, and lacerated by such severe injuries as a crush of the thorax or fracture of a vertebra. More commonly, a rupture of the duct has occurred as the result of closure of the lumen of the tube by the pressure of a tumour, or by contraction after destructive inflammation.

In certain of the reported cases of division of the duct, associated with external wound, a whitish fluid, which was whiter and more opaque after meals, escaped at the external opening. In instances in which there was no surface wound the fluid has been effused into the pleural cavity in such amount as to call for repeated tappings. The fluid thus evacuated coagulates on exposure. A diagnosis of the injury is impossible without evidence of chylous fluid either escaping from a wound or obtained from the pleural cavity by tapping. In every case death must follow (in a period of weeks rather than of months) from marasmus, consequent upon the discharge. The *treatment* is purely symptomatic.

Lymphatic fistula, giving rise to **lymphorrhœa,** or **lymphorrhagia,** occurs, as has been already stated, sometimes from wound. It occasionally also arises as the result of obstruction in the glands or vessels due to inflammatory changes, and is more often met with in the lower than the upper limb. It is characterised by the discharge of a thin colourless fluid, which rapidly coagulates into a gelatinous film. Sometimes the fluid has a milky appearance, leading to the probability of a communication existing with the lacteals or the thoracic duct. The discharge does not usually proceed from a single spot, but exudes from numerous small vesicles which appear upon the surface of the skin around the site of the obstruction (Fig. 186). This occurs to a greater extent after exercise, and is intermittent in amount, subsiding when the limb is raised and the patient at rest. In a case recently under my care, which occurred as the result of suppuration of a gland over the saphenous opening during an attack of small-pox, ten ounces were collected in a few hours, and in some instances the quantity is much more profuse. This fluid was examined by Dr. Manson, and pronounced to be chyle-like, and not pure lymph. On analysis it was found to be composed of —water, 95 per cent. ; total solids, 5 per cent. The latter contained 53 per cent. of fat ; ash, 47 per cent. (sodium chloride ; traces of phosphates ; no sulphates). A second examination, six months later, gave a nearly identical result. In this case there was obstruction at the site of the inflamed gland, but in most other instances there has also been noted some diseased or dilated condition of the lymphatic vessels (lymphangiectasis), which has been evident upon the surface of the skin. The limb below the site of obstruction has sometimes shown hypertrophy of all its tissues, but in all cases it is considerably enlarged, and the skin is affected with a fibrous hyperplasia.

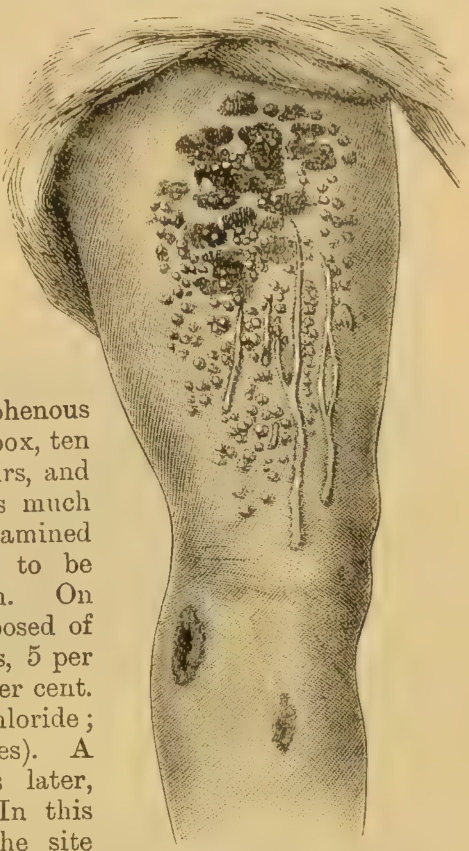
Fig. 186.—Lymphorrhœa due to Blocking of Lymphatics. (Lancereaux.)

The only *treatment* consists in raising and bandaging the whole limb, with pressure upon the site of the fistula. All operative proceedings are worse than useless.

Dilatation of lymphatic vessels.—The lymphatics are subject to the same congenital affections as the blood-vessels, and give rise to conditions which will be mentioned later (page 678).

Lymphangeioma is the term given to a dilated condition of the larger lymphatic vessels, in which cystic spaces are formed, often of large size, associated with hypertrophy of the vessel wall and atrophy of the intervening tissue (Fig. 187). Sometimes this dilatation gives rise to a single cyst, the walls of which consist of fibrous tissue lined with epithelium, and the contents of more or less clear fluid.

Lymphangiectasis is the result of a varicose condition of the lymphatics analogous to that of the blood-vessels, and is most common in the lower limb, especially in the inguinal region. It consists of a circumscribed dilatation of the vessels, and gives rise to swellings on the surface, varying in size from that of a pea to that of a walnut. These are not painful, except when over-distended ; and if aspirated or punctured—as they have sometimes been under a misapprehension of their true nature—they yield a clear or milky coagulating fluid. To a dilatation of these natural channels, causing distension and rupture of lymph spaces in the papillary layer of the skin, Hutchinson has applied the term lupus lymphaticus—a somewhat misleading appellation (Fig. 188).

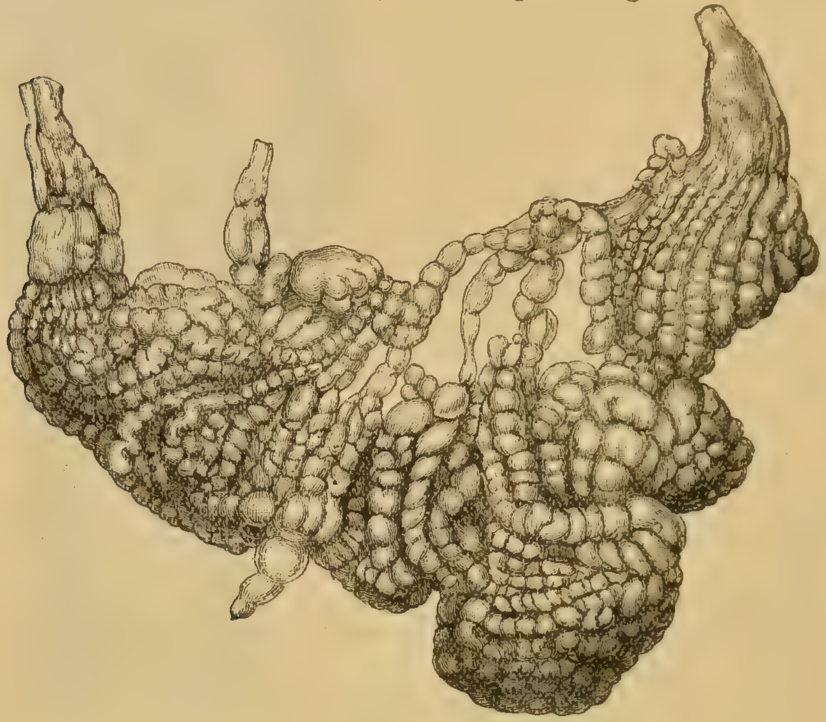

Fig. 187.—Lymphangeioma from the Inguinal Region. (Lancereaux.)

Both these conditions may be brought about by the pressure of tumours—such as an aneurysm—compressing the thoracic duct, or of malignant tumours in the course of that vessel, or arising in the groin or axilla. The pressure thus exercised may so obstruct the return of chyle in the one case or of lymph in the other, that the vessels beyond become dilated and varicose, and may either burst and discharge their contents, or may form dilated pouches at the site of the valves in which the lymph remains stagnant. In this way may be accounted for many instances in which chyle is found to be mingled with the lymph flowing from fistulæ in the groin or scrotum, or passed by the natural passages with the urine, and occasionally poured out in large quantities into the peritoneum.

From whatever cause an obstruction to the lymphatic circulation may arise, it is attended by a hypertrophied condition of the limb below, causing it to assume a permanent enlargement, due mainly to a solid œdema of the cellular tissue. The skin may remain smooth or brawny, or may become rough and coated with a thick incrustation of epithelium, which resembles the condition seen in elephantiasis Arabum. Repeated attacks of inflammation of an erythematous character are prone to involve all the tissues, and leave the enlargement greater than before.

Angeioleucitis or **lymphangitis.**—These terms are applied to an inflammation occurring in the lymphatic vessels, as a result of the entrance into their channels of some irritant and probably septic product. This may originate in the tissues from which the lymphatic radicles arise: as, for example, from the irritation of a blister, the application of iodine, or even from sunburn. Lymphangitis is a frequent concomitant of erysipelas, especially in its milder and cutaneous forms. Acute inflammation of lymphatics is much more frequent in young adults and children than in old people; but in those debilitated by excesses, or alcohol, or in the subjects of Bright's disease, it occurs on very slight provocation.

But the more usual source of origin is the direct absorption of a morbid material through an abrasion, often slight and unperceived, such as a scratch, or prick, into which the septic products of some decomposing tissue have found entrance. Thus it is of frequent occurrence amongst butchers and fishmongers, or amongst those engaged in dissecting or conducting post-mortem examinations, and especially where these duties entail contact with septic or much decomposed tissues. In a wound where suppuration is free and drainage is secured, as from the flaps of an amputation, absorption does not take place, since the current of discharge from the granulations sets in the opposite direction; but when the pus is confined by a scab or by any form of dressing, such as collodion, the lymphatics may readily become inflamed by the material which they convey. Thus it is not uncommon to find it occur as the sequel to a small pustule or the sting of an insect, and it will even result when the skin of the hand is brought in contact with some particularly virulent material when no abrasion can be detected.

An example of tuberculous lymphangitis is shown in Fig. 62, page 341.

Pathology.—The exciting cause of this affection is undoubtedly the entrance into the lumen of the vessels of some septic alkaloid (ptomaine), derived from decomposing tissues or from a parasitic micro-organism which has gained access to the wounded part This excites an inflammation of the vessel, and particularly of its outer coats, which, spreading to the surrounding tissue, causes a perilymphangitis; the affected vessel can then be perceived superficially as a thickened cord beneath the skin. The contents of the vessels are increased by the cells from the intima, by white blood corpuscles, and later by pus. The inflammation may subside and the lymphatics regain their natural condition, but localised abscesses may form, and, bursting on the surface, will lead to a complete obliteration of the vessels in which they arise. When this happens in the deeper chain of lymphatics, a very severe and extensive cellulitis may ensue.

Symptoms.—The affection is first notified by pain and throbbing, with great tenderness at the site of origin, around which there appears a red areola. Radiating from this spot, the skin of the arm or leg becomes marked with red striæ, which sometimes coalesce to form an erysipelatous area. Although marking the course of the lymphatic vessels, these striæ are much broader than the vessel itself, and are lost as they approach the next set of glands, disappearing at the elbow or popliteal space, and never extending beyond the axilla or groin. At the same time pyrexia supervenes, and may be extreme, with rigors of more or less frequency. The principal pain is seated in the region of the glands, but there is great tenderness along the line of the vessels, which are evident as hard cords beneath the skin. The inflammation is more often acute than chronic, and in the former case may last only a few days, and terminate in resolution, the parts returning to a normal state. But if, as is usual, pyogenic organisms have formed part of the infecting virus, and suppuration takes place, abscesses will form at various parts in the course of the vessels, and the case may last for many weeks, and the abscesses may not close until the granulations lining their cavities have been scraped or treated with some such caustic antiseptic as a solution of chloride of zinc. When the deeper set of lymphatics is involved, the superficial redness is not so marked, but the pain is deep-seated and severe, and there is œdema along the whole

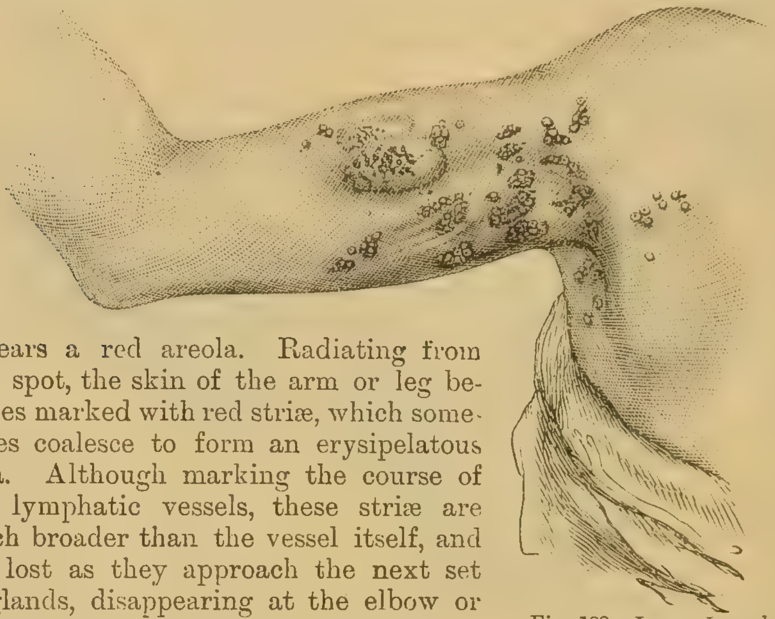

Fig. 188.—Lupus Lymphaticus. (From a drawing in St. Bartholomew's Hospital Museum.)

or part of the limb. Suppuration in these parts extends widely and leads to extensive loss of tissue, and the subsidence of inflammation is followed by an obstinate and chronic œdema. The glands are very early affected, as shown by dull aching pain, followed by acute tenderness even before there are the above signs of lymphangitis. They are at first swollen and movable in the surrounding tissue, and may remain so for some time after the inflammation has subsided in the vessels, when they gradually resume their normal condition. But often suppuration occurs in them, and this is marked by increasing pain, tenderness, and throbbing, by continued and often excessive pyrexia with rigors, while the gland enlarges and the cellular tissue around becomes inflamed, and signs of suppuration are soon evident. The abscess thus formed may attain a large size, but the glands are not affected beyond the axillary or inguinal regions.

The *constitutional symptoms* vary with the acuteness of the process. The fever, the superficial redness, the pain in the glands may pass off in a few hours; but, on the other hand, they may be followed by rigors, and all the evidences of septicæmia or pyæmia. When the pain in the glands persists even after all redness of the striæ has disappeared, suppuration generally occurs at a shorter or longer interval.

Treatment.—In dealing with this affection, the nidus from which the poison has been absorbed must be sought. If this be an open wound, it must be cleansed thoroughly and washed with strong solutions of perchloride of mercury; if covered with a scab, the crust or epithelium must be removed. Should there be no perceptible wound, an incision into the red and throbbing areola must be made, and lotions of carbolic acid or perchloride of mercury applied. Warmth is the greatest alleviator of pain, and much relief is given by soaking the whole limb in hot boracic baths, and by warm boracic fomentations along the whole course of the inflamed lymphatics. A mercurial purge, followed by salines, should be administered, and the limb raised throughout its whole length. Quinine should be given in large doses from the first onset of pyrexia, and opiates if the pain be excessive. To subdue the swelling of the glands, nothing is so effectual as warmth, which may be applied in the form of hot lead and opium lotions or boracic fomentations; and this also has the effect of encouraging the pus, if it has formed, to come to the surface, and as soon as fluctuation can be detected, free incision and drainage give instant relief, the wound being dressed with strict antiseptic details.

The œdema which occurs with inflammation of the superficial lymphatics will gradually subside as the lymph finds its way through its former channels, or when an anastomosis is established through the deeper set of vessels; but when the latter set of lymphatics are blocked or destroyed, the œdema is more severe, and often permanent. Frequent bathing in hot water, massage, and the application of elastic bandages are means by which this may be diminished, or even cured.

Lymphadenitis. **Symptoms.**—As described in the preceding section, inflammation of the lymphatic glands is generally due to the absorption of septic material, although the effects may be first perceptible in the glands, and no traces of its passage along the lymphatics may have been evident. When the process is *acute*, the symptoms are well marked and the diagnosis is easy. Pyrexia of greater or less intensity, often accompanied with rigors, precedes a feeling of swelling, with pain of dull aching character, rapidly becoming more acute and throbbing, and situated in the known position of lymphatic glands. All movement of the limb or part affected is extremely painful. Sometimes a single gland, sometimes a whole chain, is attacked. The swelling increases, the skin for some distance around becomes œdematous, and afterwards red and tender. So soon as suppuration has taken place the pain may be less severe, but the swelling increases. Pus may form in several portions of the gland at once, or the whole may be converted into an abscess, confined by the fibrous structures of the gland. At the same time, the surrounding parts break down, and an abscess is formed in the loose cellular tissue, which may spread over a large area. The changes that take place affect all the component parts of the gland. The fibrous cortex is first affected, and becomes thickened by the increase in size and number of its blood-vessels, and by the exudation of blood and leucocytes. The follicles of adenoid tissue are at first red and hyperæmic, but soon become paler as the corpuscles are converted into pus-cells. Thus small collections of pus are formed, and these, by coalescing, convert the whole structure into an abscess, confined by the fibrous coat of the gland, which gradually softens, and allows the matter to escape into the already inflamed and suppurating cellular tissue. Other glands in the chain may become inflamed and follow the same extreme course.

The process, however, may fall short of this condition and assume the *subacute* form. In this, all the component tissues, and particularly the fibrous portions of the gland, become congested and swollen, but pus is not formed in the surrounding cellular tissue; and if it occur in the substance of the follicles, the collections do not coalesce, the gland subsides to a smaller size, but remains larger than natural and is prone to recurrent attacks of inflammation on slight provocation. A further and subsequent change may result in calcification either of a part or of the whole gland. Sometimes the substance of the gland breaks down, forming what is known as a *suppurating bubo*, and the skin gradually becoming involved, the contents are discharged on the surface, and a sinus is formed, which leads down to the necrosing tissue of the gland. In cases where the primary inflammation is of a simple nature (*sympathetic bubo*), this readily admits of cure, but when it is due to the absorption of a specific virus, as from a soft chancre, the discharge from the suppurating gland has not only the same characteristics as that from the primary sore, and is likewise inoculable, but will cause the surrounding tissue to break down even to a considerable distance, and to present the ragged edges and the indolent grey slough which characterises the initial sore.

In *syphilis* the glands nearest to the sore are early affected, and later those throughout the whole body undergo changes which render them hard and prominent, forming what is known as the "bullet bubo." They rarely suppurate, except when the sore or its surrounding parts are inflamed. They remain isolated, rounded, and very hard, or they may be elongated and moderately firm—the amygdaloid bubo—the change being due to effusion of plastic material confined to the gland, and accurately circumscribed by its capsule, so that the surrounding cellular tissue is not affected. General glandular enlargement is very frequent in the secondary stage of syphilis, but possesses no distinctive histological characters. The capsule and tissue of the hilum are infiltrated with cells, and leucocytes accumulate in the follicles and alveoli. (*See* pages 388 and 398.)

Chronic lymphadenitis often occurs in the neck or axilla as a sequela of the specific fevers, and the source from which absorption has taken place may be impossible to detect. Where the glands of the neck are thus affected after scarlet fever, it may be presumed that the throat or tonsils are the site of primary irritation, or, in later stages, the external or internal parts of the auditory apparatus. The glands, once enlarged, become prominent, but are not painful, though they diminish in size or increase in accordance with the general health of the patient. The same condition may be set up by the existence of a carious tooth. The condition is characterised by an increase of bulk, causing the gland or glands to feel firm and dense and to attain a considerable size. It is due to a hyperplasia of the fibrous elements of the capsule and septa, and a consequent diminution and compression of the lymphoid tissue. In the diagnosis of these swellings it must be remembered that the border line between inflammatory hypertrophies and other morbid conditions of the glands is clinically extremely narrow.

The **treatment** of adenitis is generally simple and satisfactory. The axilla and groin are the parts in which inflammation of the glands is most frequent, and the source from which absorption has taken place can generally be discovered by searching the regions from which the absorbents converge. The glands of the *neck*—receiving as they do the lymphatics from so many parts in which irritation may arise—are very frequently the site both of chronic and acute inflammation. The mucous surfaces of the posterior nares, the tonsils, and pharynx are amongst the deeper parts from which absorption of irritant and septic materials are particularly liable to cause enlargement of the glands of this region. In the case of glands in the upper part of the neck, the scalp, the ear, and external meatus must be carefully searched for any source of irritation. The alveoli of the jaws must be inspected for any carious teeth or periosteal inflammation in the case of glands enlarging beneath the lower jaw and in the lower part of the neck, though the source of origin may be obscure; the glands often become acutely inflamed, and in this situation, owing to their confinement beneath the deep fascia, any collection of pus is deep and extensive, and gives rise to considerable pain and swelling, with redness and œdema of the skin, before it makes its way towards the surface. In the pelvic region enlargement and suppuration of glands frequently occur, as the result of products absorbed from an inflamed uterus.

In all cases, the first efforts of the surgeon must be directed to the treatment of the primary focus, if it can be detected; the part must be cleansed and, if necessary, scraped, and lotions of perchloride of mercury, carbolic acid, or other antiseptic applied. Before suppuration has taken place, the pain and swelling of the glands will be greatly relieved by warm lead and opium lotions, or by hot boracic fomentations, and in the less acute forms by the application of glycerine of belladonna. When suppuration is manifestly present, no time should be lost in giving exit to the pus, even if this involves a somewhat deep dissection. The surface of the gland must be exposed, the capsule incised, and a pair of dressing forceps inserted and opened. If the substance of the gland be much destroyed, all necrosed tissue should be removed with a sharp spoon, and a drainage-tube inserted for two or three days. The parts must be thoroughly syringed with an antiseptic solution and dusted with iodoform powder or scrubbed with iodoform emulsion. If only one gland has suppurated, those in its neighbourhood will subside. In the case of chronic enlargement, the removal of the gland may be necessary, on account of its unsightliness or its tendency to recurrent inflammations. This involves a careful dissection, and must be performed without rupture of the capsule, the gland being cleared from its surroundings by the fingers or by blunt instruments.

Tuberculous lymphadenitis. **Pathology.**—Tuberculous glands may occur in any part of the body, but are far most frequently met with in the glands of the neck, the mediastinum and the mesentery; this frequency, as pointed out by Mr. Treves, being due to the extensive collections of adenoid tissue in the mucous membranes from which these glands derive their lymphatic supply. It occurs generally in individuals predisposed by heredity or other causes to the tuberculous diathesis, and is much more frequent in the young, though occasionally seen as *senile scrofula* in persons between seventy and eighty. The bacillus absorbed from the surface of a mucous membrane is conveyed by the lymphatics to a gland where it finds a suitable nidus and develops the tubercle, which is surrounded by an area of inflammation, and passes through all the various stages of the disease which are seen to occur in other tissues. (*See* page 340.) The condition may be confined to a single gland, or may be disseminated over many in the neighbourhood, or even throughout the body. The further changes are characterised by a fatty metamorphosis, which converts the substance of the gland into a whitish caseous material, often mingled with thick curdy pus, and contained within a thickened capsule. This pus, gradually accumulating, eventually makes its way towards the surface and involves the superjacent skin, which becomes thinner and redder till it gives way and allows the matter to be discharged. The discharge is usually thin and serous, mixed with some of the caseous material. The sinus thus formed has a characteristic appearance: its margins are surrounded by œdematous granulations, and it leads to the broken-down structure of the gland often at a considerable depth. This may be found in some instances entirely destroyed, but more often exhibits in its various loculi all the stages through which it has passed. The surrounding tissues are not infiltrated. The granulations of the gland and of the sinus are often infected with bacilli, and in scraping these abscesses all granulations must therefore be destroyed, as well as the walls containing them.

The cicatrisation of the sinuses and cavities, if not freely dealt with by the surgeon, leads to a peculiarly disfiguring and characteristic form of *scar*. The cicatrix is often adherent to the deeper parts and, therefore, depressed, or it will present ridges of skin with small processes and deep furrows.

Symptoms.—With regard to the clinical features of this affection, the enlargement is generally insidious and painless, and progresses but slowly at first. The involved glands are in the early condition freely movable, but with their increase in size the surrounding parts become involved in a periadenitis, and the gland becomes adherent. Their increase in size and the rate of implication of neighbouring glands are very irregular; but large masses of glands will often be found aggregated and in various stages of the morbid condition, and will often, by their pressure, cause serious complications, in the shape of œdema, dyspnœa, or dysphagia. When one gland has discharged or has been opened by the surgeon, the others may subside and become quiescent. Even when pus has formed, it may become inspissated, or there may remain a focus of caseous material, round which the gland shrinks, or the whole may be converted into a calcareous mass. Even after many years, however, such glands are liable to recurrent attacks of inflammation.

Treatment.—In the treatment of these cases, *local* must be combined with constitutional remedies. The source of irritation must, if possible, be ascertained, and inflamed or ulcerated surfaces must be scooped or swabbed with an antiseptic; carious teeth must be removed, and enlarged tonsils excised. In delicate patients the least decline in health induces a fresh enlargement of glands which have once been inflamed. Whenever pain or swelling occurs, warmth, as applied in hot fomentations or warm lead and opium lotions, affords greatest relief, and protection should be continued by surrounding the part with flannel or cotton-wool. Where enlargement remains in a passive state, and there is no evidence of the presence of suppuration, it may be diminished by careful rubbing with the iodide of lead ointment; but the application of tinctura iodi is to be condemned, as also the injection of anything like carbolic acid or tincture of iodine into the substance of the gland. When permanent enlargement has taken place, or the size of the swelling appears to be on the increase, and when it has not acquired very firm connection with other parts, by far the most satisfactory results are gained by dissecting out the entire gland and its capsule. Often this can be effected through a very small incision, and at the same time other glands which threaten to follow the same course can be removed through the same wound. Care must be taken to tear the gland from its surroundings, and the free use of any sharp instrument should be avoided, on account of the importance and size of the vessels in the parts where such glands are most frequently situated, and the difficulty of restraining hæmorrhage in the deep wound that is left. If possible, the gland should be removed entire, without rupture of its capsule; but if this be found impossible on account of its close connection with surrounding parts or the fluid nature of its contents, the fibrous capsule must be opened and the cavity freely cleared by means of a sharp spoon, and the interior must be wiped out with a solution of chloride of zinc (gr. 20 ad ℥j), and if necessary, a drainage-tube inserted for a day or two. But this should be avoided, if possible, and stitches must not be used unless absolutely necessary, though both may be required when the gland is situated beneath the deep fascia; and horse-hair or silkworm gut is the best material. At the same time, any neighbouring glands which can be seen should be punctured, and, if necessary, dealt with in a similar manner. When suppuration is evident, and before the skin becomes involved, it is better to make an incision directly into the gland, and to evacuate its contents thoroughly by means of the spoon, and after swabbing with chloride of zinc solution or iodoform emulsion, to drain for a short period. Others prefer to puncture with the fine point of a thermo-cautery at a bright red heat, which should be passed in several directions into the substance of the gland. After any of these operations, the parts must be kept at absolute rest; and in the case of the neck, this may render necessary the application of a stock made of guttapercha or of poroplastic felt, such as is used in cases of cervical caries. The great object to be kept in view is the avoidance of sinuses and the formation of those ugly cicatrices which are characteristic of long suppuration from this cause, and this is best secured by providing complete immobility of the parts concerned.

The *constitutional treatment* consists in careful dieting and regulation of the digestive organs. Cod-liver oil, especially during the winter months, must be given for long periods, and it may be associated with some preparation of iron or of cinchona, with a mineral acid or strychnia. Sea air, particularly that of the east coast of England, works wonders for the generality of these patients; but it must be continued for long periods and repeated for several years, while at the same time every opportunity for out-door exercise must be given where possible. (*See* page 359.)

Hypertrophy and atrophy of glands.—The lymphatic glands even in health vary greatly in size, being proportionately larger in children and of greater size in adults than in old age. They are small in thin, wiry persons, and large in individuals of soft fibre. In emaciation from any cause the glands waste, but the transition between indolent enlargement, due to a condition of the tissues from which the lymphatic vessels arise and a morbid change, is often imperceptible. In cases of prolonged suppuration amyloid degeneration is not infrequent, and commences with a hyaline thickening of the reticular trabeculæ, and as this increases the lymphoid corpuscles diminish. The follicles of the lymphatics are often stained by the retention of foreign matters conveyed to them through the lymphatics. This is most frequently observed in the bronchial glands through the deposit of matters inhaled with the air, and varies with the occupation of the patient. If the substance be large in amount, it leads to shrinking of the gland, through increase of fibrous tissue and proportionate discoloration of the reticulum.

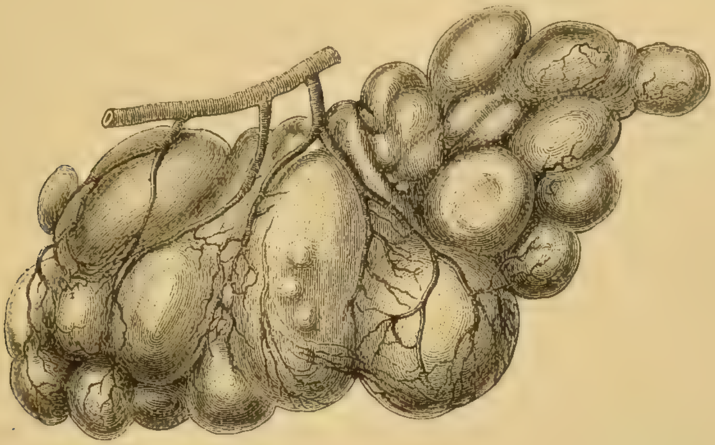

Fig. 189.—Mass of Lymphadenomatous Glands from the Axilla. (Lancereaux.)

The disease first described by Dr. Hodgkin under the title of Lymphadenoma as a painless hypertrophy of lymphatic glands, more commonly occurring in the neck, but often in other and sometimes in many regions, may be to a certain extent subdivided; but the characteristics first pointed out by Dr. Hodgkin are more or less common to all forms. The enlarged glands increase gradually, though intermittently and without pain. They never suppurate. At first the glands are separate and distinct, but later they may become confluent, and form large lobulated masses, and may be followed by lymphatic growths in various organs, particularly the spleen. Three conditions may be described.

(1) **Lymphoma.**—In simple lymphoma, unassociated with any diathesis, a gland usually in the neck, but often in the groin or axilla, or other parts of the body, enlarges gradually and painlessly, without signs of inflammation. Sometimes this may appear to arise as the result of irritation; but on removal of the cause the increase of size continues. The health is not affected, though one or two neighbouring glands may be implicated, and may unite to form a lobulated tumour.

The surface is smooth and elastic and painless, and they contract no adhesions. On section, they exhibit a hyperplasia of lymphoid tissue, in which the trabecular structure disappears, and is replaced by lymphoid cells, which are identical with the white corpuscles of the blood; but their proportion varies considerably with the rate of growth of the tumour. Under *treatment* by arsenic, iron, phosphorus, and especially sea air, the enlarged glands will often subside or remain quiescent; but the enlargement may continue and the glands fuse together, and cause danger, and even death, by pressure upon important structures. There is little tendency to degenerative changes. When single, removal at an early stage may be attempted. There is no tendency to recurrence.

(2) **Lymphadenoma.**—Lymphadenoma is a more extensive development of a like condition, and may or may not be accompanied by leukæmia. It is a disease of early life, and more common in males than females. Beginning usually in the neck, a whole chain of glands becomes involved, and later the adenoid tissue throughout the body becomes implicated (Fig. 189). The spleen is most frequently affected, but the liver, kidneys, intestines, and many other tissues, including the medulla of bones, may share the condition. When cut across, the glands present a uniform grey or yellowish colour. Microscopically, the trabecular structure is found to have largely disappeared, and to be replaced by lymphoid cells. In some specimens the reticular and fibrous parts of the glands preponderate, to the exclusion of the lymphoid tissue, and thus give a greater firmness and consistence to the tumour. In the viscera the lymph corpuscles are seen in enormous numbers in the sheaths of lymphoid tissue, which naturally enclose the vessels, and their encroachment leads to an atrophy of the natural structures. Death, when leukæmia is not present, occurs either as a result of pressure upon important structures or from diarrhœa or dropsy. When leukæmia accompanies this affection of the glands, the enlargement of the spleen is very marked, and the blood shows, on examination, a considerable increase in the proportion of white corpuscles, and a diminution in the number of red. Anæmia and exhaustion set in early, and epistaxis or subcutaneous hæmorrhages are frequent, and death occurs early from debility.

In the matter of *treatment*, operation is of little or no avail, and reliance must be placed upon iron, arsenic, iodide of potassium, and change of air.

(3) **Lymphosarcoma.**—Sarcoma, usually of the round-celled variety, occurs rarely in single glands, or in a group of neighbouring glands, as a primary affection, and quickly invades the surrounding tissue (page 466). Clinically, it is to be distinguished from the simpler forms only by its more rapid growth, and by its tendency to invade the neighbouring parts and to protrude through the skin, and by the absence of leukæmia. Very early removal may sometimes be warranted, but the progress is generally rapid and the prospect of cure is hopeless.

Elephantiasis Arabum, lymph scrotum. **Ætiology.**—In certain countries, particularly the West Indies, the Brazils, China, India, and Australia, the lymphatics form the habitat of the embryos of a mature nematode worm—the filaria sanguinis hominis (*see* page 331)—and, by setting up inflammation, or otherwise blocking the circulation, give rise to various diseases. The parasite is introduced into the human body either by absorption from the surface through bathing, or by drinking water in which the embryos, freed from the dead bodies of mosquitoes, which act as intermediary hosts, have fallen. The female, which has been found in the lymphatics, is a fine thread-like worm of white colour, from $3\frac{1}{2}$ inches long and $\frac{1}{100}$ inch wide. It is viviparous, the embryos measuring $\frac{1}{75}$ to $\frac{1}{3500}$ of an inch, enclosed in a delicate sheath, and when freshly removed exhibit lively eel-like movements, and when searched for at night about 10 to 100 can be counted on a slide. (*See* Fig. 59.) If looked for during the daytime, few or none can be detected, the cause of their disappearance being a matter of dispute. Dr. Manson's view as to the causation of stasis in the lymphatics is that the parent filariæ occupy the lymphatics; and if the lymph channel be free, the embryo will be carried with the blood without necessarily causing disease. But if the lymph channel be obstructed, the embryo will be confined to the distal branches of the occluded lymphatic. He further believes that aborted ova escaping from the female, being too large to pass through the lymph glands, act as emboli, and cause stasis of lymph, this condition spreading backwards until the whole of the glands connected with the vessel into which the female ejects her ova are obstructed. Dr. Stephen Mackenzie, who has minutely studied the subject, suggests that the presence of the parent worm alone may be sufficient to cause inflammation and obstruction, just as any condition which causes mechanical obstruction of the lymphatics may produce, as it does, all those conditions which are found to co-exist with the presence of filariæ.

Symptoms.—The leading clinical phenomena are (1) *Chyluria*, in which the urine is noticed to have a heavy milky appearance, due to the admixture of chyle, and sometimes of blood, and emits a heavy urinous odour. After standing, it coagulates into a reddish jelly, which, on examination by the microscope, shows quantities of minute granules of fatty matter, together with crystals of urinary salts, epithelium, and embryo filariæ in a more or less moribund condition. This admixture of chyle is due to a distended or varicose condition of the lymphatics, brought about by the presence of these parasites, and leading to exudation of chyle, or to a fistulous communication between the lacteals and lymphatics in the lumbar region, the pelvis of the kidney, the ureter, or bladder. Similar communications and results may be produced by the pressure of tumours on the thoracic duct or lacteals, when the urine presents similar appearances, but without the presence of filariæ. The peritoneum and the tunica vaginalis are the seats of chylous effusions, due to a similar cause (fatty or chylous hydrocele). Varicose groin glands is the name given by Dr. Manson to a condition in which both groins are occupied by large obscurely lobulated swellings, which to the touch feel boggy, doughy, semi-fluctuating, with here and there a firmer kernel-like lump, from which a needle will extract chylous or lymphous fluid. These are due to filarial obstruction of the thoracic duct.

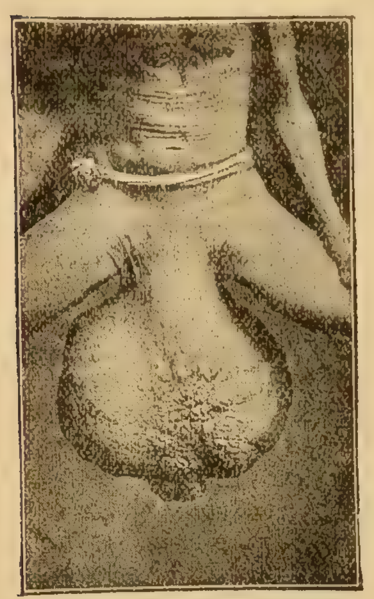
Fig. 190.—Lymph Scrotum. (Manson.)

(2) *Lymph scrotum*—elephantiasis scroti — results from the plugging of the upper group of the superficial inguinal glands if anastomosis is not established. It attacks Europeans, but is much more frequent among native races. The condition commences by an attack of inflammation, accompanied with *elephantoid fever* (Fayrer), which differs from the fever of malaria in the irregularity of the attacks, and in the greater length both of the febrile paroxysm and of the intervals. This is followed by a discharge of lymphous or chylous fluid, which recurs at intervals. The scrotum is red and enlarged, and its surface covered with herpes-like vesicles, from which a milky or sanguinolent fluid escapes, and in which almost invariably living filariæ are to be detected. These conditions continuing, the skin of the scrotum and penis becomes rough and coarse, and gradually the parts attain enormous size, until the scrotum reaches a weight of 10, 20, or 30 lbs., whilst masses weighing 110, 140, and even 224 lbs. have been recorded. The tumour is pear-shaped, with its narrowest part at the pubic and perineal attachments. The testes are dragged down and are found at the back part, considerably below the middle of the tumour. The penis is to be distinguished at the upper part of the neck of the mass at the extremity of a tunnel formed by the hypertrophied skin of the penis and prepuce (Fig. 190). The weight of the tumour draws down the skin of the abdomen and alters its form. The discharge from the varicose lymphatics

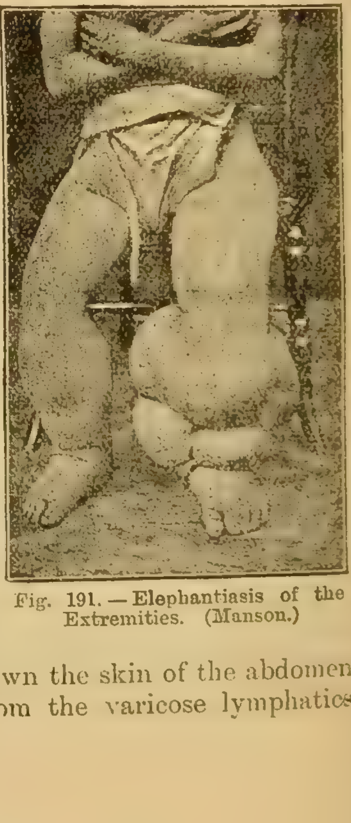
Fig. 191.—Elephantiasis of the Extremities. (Manson.)

forms crusts upon the surface, and sores and ulcerations are numerous where these are removed. A similar condition affects the external genitals of females.

(3) Elephantiasis Arabum sometimes affects the upper, but much more frequently the lower, *extremities.* It commences with attacks of lymphangitis, accompanied with elephantoid fever, of which relapses take place at irregular intervals. Each attack leaves the part more enlarged, until the ankle measures as much as 24 inches, or, on an average of 340 cases, 11 inches in circumference. The hypertrophy exists principally in the skin and subcutaneous tissue, the fascia, and periosteum; the muscles are wasted, the lymphatic trunks much enlarged and distended, the glands swollen and dense (Fig. 191). Both in the scrotum and in the leg the morbid tissues are seen to consist of two parts: "the hypertrophied layer of the skin and more superficial layers of the subcutaneous tissues, and an inner layer of loose blubber-like, dropsical, yellowish connective tissue—the hypertrophied subcutaneous fascia." The epidermis is greatly thickened in some places, hardly affected in others. The blood-vessels are very numerous and of large size, the lymphatic channels fairly numerous and of considerable diameter.

Treatment.—In the treatment of both these conditions, drugs, except quinine in case of malarial complications, are of no service. Lymphangitis must be treated on general principles. In elephantiasis of the leg, Martin's bandage is successful in the prevention of hypertrophy. Chyluria may be modified by rest and careful dieting. Removal of the enlarged scrotum is justifiable on account of the deformity and inconvenience, the sexual disability, and the impairment of general health which ensues from the recurrent attacks of pyrexia, which are undoubtedly diminished by removal of the tumour. In undertaking the operation, the tumour must, by elevation and bandaging, be emptied as far as possible of blood, and the restraint of hæmorrhage during the operation is best effected by means of an elastic cord which embraces the neck of the tumour. All diseased tissue, both of the penis and scrotum, must be removed, the testes must be secured in position, and every precaution taken to keep the wound aseptic. The successful results which follow this apparently formidable undertaking are remarkable.*

Secondary implication of glands.—*Sarcoma* rarely, but *carcinoma* very generally, implicates the lymphatic glands, the frequency very largely depending upon the site and nature of the original tumour. In cancer, the affection of the gland differs in no respect from that of the primary growth, and the series of changes which take place are of a similar character, and through the lymphatic system the growth may be disseminated throughout the body. The cancer cells are conveyed by the lymphatics to the gland and germinate in the sinuses, and the stroma of the tumour is developed from the lymphadenoid tissue. (*See* pages 480 and 485.)

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* The fullest and most complete account of these diseases is to be found in Dr. Patrick Manson's article in Davidson's "Hygiene and Diseases of Warm Climates," to which I am largely indebted for this brief account and for the use here of Figs. 190 and 191.

Congenital defects of the lymphatics.—Certain congenital defects of the lymphatics give rise to tumours which, though not noticed at birth, make their appearance soon after. Foremost amongst these is **congenital cystic hygroma,** which occurs most frequently in the neck (hydrocele of the neck), in the axilla, on the back, and occasionally in the region of the kidney. It is very rarely, but sometimes, seen in the limbs. It consists of an aggregation of numerous cysts, which spring, in the neck, from beneath the deep cervical fascia, and presents on either side of the sterno-mastoid at any point between the clavicle and the mastoid process. (*See* Art. XLIII., on INJURIES AND DISEASES OF THE NECK, Vol. II.) When occurring beneath the lower jaw, they constitute a form of ranula. (*See* Art. XLV., on AFFECTIONS OF THE MOUTH, PALATE, TONGUE, AND TONSIL, Vol. II.) (The connection of these cysts with the branchial clefts is alluded to on page 490.) In size the cysts vary from that of a pea to that of an orange, or even larger, and their increase is capricious, the whole mass often attaining very large proportions. They distend the superficial skin so that their shape, and even contents, may be seen on the surface. Prolongations often extend down into the thorax, and any increase in this direction may produce fatal effects from pressure. On dissection they are found to be composed of innumerable cysts, formed by the dilatation of lymph spaces, with walls formed by areolar tissue, and lined by a single layer of endothelial cells. The size and contents of the component cysts vary greatly both as to

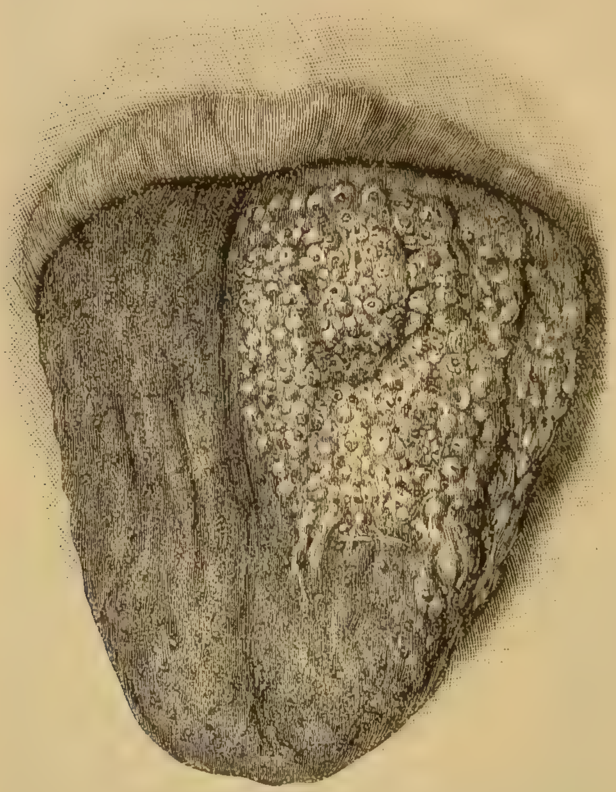

Fig. 192.—Congenital Dilatation of the Lymphatics of the Tongue. Lymphangeioma cavernosum. (From a patient in the London Hospital.)

colour and consistence, the walls of some being thin and containing clear serum, others being surrounded by hypertrophied connective tissue, and containing dark blood-stained serum, in which masses of lymph coagula lie free. The lymphatic glands are not enlarged, but the vessels and nerves are surrounded by the tumour. From fatty tumours they may be distinguished by their congenital origin, and from large venous nævi by their not diminishing under compression. Occasionally, single cysts of a similar character arise in adults, and are most usually met with in the neck or axilla.

When single, these cysts may be *treated* successfully by tapping, and subsequent injection of tinctura iodi or Morton's fluid; but when multiple, their treatment is made difficult by the proneness of this tissue to acute and extensive inflammation, which is often alarming in its extent and severity. The introduction of setons has, notwithstanding, been satisfactory in many instances. On the whole, the ablation of the entire or major portion of the tumour is to be advised; and although even under antiseptic precautions considerable inflammation of the neighbouring parts will ensue, the result after a while is satisfactory, the parts not removed shrinking and the skin contracting over them.

Macroglossia is due to a congenital defect in the lymphatic vessels of the tongue, which does not, however, always show its results at birth, but which leads to hypertrophy of the tongue and of all its component tissues. (*See* Art. XLV., on AFFECTIONS OF THE MOUTH, PALATE, TONGUE, AND TONSIL, Vol. II.) Other names have been given to the condition, such as lingua vitulina or propendula; but the pathological condition is expressed by the term given by Virchow—"lymphangeioma cavernosum." As a result of the pressure of the lower incisors in the act of sucking, or following a bite during a fit (these patients are frequently epileptic), or from some slight injury, the tongue becomes inflamed, and the consequent enlargement does not subside, and recurrent attacks of inflammation leave it still further increased in size, until it attains such proportions that it may measure 6½ inches along the dorsum beyond the upper lip, and 10 inches in circumference at its base. As a result of exposure, the surface becomes dry, brown, and is covered with fissures and small ulcerations; the papillæ are enormously swollen and covered with a crust of epithelial *débris;* the veins on the under surface are large and tortuous and unevenly dilated. The muscular tissue is not increased, but the fibrous elements are dense and firm, and are infiltrated with leucocytes, while the lymphatics are dilated and varicose.

In some cases the hypertrophy of the tongue is not so marked, but the surface of the organ is covered over with minute translucent or semi-opaque blebs or vesicles. These, if pricked, give issue to a clear fluid. Fig. 192 shows this condition. The patient was a man of twenty-five, who had been the subject of the condition described since boyhood. There was often a copious discharge of lymph from the surface of the tongue, which was often the seat of a painful but superficial inflammation.

Compression by means of isinglass plaster, beneath which are strips of calico steeped in alum or tannin, has in some instances effected a cure, but is difficult of application. The best result is obtained by the removal with scissors of a V-shaped portion from the extremity of the tongue, and uniting the parts thus separated with deep sutures of silver wire or silkworm gut. These should be removed before suppuration occurs. The bleeding is seldom troublesome and can be easily restrained by ligatures.

Macrocheilia, or congenital hypertrophy of the lip, is due to a similar cause. The lip and cheek are greatly enlarged and continue to increase with the growth of the patient. The swelling is firm and is not compressible, but its resemblance to a nævoid swelling is very intimate in consequence of the veins partaking of the varicose condition which affects the lymphatic tissues. Excision of the whole or of a large portion of the mass is the only remedy and can be performed without much hæmorrhage. (*See* Art. XLV., on AFFECTIONS OF THE MOUTH, PALATE, AND TONGUE, Vol. II.)

XXVII. INJURIES AND DISEASES OF NERVES.

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PART I. INJURIES.

I. THE SECTION OF A NERVE.

The effects of section of a nerve are much greater in proportion to the size of the part injured than are the effects of injury to most of the other tissues of the body. Not only do changes ensue in the divided nerve itself, but the various parts to which it is distributed also undergo structural and functional alterations, which, in turn, give rise to the most diverse pathological conditions. It is necessary, therefore, before describing the clinical symptoms and the treatment of nerve injury, to consider in detail the results of section of a mixed nerve.

1. **Changes in the nerve itself.**—After section, the whole of the peripheral portion undergoes degenerative changes in its entire length and distribution, and it has been experimentally demonstrated that this degeneration is due to the separation of the motor fibres from the large nerve cells in the anterior cornua of the spinal cord, and of the sensory fibres from the similar cells in the ganglion on the posterior root, these ganglion cells constituting the trophic centres for the fibres which originate in connection with them. The first and most evident alteration in the degenerating nerve is seen in the white substance of Schwann, which becomes broken up and forms globules of varying size within its sheath. This change is seen within the first four or five days, and the disintegration thus early commenced continues with such rapidity that in the course of four or five weeks the whole of the myelin has been destroyed. The degenerative changes are not, however, limited to the myelin; they affect also the axis cylinders, which split longitudinally into numerous fibrils, and completely disintegrate even more rapidly than does the myelin itself. In this way the whole of the axis cylinders and their white myelin sheath are completely destroyed, and the peripheral end of the severed nerve after a few weeks consists only of

the perineurium and the endoneurium, with the sheath of Schwann . structures which are chiefly fibrous and incapable of conducting nervous impulses. A nerve which has undergone these changes is shrunk to about half its natural size, and is not so white and translucent in appearance as is a healthy nerve. The changes which occur in the part of the nerve above the seat of section are of much less extent, being practically limited to the part immediately above it. Here there is formed, within three or four weeks, an oval swelling about twice the size of the nerve itself, and commonly spoken of as the "bulb." It is much harder than the nerve above it and, on section, looks dense and fibrous. Under the microscope it is seen to consist of numerous small nerve-fibres, which are probably of new formation, embedded in fibrous tissue. No other alteration occurs in that part of the nerve which is between the bulb and the spinal cord, with the exception of some slow atrophic change, which causes, after some years, a general shrinkage of the whole trunk, and only ensues when from some cause union of the severed nerve has not taken place.

2. **Trophic and degenerative lesions.**—It has been already mentioned that the parts which are cut off from their nerve supply undergo certain changes, and it is necessary to describe these in some detail, as a proper appreciation of them not only explains many of the symptoms of nerve injuries, but also supplies information as to their proper treatment.

The *muscles* supplied by the severed nerve are at once paralysed, and within a few days begin to show evidence of atrophy and of degeneration. The atrophy is much more than can be accounted for by simple want of use and is infinitely more rapid and complete. It is accompanied by degenerative changes in the muscle fibres themselves, which result in the development of fat granules in the sarcolemma and the disappearance of the muscular fibres. Within a few months the muscular tissue is practically destroyed; and not only does the affected muscle diminish in bulk, it also becomes shrunken in length and may, by its shrinking, cause interference with the movements of neighbouring joints. A muscle so altered is much paler than natural and much more fibrous in appearance.

The *skin* is liable to be altered in various ways. It usually becomes smoother than is natural and occasionally, in addition, shiny and glistening, the papillæ appearing to be smoothed out. Eruptions of eczema, herpes, or acne may result and occasionally ulcers appear in the anæsthetic area. These are frequently due to injury of some kind or another, which, on account of its painlessness, has passed unnoticed by the patient. Thus, an anæsthetic finger may be burnt or cut without the knowledge of the patient, and the resulting sore is slow to heal, partly because of the deficient nerve supply, and partly also because, on account of its painlessness, the part is often insufficiently protected. More rarely nerve injuries are followed by gangrene of the pulps of the fingers or toes and occasionally of a whole digit. Painless whitlows, with superficial collections of pus, sometimes develop, and from these a slow form of ulceration occasionally extends.

The *hair* of the affected area often falls out and in other cases becomes brittle and stubbly. Microscopically examined, it is seen that all the glandular tissues of the skin are atrophied and that the papillæ are so shrunk as to be practically obliterated. A section of skin so altered is more like the section of a scar.

The *nails*, as might be expected, share in the trophic changes of the skin. They may merely become fibrous and brittle, or may grow so as to be excessively curved and talon-like, and present deep grooves or raised ridges on their surfaces. In other cases they become loose and are cast off without pain.

The *subcutaneous tissue* atrophies with the skin.

The *joints* undergo changes which are apparently chiefly of an inflammatory nature, those of the fingers being most often and most characteristically affected. The process is closely allied to that of rheumatism and may commence within a few days of the nerve injury, with swelling of the affected articulation, and often with pain on movement, although the skin may be anæsthetic. There appears to be at this stage a synovitis, with exudation of plastic lymph and inflammation of the fibrous capsule. Such changes are very liable to terminate in the formation of adhesions and the development of a fibrous anchylosis. When many of the finger joints are affected, the hand may be crippled by these articular lesions to an extent that can scarcely be exaggerated.

3. **Alteration in temperature.**—For the first few days after nerve section the paralysed parts are flushed with blood, as the result of section of the vaso-motor filaments; but whilst this condition is barely noticeable and the increase in temperature only recognised by careful investigation, at a later period the paralysed parts become colder and more bloodless. The loss of temperature is very rapid and may amount to as much as eight or ten degrees, the coldness being very noticeable both to the patient and the surgeon. It is probable that this condition results from vaso-motor spasm, which ensues some two or three weeks after section and continues so long as the nerve remains ununited.

Symptoms of section of a nerve.—The two chief symptoms which indicate at once that one of the nerves of the extremities has been injured are loss of power and loss of sensation.

Loss of power.—The muscles supplied by the divided nerve are immediately paralysed and remain in this condition so long as the nerve remains ununited. Later, they rapidly waste and degenerate. This atrophy is much more rapid and complete than can be accounted for by inability to use the affected muscles, and proceeds so quickly that within three or four months but little of the muscular tissue remains. The atrophy, moreover, affects not only the bulk of the muscle, but causes it also to shrink in length and, by so shortening, to interfere with the action of other muscles and to produce deformity.

If the condition of the paralysed muscle be examined electrically, further evidence may be obtained, which, in doubtful cases, will greatly aid diagnosis. Within twenty-four hours of the nerve section Faradic irritability begins to diminish, and within four or five days is completely lost, so that even the strongest currents will produce no contraction. It is pretty certain that this loss of excitability is due to the destruction of the motor nerve fibres.

The galvanic current, which acts more directly on the muscle substance itself, does not at first appear to lose its effect, for during the two or three days succeeding the injury the galvanic irritability of the muscles seems to be actually increased. After this time, however, muscular contractions grow more feeble, and in the course of about two or three months gradually disappear. It is during this period of diminishing galvanic irritability that the "*reaction of degeneration*" becomes marked. Normally, the kathodic closure contraction of a healthy muscle is greater than the anodic closure contraction. After nerve section, when the muscle is degenerating, the anodic closure contraction first becomes equal to, and then greater than, the kathodic: a reaction which indicates degeneration of muscle and nerve, and is, therefore, named the "reaction of degeneration." It is strange that after union of a divided nerve has taken place voluntary power returns many months before the muscles show any reaction to either the galvanic or Faradic currents.

Loss of sensation.—When a nerve containing sensory fibres is divided, the patient is usually almost at once conscious of the numbness of the skin area supplied by the severed nerve. In those cases, however, where the nerve lesion is only a small part of a serious laceration of the limb, the loss of sensation often passes unnoticed, on account of the pain and shock accompanying such an accident, and thus, unless sought for, this evidence of nerve injury may remain unrecognised. In most cases complete anæsthesia of at least some of the skin results, and this is always the case when a nerve of considerable size is cut across; but in those cutaneous areas where more than one nerve is distributed, as on the ring finger, more or less sensibility may persist. Even in these cases, however, the patient is conscious that sensibility is greatly impaired, and although the sense of touch may be retained to some slight extent, sensibility to pain and to heat and cold are almost always lost. At the edge of the affected skin area the line dividing anæsthetic parts from healthy skin is usually not very sharply marked, and an area of impaired sensibility is generally to be found.

In testing the amount and extent of the loss of sensation, it is necessary to be very careful, and the possibility of exciting sensory impressions through the medium of neighbouring healthy nerves must always be kept in mind. Thus, if a finger be pressed or pushed, the patient may perceive, through his "muscular sense," that it is being touched; or if the supposed anæsthetic area is tested by the surgeon rubbing it with his finger, the vibrations conveyed to the neighbouring healthy nerves will correctly convey tactile sensations. The point of a pencil is as good as anything for testing the sense of touch and the surgeon should notice not only whether the patient can *feel*, but whether he can also correctly *localise* his sensations.

But whilst the alteration in sensation and the loss of muscular power constitute the most important immediate evidence of severe injury, at a later period the diagnosis is aided by the development of the various trophic lesions which have been already described ; and the wasted muscles, the contracted joints, the shiny and stretched skin, and the coldness of the paralysed parts, all point unmistakably to the injury that has been sustained.

Union after section of a nerve.—If the recently severed ends of a nerve be placed and maintained in apposition, they will unite. It is probable that in a few exceptional cases this union may occur without any preceding degeneration of the distal end, such as has already been described, but it is quite certain that this is not usually the case and that, even if the cut ends are maintained in contact, the lower end degenerates before regeneration and repair result. The union is ultimately brought about by a development of new axis cylinders from the nuclei of the sheath of Schwann in both the proximal and peripheral ends, and, subsequent to the formation of these, there is developed a new myelin sheath. The development of this new nerve tissue requires a great length of time, and may extend over a period of from six to twelve months before material improvement is demonstrable, and sometimes requires a year or two for its completion.

Treatment of a divided nerve. Primary suture.—In all cases of recent section of a nerve trunk the cut surfaces should be approximated by suture. On account of the elasticity of the nerves, there is liable to be much separation of the cut ends, and it is often necessary to enlarge the wound in order to bring them into view. If much tearing or bruising has been sustained, it is well to refresh the cut surfaces ; and it is of the utmost importance in such cases to take every precaution to render the wound aseptic and to prevent suppuration. Sutures should always be of some substance which is capable of undergoing absorption, because such materials as silk or horse-hair remaining in the nerve trunk are liable to keep up constant pressure on the surrounding tubules, and to cause severe pain. The best material is, probably, kangaroo tendon, but fine chromicised catgut is very efficient. Small needles should be used and the sutures should be passed right across the nerve, so as to include its sheath.

Two such sutures passed at right angles to each other, about one-eighth of an inch from the cut surfaces, will generally suffice to bring the ends into good apposition. If it appear necessary, one or two more fine sutures may be passed, so as to draw the sheath together at some place where it gapes, but no more sutures than are necessary to obtain good apposition should be employed, as they only tend to split up the cut ends. The sutures should only be tied tight enough to ensure proper approximation, for if tied very tightly they will most likely cut their way through almost at once. After suturing such tendons as may have been divided, the wound must be closed, and the limb put up in such a position as to ensure, as far as possible, complete relaxation of the wounded nerve; splints and bandages should be applied, so as to prevent any sudden stretching of the wound.

If the wound heal without suppuration, the nerve, if sutured with the precautions advised, will in time unite; but, as has already been said, the evidences of repair may not be demonstrable for many months. Union is more rapid in the young and in them is also more perfect. It is seldom that sensation and muscular power are completely recovered, and even in the best cases there is liable to be some permanent numbness, so that whilst the part involved is nowhere anæsthetic, the touch loses its delicacy. In some cases sensation may return and motion may never be regained; in others, whilst motion is restored, the skin remains numbed. In order to promote union of the divided nerve, as soon as the wound is soundly healed the paralysed muscles should be daily galvanised and massaged, and the joints should be moved to prevent the formation of adhesions. It is, further, of the utmost importance that the whole extremity affected, especially the paralysed part, should be kept warm. Cold is most harmful, and repair is in many cases apparently more rapid during the summer than in the winter months.

Secondary suture.—In many cases a surgeon does not have the opportunity of seeing a patient till some weeks have elapsed since the injury of a nerve, and if there be then evidence of division of a nerve trunk, and no attempt at primary suture has been made, it is necessary to reopen the wound and ascertain the extent of the injury. In such a case it is well to wait till a suppurating wound has healed before undertaking any operation, so as to be able to operate on aseptic tissues. The incision should be made in the long axis of the injured nerve, and should be of sufficient length to enable the latter to be exposed an inch or more above and below the seat of injury. It is much easier to find the nerve ends in this way than by simply cutting into the scar where the various injured tissues are matted so as to make it difficult to distinguish them from each other. Having thus found the injured nerve above and below the scar, it is necessary to dissect the ends from the fibrous tissue which binds them down. A fresh section of each must then be cut. In the lower end as little as possible should be removed, for no good results from cutting away any considerable length, the whole peripheral end being all equally atrophied. As regards the upper end, the section should be carried through the upper part of the "bulb"; but the whole of the latter need not be resected. If the nerve seems much shortened, the ends may be stretched before sutures are inserted. The latter should be of the same material, and passed in the same way as has been described in the account of primary suture.

Punctured wounds and partial division of nerves.—

Punctured wounds of sensory nerves are sometimes followed by severe pain and hyperæsthesia of the skin area supplied by the injured trunk. The pain is, in turn, sometimes accompanied by muscular spasm, and is made worse by movement. These conditions apparently result from some neuritis set up at the seat of injury, and should be treated by complete rest and the local application of anodynes, such as belladonna or opium. If these fail to relieve, small blisters to the hyperæsthetic parts are often successful.

In recent cases of partial division, when the patient is seen at once, it is advisable to suture the severed parts of the nerve trunk in the way already described. If, however, the patient be not seen until the wound is healed, the treatment adopted must depend on the extent of the loss of sensation and muscular power. If this be considerable, the injured nerve should be exposed, and, after separation from the surrounding scar, the extent of the injury must be carefully ascertained. If it appear that the nerve has been practically divided, the injured portion should be resected and the ends sutured ; but if it seem that the symptoms are rather due to scar pressure, then the surgeon should be content with freeing the trunk. Cases of partial section, when seen at once, generally do very well, because the part of the trunk which remains intact prevents any more separation of the cut fibres.

II. CONTUSION OF A NERVE.

A severe contusion of a nerve may be followed by all the symptoms of complete section. Slighter injuries cause a greater loss of motion than of sensation, and muscles may be quite paralysed whilst the sense of touch is but little impaired. The treatment of these cases depends on the severity of the symptoms and the amount of time that has elapsed since the injury. In all recent cases no operation should be undertaken, because a very large number of patients recover without any surgical interference. Again, if there be evidence that the nerve trunk is capable of conveying sensory impressions and that the muscles are not all paralysed, then the prognosis is that with rest, massage, and galvanism a good result will be obtained. On the other hand, if, after six or eight weeks, there be complete loss of sensation and of motor power, the injured trunk should be exposed and examined, for it may be that it has been torn across or divided by being crushed against a bone. In such cases resection of the injured part and suture of the ends would be required.

III. NERVE GRAFTING.

Cases occasionally occur in which a considerable portion of a nerve trunk has been destroyed, and in which it is evident that the separated ends are too far apart for the application of sutures. In several such instances a portion of nerve has been successfully transplanted between the severed ends, after separating the latter from the scar tissue surrounding them, and refreshing them. The nerve transplanted may either be taken from a recently killed animal, such as a rabbit, or from a freshly amputated limb. In either case it should be removed immediately before it is required, and should be placed in warm sterilised water (or in normal salt solution) pending its insertion into its new bed. Care should be taken that a sufficient length is cut to prevent any tension in suturing it in position; and, in removing the nerve from the animal or limb from which it is taken, all pinching or holding with forceps should be avoided.

IV. PRESSURE ON A NERVE.

Nerves may be compressed in the most various ways. Thus, tumours, and especially malignant ones, may involve the neighbouring nerve trunks, or may become adherent to the nerves. The brachial plexus is often thus involved in the axilla in cases of recurrent carcinoma of the breast. In other cases nerves are involved in inflammatory swellings—*e.g.* in inflamed glands of the neck—and more often are compressed by scars resulting from wounds of structures near to the nerve trunks in the upper extremity. In other cases, again, as in deep-seated pelvic or gluteal suppuration, the neighbouring nerves may be compressed by abscesses. Dislocated or fractured bones may also cause pressure. In another class of cases, nerves are compressed by external objects, such as by crutches or by some implement used in work, whilst paralysis from pressure on the nerves of the arm during heavy sleep—especially in drunken sleep—is tolerably common.

In most of these cases the pressure is gradual and its effects are only slowly developed. The first symptoms are commonly numbing and tingling pains, with sensations of "pins and needles" in the peripheral parts, and these are succeeded by a slowly developing and seldom complete anæsthesia. The evidence of pressure on the motor nerve fibres is supplied by feebleness of muscle, with some tremor, and subsequently by complete paralysis. Thus, in crutch palsy, which is due to pressure of the crutches on the axillary nerves in a patient unused to their management, there is some numbness of the hand and fore-arm, with tingling, and subsequently loss of power in the extensors of the fore-arm, with "wrist drop." These symptoms are due to the compression of the musculo-spiral nerve against the humerus, this trunk being the most easily affected, on account of its proximity to the bone. In other examples, and especially in cases where there is some neighbouring inflammation, severe pain is the chief symptom, and many cases of so-called "sciatica" prove to be due to pressure on the sciatic nerve by an abscess or a pelvic tumour. Trophic lesions are more rarely developed and are usually of late onset.

Pressure by the contraction of scars is of sufficiently frequent occurrence to merit special mention. In some cases merely the terminations of sensory nerves are so included, but the results are far more widespread than might be thought. The scar is often exquisitely tender and the least touch of it is liable to produce muscular spasm. Occasionally there result spasm and rigidity of many of the muscles of the affected limb. The implication of nerve trunks in scar tissue causes an interference with the function of the nerve proportionate to the amount of pressure exercised. Pain is not so prominent a feature as in cases of implication of sensory nerve twigs.

The treatment of pressure on a nerve is to remove the cause. Thus, tumours may be removed, crutches given up for a time, and painful scars excised. If a nerve trunk be compressed by scar tissue, it should be dissected from it and then thoroughly stretched. The paralysis and loss of sensation resulting from pressure are best treated, after removal of the cause, by galvanism and massage.

V. INJURIES OF NERVES COMPLICATING FRACTURES AND DISLOCATIONS.

In fractures.—Considering the frequency of fractures, nerves are but seldom injured at the time a bone is broken. They may, however, suffer in one of two ways. First, they may be torn at the time of the accident; or, secondly, they may be subsequently compressed by callus. In the first case the symptoms of loss of power and of sensation supervene at once; in the second case their onset is gradual. Nerves lying close to bones are those most likely to be involved, the musculo-spiral being probably more often injured than any other. In many cases the nerve injury is overlooked in the face of the more evident lesion, and the subsequent splinting and bandaging of the limb frequently prevent a patient from having any clear idea of the condition of his sense of touch and of muscular power. It is often only when splints are removed that the symptoms of nerve injury are first noticed. (*See* Art. XXIX., on INJURIES OF BONES, page 789.)

Treatment.—Considering that mere contusion or stretching of a nerve may also cause a complete, though transient, paralysis, it is evidently not advisable to undertake any operation at the time of injury; and, again, when the symptoms have only slowly developed and are apparently the result of callus pressure, time should be allowed for the provisional callus to be absorbed before concluding that surgical interference is necessary. When, however, the surgeon is convinced that no improvement is to be expected, he should expose the nerve at the seat of fracture and, if he finds it torn, should suture it. In other cases excessive callus must be cut away, and occasionally displaced fragments of comminuted bone must be excised. Sometimes paralysis of a nerve trunk seems to result from a faulty position of the bones after the fracture has been set; in such cases re-fracture and re-position of the fragments may give good results.

In dislocations.—It is only in certain dislocations that injuries of the nerves occur with any frequency, such complications being most common in the case of the shoulder, and after that in dislocations of the elbow. In each case the nerves may be either torn by the displaced bone, or may be so stretched that their fibres are lacerated.

In the dislocations of the shoulder joint, any of the cords of the brachial plexus may be torn, or the circumflex alone may be contused and stretched by the downward displacement of the humerus. Rupture of the plexus in the neck or tearing out of the roots from their insertion into the spinal cord may also result. Of these conditions, mere contusion and stretching are fortunately the most common, and in such cases it is generally evident that there is no paralysis of any one nerve, but rather a partial loss of sensation and a want of power, without absolute muscular paralysis.

In rupture of the brachial plexus, however, there is usually a very widespread paralysis; and when an opportunity of examining such cases has been afforded, it has generally been found that the roots are torn out from the spinal cord instead of the cords themselves being lacerated. The extent of the paralysis will depend on the number of nerve roots so involved, but in almost any case it is noticeable that such muscles as the supraspinatus, the rhomboids, and other scapular muscles are paralysed, which could not be affected if the lesion were in the axilla. In these cases also of tearing out of the roots, the motor nerves for the dilator fibres of the iris, which pass from the cervical cord to the sympathetic trunk, are usually involved, and thus the pupil on the affected side is smaller than its fellow and does not actively dilate in dim lights.

PART II. DISEASES.

I NEURALGIA.

Neuralgia may be shortly defined as local pain without local signs of inflammation or disease. In many cases there is no definite cause, but such general conditions as anæmia and feeble health appear to account for a large proportion of these cases. Other more definite constitutional conditions, such as gout and rheumatism, appear to act as predisposing causes in many patients, whilst malarial fevers of any kind and influenza may be cited as acquired diseases, which directly cause neuralgia in a large proportion of those who suffer from them.

Causes.—The chief *exciting causes* are: (1) exposure to cold; (2) pressure on or irritation of nerve trunks; (3) peripheral irritation; (4) irritation or disease of the central nervous system.

It appears probable that cold acts by causing inflammation of the nerve sheath, but from a surgical point of view, neuralgia caused by pressure is of more interest and importance. It has already been pointed out, in dealing with pressure upon nerves, that loss of power and of sensation may be caused thereby, but pain also may be very severe. Thus, the neuralgic pains caused by the pressure of thoracic aneurysm on the intercostal nerves may be a prominent symptom of an obscure affection, and, in the case of intra-pelvic tumours, pain in the course of individual nerves may be very severe. In any case of persistent pain in the course of one of the nerve trunks of the limbs, a careful examination should always be made to ascertain if there is any pressure cause which can be discovered.

Neuralgia caused by peripheral irritation is well illustrated in the case of facial neuralgia due to a carious tooth; and the pain felt in the testis as a result of renal calculus affords another well recognised example of the same kind. The peripheral pains due to disease of the spinal cord are unfortunately common in tabes dorsalis, and severe neuralgic pain is also of common occurrence in cases of spinal caries; in many of the latter the pain felt in the chest and stomach is much more severe than that in the back.

The pain of neuralgia.—The pain of neuralgia is localised, more or less accurately, to the area supplied by a certain nerve or nerves. It is usually of a darting or shooting character, and radiates from a central point. Pressure upon the spot where the affected nerve emerges from a bony canal or from beneath the fascia frequently intensifies the pain.

Epileptiform neuralgia is a very severe variety of this affection. It almost invariably attacks the face, and may follow the course of any one or of all the branches of the fifth pair of nerves. Two varieties are described. In the one the pain is accompanied by spasm, and twitching of the facial muscles; in the other there is no accompanying spasm. This form of neuralgia does not appear to be associated with any condition of ill-health, and occurs in perfectly healthy people of all ages. Fortunately, it is a rare affection. The attacks are intermittent, sometimes lasting for only a few days. They may extend over many weeks or months, and the intervals between the attacks may be weeks, months, or even years. The paroxysms of pain have usually a very sudden onset. Their duration varies from a few seconds to half a minute, seldom exceeding the latter limit. They may be repeated so rapidly that the intervals are scarcely appreciable, or several minutes may elapse between the paroxysms. A paroxysm may be started by any stimulus applied to the branches of the fifth nerve. Thus, a draught of cold air, an attempt to swallow liquid or to masticate food, the slightest touch of the hair of the face, or an attempt to speak, may initiate an attack of pain. This, commencing usually at a single spot, quickly radiates over the neighbouring skin and mucous membrane, and in some cases spreads over the whole face. The mucous membrane of the lips, gums, and nostrils is just as much the seat of pain as is the skin, and in some instances the secretions are altered, the nostrils becoming hot and dry, or the eyes filled with tears. The pain varies in character as well as in intensity, patients usually describing it as burning hot, or as if the flesh were being crushed and torn.

During the paroxysm the patient usually clasps his face in his hands, and seems to obtain some relief by the compression of the seat of pain. On account of the frequency with which movements of the mouth cause an attack, all movements of the facial muscles are avoided as much as possible. Thus, in speaking the teeth are kept clenched and the lips are hardly moved ; and in eating or drinking the same precautions are exercised. On account of the sensitiveness of the skin, the face is commonly wrapped up and bright light is avoided. In some cases of epileptiform neuralgia the general health gives way under the strain of constant pain, but in others it is surprisingly little affected.

Treatment of neuralgia.—In many cases of neuralgia, where no definite cause can be discovered, much more good is done by paying attention to the general health than by giving drugs to relieve pain. In the neuralgias which are so common in anæmic women, it will often be found that dyspepsia and insufficient nourishing food are at the bottom of the trouble, and attention should in such cases be always directed to the remedying of these conditions. In other cases, insufficient healthy exercise, working in overheated and ill-ventilated rooms, and bad hygienic surroundings appear to account for the frequent neuralgic attacks. In those cases where neuralgia supervenes upon malarial fever or influenza, and in many others where it is the sequel of some prolonged illness, temporary residence in a dry bracing climate—such as the mountain resorts of Switzerland—will bring about a cure when all other remedies have failed. The treatment of neuralgia by drugs can scarcely be discussed in the present work, but it may be stated that in many cases arsenic is an excellent remedy, and that in cases where gout appears to be the cause of the malady the usual remedies for this disease may be very useful.

But whilst the vast majority of cases of neuralgia may be successfully combated by some such means as have been mentioned above, it must be admitted that where a local cause exists local measures are alone, as a rule, successful. Thus, the removal of carious teeth will often suffice to terminate an attack and to prevent others ; and if any tumour or inflammatory swelling is found to exercise pressure on a nerve, the removal of such a cause is evidently indicated. With regard to the extraction of teeth, however, a word of warning is necessary, for the pain is often referred to teeth which are quite sound, and the removal of these, however much the patient may desire it, is to be strenuously resisted. For such treatment, indeed, is of no avail and, on the other hand, is often most harmful, the gums being left tender and sore, and the patient having further trouble from insufficient powers of mastication and consequent dyspepsia. In neuralgias of the extremities there is more often some local cause than in those of the face, and in this class of case, where no local cause can be discovered, reliance must be placed rather upon local treatment than in the use of drugs. Counter-irritation by iodine or by blistering fluids is sometimes very efficacious, whilst in others, massage gives very satisfactory results. Galvanism is also of use in some cases, especially in inveterate sciatica. It should be used for fifteen to twenty minutes daily and should be persevered in for several weeks.

Operations on the nerves for the cure of neuralgia are only very exceptionally indicated. Where a nerve twig is caught in a scar, or where a trunk is compressed by inflammatory effusion, it is evident that an operation should be undertaken to free the nerve from its surroundings. But such cases form only a very small percentage of the total and cannot really be compared with the more common neuralgias which own no such exciting cause. There are, however, a certain number of cases of intractable facial neuralgia, where operations on the affected nerve trunks may be undertaken with much temporary benefit; and in no class is operation more called for than in the so-called epileptiform neuralgia. It has already been said that in many of these cases any slight peripheral irritation may excite an attack, and the object of an operation is to prevent the transmission of any stimuli along the trunk or branches of the affected nerve.

It is not to be supposed that any operation on nerve trunks for the cure of neuralgia is based upon the supposition that the nerves are the seat of disease. There is no reason to believe that this is the case, but it appears probable that when relief is obtained it is really due to the rest secured to an over-stimulated nerve centre, and the operation may be considered to act by procuring physiological rest to the sensorium. It will, therefore, be evident that such operative treatment does not aim at eradicating the disease by the removal of any diseased structure; and this should clearly be kept in mind in considering the advisability of operative interference, and in the choice of the operation itself. It must further be explained to the patient that operations of neurotomy, or nerve-stretching, cannot be relied upon to bring about a cure, but that, while they do in some cases apparently bring permanent relief, in many others the freedom from pain is merely temporary.

Nerve-stretching.—Before considering the operation of nerve-stretching, it is necessary to consider very briefly the *strength and elasticity* of the nerves. It has been estimated, as a result of experiment, that a nerve is capable of being stretched about one-twentieth of its whole length, and in general terms it may be stated that the elasticity of different nerves is, for all practical purposes, equal. The strength of a nerve depends necessarily on its size, and the larger trunks are capable of resisting very considerable force. The breaking strain of the sciatic nerve varies from about one hundred to one hundred and sixty pounds, while such nerves as the median, musculo-spiral, and ulnar will support from about fifty to eighty pounds' weight. The small nerves, such as the branches of the fifth, of course offer but little resistance, and break with a strain of from five to ten pounds.

Anatomical changes.—The anatomical changes which follow nerve-stretching are the result of laceration of the various structures included in the nerve sheath, and may be briefly summarised.

(1) The sheath is partly loosened from its attachment to the nerve, and at the same time is narrowed, and so tends to constrict the tubules.

(2) The blood-vessels of the sheath are torn, and ecchymoses occur. The vessels in the substance of the nerve become tortuous and dilated immediately after the stretching, and the nerve trunk becomes subsequently more vascular, by reason of the formation of new blood-vessels.

(3) There is an increase of connective tissue cells in consequence of the injury inflicted.

(4) The nerve tubules are torn in proportion to the violence used. The axis cylinder breaks less easily than does the myelin sheath and the sheath of Schwann.

(5) In proportion to the amount of injury there ensues degeneration of some of the nerve fibres, such as has been described as resulting from section of a nerve.

(6) In the process of time new nerve fibres are formed and the nerve is completely regenerated.

The **physiological effects of nerve-stretching** also depend on the amount of force employed and the amount of injury inflicted. It appears that very slight stretching increases the irritability of the nerve, and results in slight hyperæsthesia, muscular irritability, and flushing of the skin area involved; but it is certain that in all cases where a nerve is voluntarily stretched by the surgeon, the conductivity of the nerve is materially diminished, and that numbness or complete anæsthesia, and diminution or loss of muscular power, ensue in most cases. When a great deal of force has been employed, the operation may be followed by wasting of muscle and by trophic changes.

The **operation.**—The operation practically consists of exposing the nerve by a suitable incision, and subsequently isolating and stretching it. If the nerve trunk be a large one, such as the sciatic, the finger, or two or three fingers, should be passed beneath it, and steady traction should be exercised, first in one direction and then in the other. The amount of force employed must vary with the size and strength of the nerve; even for the sciatic it should not exceed thirty or forty pounds. When the nerve is small and the wound narrow, as in operations on the face, the nerve is best lifted up and stretched by passing an aneurysm needle beneath it. The stretching should be continued for several minutes.

The so-called "bloodless stretching" of a nerve can practically only be performed on the sciatic trunk. In this procedure the thigh is flexed on the abdomen, whilst the leg is kept extended on the thigh, and thus the sciatic nerve—together with all the tissues around it—is put on the stretch. The extension should be maintained for ten or fifteen minutes under an anæsthetic, and at the same time vigorous kneading and massage may be employed along the course of the nerve. It must be remembered that if the patient be old and the arteries atheromatous, this treatment is liable to put a dangerous strain on the popliteal artery.

Clinical application of nerve-stretching.—When nerve-stretching was first introduced, it was employed in many cases of disease of the central nervous system, but it may be said with confidence that in such cases it is certainly of no material advantage in promoting a cure, although it may improve symptoms. Its utility is best demonstrated in cases of neuralgia and in certain nerve lesions. Thus, in cases of inflammatory thickening, either of a nerve itself or of the tissues around it, the neuralgic pain caused by the pressure is often greatly relieved by exposing the nerve at the affected area, separating it from the tissue around it, and thoroughly stretching it. It may also be employed in cases of spasm—*e.g.* in facial spasm—and to alleviate hyperæsthesia, from whatever cause arising. In a few cases of tabes dorsalis the operation appears to have relieved the "lightning" and "boring" pains, and in some cases other symptoms are said to have been improved.

In certain cases of facial neuralgia nerve-stretching appears to act very beneficially, and is preferred by some surgeons to neurotomy or neurectomy. The way in which nerve-stretching causes improvement is probably not always the same. It is evident that when a nerve trunk is more or less compressed by scar tissue or fixed by adhesions, the mechanical separation of the nerve is likely to be beneficial; and the many recorded cases of improvement in cases of pressure by cicatrices, of neuralgic pains following injuries, etc., are in this way most readily explained. The action of nerve-stretching in chronic neuritis is probably of a similar nature. It has also been suggested that the operation may act by freeing the nervi nervorum from surrounding adhesions. In many other cases, however, especially in neuralgia, any good results that may ensue are probably due to rupture or injury to the nerve tubules themselves, and to the resulting impairment in the conductivity of the nerve. It is probable that in nerve-stretching we tear the tubules not only of the trunk we operate upon, but of other branches as well, which are difficult—perhaps impossible—to reach with a knife. Thus in stretching the infra-orbital nerve, it is probable that the branch passing downwards in the floor of the orbit to the teeth is also stretched, and in operating on the inferior dental, the branches to the individual teeth are influenced. Thus, the effects of nerve-stretching may be more widely spread than those of neurotomy, and in dealing with the mixed nerves of the extremities it is evident that such an operation is often preferable to division, considering that the latter will cause not only anæsthesia, but permanent muscular paralysis as well.

Neurotomy and neurectomy.—The operations of cutting down upon a nerve and dividing it, and of cutting out a portion of the trunk, are, like the similar operations for exposing arteries, performed at certain "seats of election," where the nerves can be most easily reached, and with the least possible injury to the surrounding parts. In all such operations on the limbs, the use of an Esmarch's bandage greatly facilitates matters, and enables the surgeon to find the nerve more readily, and to ascertain its condition with much greater certainty.

Nerves of the head and neck. *The supra-orbital nerve and the supratrochlear nerve.*—These are best exposed by an incision carried parallel with the eye-brow and just beneath it, so that the line of the incision is hidden by the folds of skin in this situation. The supra-orbital notch can be easily felt in most cases, and the nerve will be found emerging from it, and turning directly upwards after the incision has been carried through the skin and some of the fibres of the orbicularis muscle. The supratrochlear nerve lies nearly half an inch to the inner side of the supra-orbital notch, and may be exposed by a similar incision.

The infra-orbital nerve.—The infra-orbital canal is in a line drawn from the supra-orbital notch to the canine tooth of the same side. The nerve is exposed by an incision carried across this line, about an inch below the lower margin of the orbit. After dividing the skin and some of the facial muscles—chiefly the zygomatici—the orifice of the canal can usually be felt with a probe-pointed director. While this is held with its point in the canal, an aneurysm needle should be passed so as to pick up the nerve and artery as they emerge from the bony channel, the point of the needle being passed on to the bone before it is dipped to pick up the nerve. In most cases the infra-orbital artery is picked up with the nerve, but the latter can easily be isolated as soon as it is brought into view. The free bleeding which results from incisions in the face usually renders it difficult to see to the bottom of the wound in this operation, and a little time is well spent, after dividing the skin and muscles, in plugging the wound with sponge for a few minutes, to stop some of the oozing.

Meckel's ganglion.—To excise this ganglion, a curved incision, with the convexity downwards, should be made, so that the centre of it is about half an inch below the level of the infra-orbital canal. The flap thus reached should be dissected up to a point half an inch above the level of the canal, and the latter should then be exposed by the free division of the subjacent fat and facial muscles, and by cleaning the periosteum off the bone with a raspatory. The front wall of the antrum, just below the canal, must then be perforated with a small trephine, all bleeding points tied, and oozing stopped by sponge pressure. A slender Bowman's probe should then be passed along the canal as far as possible, and, using this as a guide, the floor of the canal must be cut away from below with bone scissors until the posterior wall of the antrum is reached. This, in its turn, being perforated by the trephine, the ganglion will be reached by tracing the infra-orbital nerve to its junction therewith. The ganglion and the nerve are then to be cut away with curved blunt-pointed scissors. Hæmorrhage must be arrested by plugging.

The inferior dental nerve.—This nerve may be exposed either from within the mouth or by skin incisions. In the former method the mouth is opened widely with a gag, and an incision is carried along the anterior border of the ramus of the lower jaw, extending from the last upper molar to the corresponding tooth in the upper jaw. After the division of the mucous membrane, the internal pterygoid must be separated from the jaw with the finger, and the sharp spike of bone which marks the orifice of the inferior dental canal must be felt. An aneurysm needle is then passed forwards from the inner aspect of the jaw-bone, and the nerve is hooked up on this and drawn forwards. In dividing the nerve, care must be taken to avoid the inferior dental artery, the injury of which in this operation has caused serious hæmorrhage.

The facial nerve.—The facial nerve may be exposed at the point of emergence from the stylo-mastoid foramen by an incision commenced behind the ear at the level of the external auditory meatus, and carried downwards and forwards to about the level of the angle of the jaw. After dividing the skin and fascia the inner edge of the sterno-mastoid is exposed and retracted, and the posterior belly of the digastric muscle is then defined and similarly dealt with. A blunt hook or aneurysm needle should then be slipped down, so as to pick up the nerve, and the latter should then be separated from the tissues around it by the use of a probe-pointed director. The parotid gland must be drawn forwards before any attempt is made to pick up the nerve. The operation is a difficult one, especially in thick-necked and muscular subjects.

The spinal accessory nerve.—This may be exposed either before it enters the sterno-mastoid muscle or else at its point of emergence therefrom. To reach it in the former situation an incision is made parallel to the anterior margin of the sterno-mastoid, and the latter is strongly retracted. The centre of this incision should be opposite the hyoid bone, and at this level the nerve should be sought for, crossing the carotids and the internal jugular vein, and entering the muscle on its under surface. At the posterior margin of the sterno-mastoid the nerve is reached by an incision carried parallel with this margin, and having its centre opposite the middle of the line drawn from the clavicle to the mastoid process. Care must be taken not to mistake the superficial cervical nerve for the spinal accessory—the former curves forwards round the sterno-mastoid, the latter runs obliquely backwards. The spinal accessory nerve is not infrequently wounded in operations for the removal of cervical glands.

Nerves of the upper extremities. *The brachial plexus.*—This may be exposed in the neck by an operation very similar to that for ligature of the subclavian artery, and in the axilla by one similar to that for ligature of the axillary in the third part of its course.

The median nerve.—In the upper arm this nerve may be exposed by an incision similar to that for ligature of the brachial artery, and in the fore-arm may be reached by an incision above the wrist on the outer side of the tendon of the palmaris longus.

The ulnar nerve.—Behind the internal condyle the ulnar nerve may be easily exposed where it can be felt rolling over the subjacent bone, and at the wrist may be reached by an incision on the radial side of the flexor carpi ulnaris.

The musculo-spiral nerve.—This may be exposed either in the middle or lower third of the arm. It is most easily reached in the former situation, and should be exposed by an incision in the long axis of the arm, in the middle of a line drawn from the front of the external condyle to the insertion of the deltoid. After the skin and fascia have been divided, the nerve can generally be felt rolling over the smooth surface of the humerus. It should be cleared by separating the triceps from the supinator longus and the brachialis anticus.

Nerves of the lower extremities. *The great sciatic nerve.*—The course of this nerve is in a line drawn from a point between the tuber ischii and the great trochanter—but one-third nearer to the tuber ischii—to another point in the middle of the popliteal space. In the upper part of this course the nerve may be exposed by an incision below the gluteal fold, the gluteus maximus being drawn upwards and outwards, and the biceps muscle inwards. If the operation be performed in the middle of the thigh, the biceps muscle must be drawn to the outer side, as in this situation the nerve lies to its inner side.

The internal popliteal nerve.—This may be found beneath the deep fascia in the middle of the popliteal space.

The external popliteal nerve.—This can readily be exposed by an incision on the inner side of the biceps tendon, about an inch above its insertion into the head of the fibula.

The anterior and posterior tibial nerves may be exposed by operations similar to those for the ligature of the vessels which accompany them.

II. NEUROMA.

Varieties.—Neuroma is a term which is usually applied to any tumour growing on a nerve trunk, whatever its structure may be. Neuromata are commonly divided into two classes: (*a*) true neuromata; (*b*) false neuromata. (*See* page 461.)

(*a*) A **true neuroma** is one which is composed of nervous tissue, and is a very rare form of growth. It may contain medullated or non-medullated nerve fibres, or else may be in part composed of ganglionic cells, with a surrounding network of fibres, but the latter variety of growth is only met with in tumours connected with the brain or spinal cord. True neuromata form rounded or oval swellings of small size. The nerve tubes of which they are composed are usually not continuous with those of the nerve trunk to which they are attached, but form an irregular network, mixed with a varying amount of loose connective tissue.

(*b*) A **false neuroma** is a tumour situated on a nerve, and not itself containing any nerve elements. The most common form of false neuroma is composed of soft fibrous tissue, or of fibrous tissue undergoing myxomatous degeneration (Fig. 193). Gliomatous and sarcomatous tumours may also occur, but they are less frequent. The latter tumours differ from the fibrous growths in the greater rapidity of their development, in their tendency to infiltrate rather than simply to push aside the nerve fibres amongst which they grow, in their greater softness, and in the gelatinous or mottled appearance of their cut surface.

All these growths are most common in the nerves of the extremities; their size varies from that of a pea to that of a hen's egg, but most of the growths are about the size of a hazel-nut or a walnut. In shape they are generally oval, the long axis of the growth lying parallel with the fibres of the trunk in which it is formed; their surface is smooth, and in many cases the tumour lies amongst the surrounding nerve bundles, without actually involving the latter. In other cases, however, one or more of the nerve bundles may pass quite through the tumour, so that it is not possible to separate the growth from the nerve.

Another form of neuroma is sometimes described: the "**traumatic neuroma.**" This is identical in structure with the "bulb" on the proximal end of a divided nerve, already described on page 682; but if a nerve be only partly cut across, a bulb will form at the seat of injury, and will thus form a swelling on the nerve trunk itself.

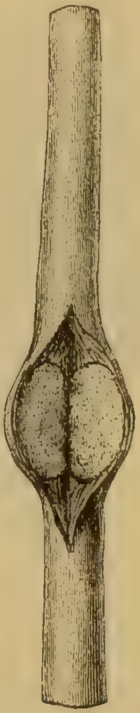
Fig. 193.—Fibro-neuroma of Median Nerve.

Symptoms.—The diagnosis of a neuroma depends partly on the symptoms caused by its presence, and partly on the physical characters it presents. The most characteristic features of such a tumour are its extreme sensitiveness, and the sharp shooting neuralgic pain caused by touching it. In addition to pain, the pressure on the nerve fibres may give rise to numbness, tingling, or pricking sensations in the parts supplied by the affected nerve, and in a few cases complete anæsthesia results. Muscular twitchings are less common symptoms, whilst clonic or epileptiform convulsions, although described, are certainly very rare complications. So-called "trophic" lesions are also uncommon, but are of more frequent occurrence than muscular spasms. They comprise such changes as have already been described as following sections of a nerve—*e.g.* glossy skin, ulceration of finger-tips, painless whitlows, increase or diminution of sweat, etc.

On examination, a neuroma is found to be a smooth oval swelling of variable size, situated in the course of a nerve trunk. It can readily be moved in a lateral direction, but is found to be scarcely, if at all, movable in the long axis of the nerve on which it is placed.

Very slight handling causes severe pain, which is often very characteristically distributed to the skin area supplied by the diseased nerve, and spasm of the muscles is easily produced by rolling the tumour beneath the fingers. The benign or malignant character of the growth may be diagnosed, though not with certainty, by consideration of the rapidity of its growth and the size attained. Finally, it should be mentioned that in some cases neuromata are multiple, and in some instances such tumours, composed of fibrous tissue, have been found to the number of several hundreds scattered over the nerves in all parts of the body.

Treatment. — Removal is the only treatment that can be practised. The incision should be made in the long axis of the nerve, and such incisions as are necessary to free the tumours should also be made in the same direction, so as to avoid as far as possible the section of the nerve fibres. If excision of the portion of the nerve containing the tumour be necessary, the trunk should first be very thoroughly stretched, so that after the resection the cut surface may be sutured. In other cases where, on account of the large size of the tumour, it is impossible to reunite the severed nerve, a suitable portion of the nerve of a rabbit or of a recently amputated limb should be introduced as a graft, and sutured between the severed ends.

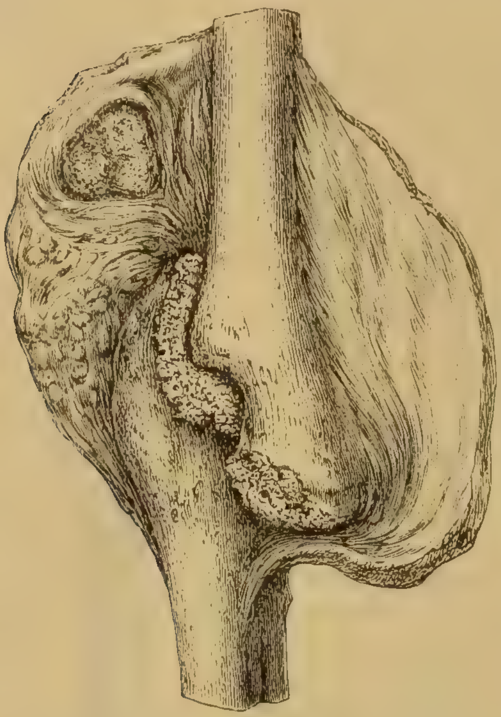

Fig. 194 —Knee Joint from a Case of Charcot's Disease.

III. SURGICAL AFFECTIONS COMPLICATING DISEASES OF THE SPINAL CORD.

It is only within recent years that many surgical affections which are now recognised as being due to disease of the spinal cord have been referred to their true source, and yet such complications are so numerous and far-reaching that it is only possible to allude here to the most important of them. It may, however, be mentioned that many of these complications are in all respects similar to those which follow on injuries of the spine, and that the cystitis, pyelitis, acute bed-sores, etc., which are so often seen after crushes of the spinal cord may also ensue upon disease. There remain, however, certain pathological processes which are peculiar to patients affected with disease of the cord, and these merit a more detailed description.

Charcot's disease; tabetic arthropathy.—Charcot's disease is a form of arthritis allied to osteo-arthritis which is usually developed in connection with tabes dorsalis, but is also seen as a rare complication of syringomyelia, itself a rare disease. It is met with in but a small percentage of cases of tabes, and often commences when the tabetic symptoms are but little marked, or even unnoticed by the patient. Very frequently it occurs before there is any evidence of ataxic gait. In many cases the disease is of sudden onset, the affected articulation becoming distended with fluid within twenty-four hours, and the swelling being unattributable to any apparent cause. This swelling of the joint is often accompanied by a swelling of the neighbouring soft tissues, which do not, however, pit on pressure to any extent. In some instances the effused fluid is absorbed, and the joint returns to its natural condition; but in others—which are, unfortunately, the more common—the effusion is but the commencement of a series of changes, which rapidly terminate in the destruction of the articulation. Within a few weeks or months of the first attack the patient notices that the joint gets weaker, and gives way under him; and very rapidly the articulation becomes so loose and freely movable that in many cases dislocation ensues (Figs. 194, 195).

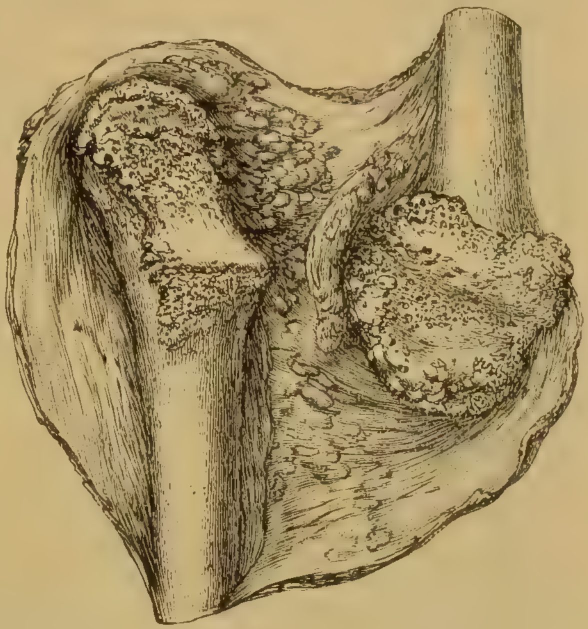

Fig. 195.—Another View of the Femur and Tibia shown in Fig. 194.

Morbid changes.—An examination of such a joint shows changes very similar to those of osteo-arthritis, but, especially in the case of the bones, the lesions are much more extensive. The thickening of the synovial membrane and the formation of fringes on it, the fibrillation and wearing away of cartilage, the growth of ecchondroses, and the wearing away and destruction of ligaments, are so similar to the lesions of osteo-arthritis that they need no special description (Fig. 194).

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The changes in the bones are of more importance, and are also more characteristic. In typical cases they are worn down to an extent never seen in osteo-arthritis, and are, moreover, often simply worn down without any new bone being produced, as is common in the latter disease. In typical cases, the whole head of the femur or humerus, the condyles of the femur, or the head of the tibia are ground down and destroyed, as if they had been rubbed away by a grindstone or a file (Fig. 195). It is this extensive destruction of bone even more than the wearing away of the ligaments which must be held accountable for the dislocations and undue mobility above mentioned. But whilst the above changes may rightly be considered as typical of certain cases of tabetic arthropathy, it must be allowed that in some examples of this affection the destruction of bone is not so rapid or so complete as here described; and whilst no new bone at all is formed in some cases, in many others very large masses of bone are formed in the substance of the synovial membrane, and may attain a much greater size than those seen more commonly in osteo-arthritic joints.

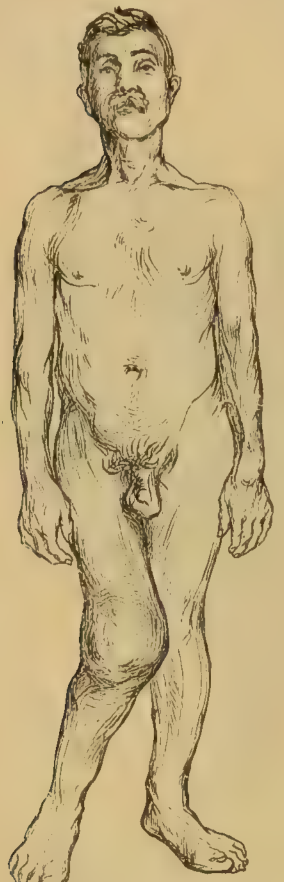
Fig.196.—Charcot's Disease of the Knee.

Symptoms.—The diagnosis of Charcot's disease rests mainly on the existence of disease of the spinal cord, associated with painless swelling and loss of power in the affected joint.

As regards the former, the symptoms of tabes may be briefly enumerated, and may be divided into those noticed by the patient and those perceived on examination by the surgeon. Pains in the limbs of a shooting character—so-called "lightning pains"—and other pains, variously described as boring, cutting, or shooting, are of frequent occurrence. "Girdle pains" and the sensation of constriction round the waist are less frequent. Numbness or partial loss of sensation in the feet is less common. Sudden and uncontrollable desire to evacuate the contents of the bladder or to empty the rectum is of occasional occurrence, and attacks of vomiting without cause—"gastric crises"—may also occur. Inco-ordinations of the lower extremities—"locomotor ataxy"—is a very characteristic feature of the disease, but in many patients it does not come on in a marked form until late, though the patient may have noticed that in bad lights his footing was insecure. Various symptoms may have been noticed about the eyes. Thus, there may have been occasionally a transient squint, or vision may be impaired, or actual blindness may have resulted.

An examination of a patient with tabes may not only verify some of the above symptoms, but may elicit others. Usually, the first thing to do is to test the knee-jerks; and in most cases of tabes these are absent, though not necessarily in all. The stability of the patient may be tested by making him stand with his heels together and his eyes shut, or by making him walk whilst blindfold, or by standing on one leg. An examination of the eyes will usually show that the pupil does not contract to the stimulus of light, but that when the vision is concentrated on some near object, it does contract during accommodation. Ophthalmoscopic examination may reveal grey atrophy of the optic disc, and the field of vision may be diminished.

If the joint affected be seen within a few days of the first attack, it will be found to be greatly swollen and tense, and to contain a large quantity of fluid; the limb in the neighbourhood is usually swollen also. At a later stage the effusion is less marked, and the surrounding swelling much diminished or absent. The most noticeable feature at such a time is the extreme mobility of the articulation, so that, instead of the movements being limited as in most forms of disease, there is abnormally free movement in all directions. Thus, circumduction at the shoulder or hip is permitted to an extent unknown in health, and in the knee or elbow there may be hyper-extension and lateral mobility (Fig. 196). When this condition is still further developed, a so-called flail joint results, and at any period dislocation may ensue. In many such cases, where the joint is sufficiently superficial, it can be felt that certain portions of articular bone are worn away, and in other cases masses of new bone may be felt in the synovial membrane. Another most remarkable feature of such a case is the complete, or almost complete, absence of pain, with or without movement, and this is all the more noticeable when the case is seen early, and the effusion present is such as to make it certain that in all other forms of arthritis there would necessarily be a good deal of pain. Lastly, it should be mentioned that all cases of Charcot's disease do not run so acute a course as is here described, and that in some of them pain is present; the development of bony nodules in the synovial membrane may also prevent the free mobility which usually characterises the disease.

Treatment.—Unfortunately, no treatment can arrest the progress of this affection. Rest in the early stages, and the application of splints to keep the joint surfaces from attrition, are all that can be advised.

Spontaneous fracture.—On account of the atrophy of the bones in many cases of old-standing paralysis, they are very liable to fracture on the application of but slight violence, and such bones are very slow to unite when broken. Spontaneous fractures, however, are liable to occur also in tabetic patients who have never been paralysed or bedridden, and a considerable number of such cases have been recorded. In most of them the fracture has occurred in the early stages of the disease, and without any premonitory symptoms to point to any antecedent disease of the bone. In many of the cases union has been obtained without difficulty, but in some it has been delayed, and in others no union has occurred. Occasionally, as in a case seen by the writer, numerous spontaneous fractures of different bones have occurred at intervals of months or years, and yet have all united. In some of these cases a great excess of callus is formed. (*See* Art. XXIX., page 726.)

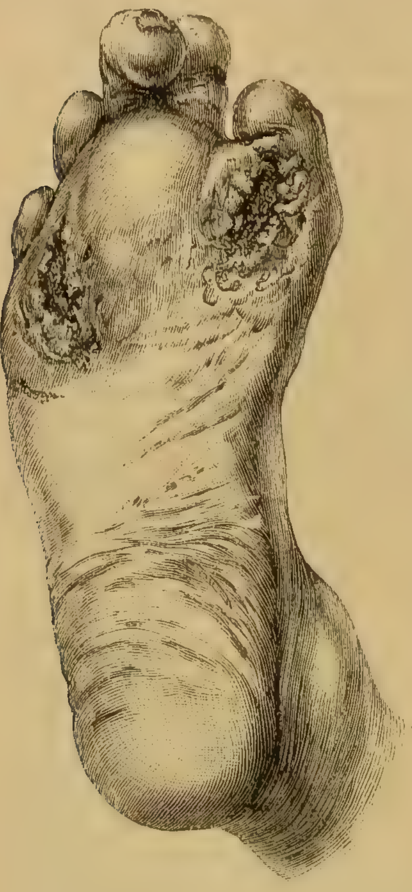

Fig. 197.—Perforating Ulcers of the Foot.

Perforating ulcers.—These are much more commonly seen in patients who are the subjects of tabes dorsalis, but occasionally complicate other diseases of the spinal cord or of the peripheral nerves and occur also in diabetic subjects. When complicating tabes, they may develop either as one of the early symptoms or at any period during the progress of the disease. (*See* page 112.)

Symptoms.—These ulcers are usually situated on the sole of the foot, and especially affect the balls of the great toes, though they may occur under the balls of any of the toes, and have been seen in one or two instances on the hand. They commence with a corn-like thickening of the epidermis, which may precede ulceration for several months; but when once found, the ulcers evince a special tendency to "perforate" the subjacent structures rather than to spread over the neighbouring skin (Fig. 197). As a consequence of this perforation, the metatarso-phalangeal joint is frequently involved, and, as the result of the suppurative arthritis set up, its cartilage may be destroyed, and its articular surface become carious (Fig. 198). These ulcers are sometimes multiple, and are often symmetrical. Occasionally the ulcers become the starting-point of extensive sloughing, or moist gangrene, and this is especially common in those cases where the patient is suffering from diabetes.

Diagnosis.—The diagnosis of a perforating ulcer is usually an easy matter, but care must be taken not to confuse the suppuration of a simple corn with the more serious disease. The presence of some nervous lesion or of diabetes may be accentuated by some anæsthesia of the skin around the ulcer, and this is occasionally associated with foul sweating of the foot. The passage of a probe will often demonstrate the presence of rough bare bone, and movement of the subjacent joint may result in grating of the carious surfaces. Pain is, as a rule, not severe.

Treatment.—These ulcers are usually most amenable to treatment, and it is quite erroneous to suppose that they do not heal. Their situation suggests at once that pressure may act as an exciting cause, and the first indication, therefore, is to keep the patient off the affected foot. Unless this is done other treatment is useless. In addition to this precaution, but little is required beyond ordinary cleanliness and care. After the surrounding unhealthy skin has been cut away, the use of a foot bath for some hours daily and boracic fomentations are generally necessary when cases first come under treatment; and at a later stage iodoform powder or some stimulating application—such as unguentum resinæ, or a solution of sulphate of zinc—is indicated. In many cases where the joint surfaces are carious, good results may be obtained without operation, but sometimes it becomes necessary to gouge away the diseased bone in order to promote a cure. When the ulcer has healed, it must be protected from pressure by a suitable felt pad with a central hole, inserted in the boot, or else applied in the form of a corn-plaister. These ulcers are very liable to break out again and again, as the result of renewed pressure in walking, and constant care is necessary to keep the foot sound.

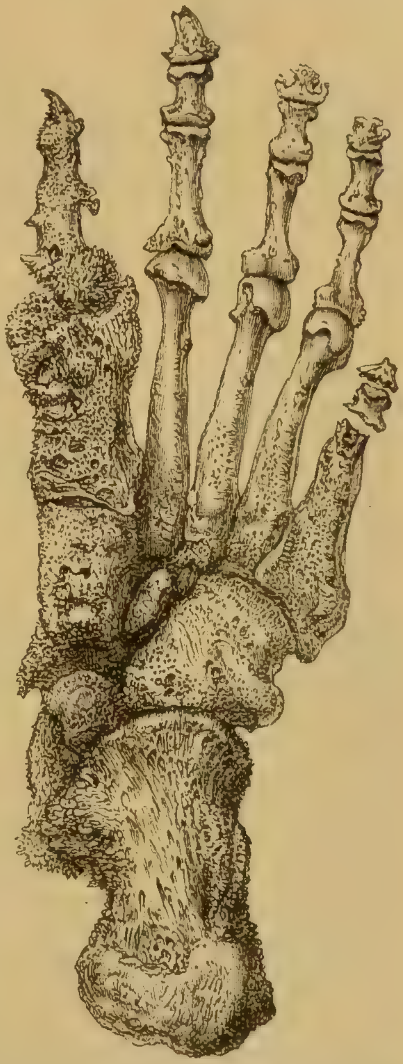
Fig. 198.—Bones from a Case of Perforating Ulcer, showing caries and also destruction of the terminal phalanges.

In a very few cases which resist treatment, as above, there is extensive sloughing of the soft tissues of the sole of the foot. Amputation may be performed; but amputation, as a routine treatment, is uncalled for and most unsatisfactory in its results, as the ulcer often recurs in the stump. It is best to reserve such treatment for those cases where there is not merely an ulcer, but sloughing or gangrene as well; and in these also the results are usually disappointing.

XXVIII. DISEASES OF THE SKIN.

By JONATHAN HUTCHINSON, Junr., F.R.C.S.,

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Corns.—As the result of prolonged pressure or friction the horny or epidermic layer of the skin is prone to take on local hypertrophy, preceded by congestion of the papillary layer beneath. A simple thickening of the epidermis at one spot, such as is often seen on the fingers of seamstresses or workmen, or on the palmar aspect of the first phalangeal bases, is known as a *callosity*. A corn differs from a callosity in the fact that there is in addition a central downgrowth of epidermis displacing the papillæ, which at the edge of the corn are elongated and enlarged as well as abnormally congested. True corns are not often met with except on the feet, where the compression of the toes by ill-fitting narrow or tight boots renders the dorsal surface of the toes peculiarly liable to them. The plantar surface of the foot under the heads of the metatarsal bones is often affected, less commonly the heel. In fact, probably few of the boot-wearing race escape the development of corns at one or other of these positions.

The epidermic cells towards the surface of a corn undergo enlargement and remarkable keratinisation, but sometimes the wedge of epidermis becomes soft, and is then especially painful and irritable. A further stage is the formation of a small abscess under or in the corn, and ultimately of an ulcer with hard raised edges. Such ulcers under the "tread" of the foot may extend deeply into the tissues of the sole, and constitute one form of the so-called "perforating ulcer." They are especially liable to develop in patients the subject of tabes or some other nervous disease involving impaired sensation in the foot, but may occur quite independently of this. (*See* page 704.)

Another variety of soft corns is met with between the toes, the excessive secretion of sweat giving them a sodden white aspect. Hard corns are occasionally complicated by the development of a bursa beneath them (constituting one variety of bunion), which, of course, under sufficient irritation may inflame or suppurate. Another complication of a corn to be noted is the occurrence of small hæmorrhages into it or the corium beneath.

Owing to the inflammatory congestion of the papillæ around the epidermic downgrowth, corns are generally painful, and any condition which increases this congestion (such as too prolonged walking or standing) may render them exquisitely tender. In many people the atmospheric change accompanying the approach of wet weather has a most marked effect in causing shooting pain or "twinges" in their corns.

Apart from the fact that inflamed corns often completely disable their sufferers from active exercise, it must not be forgotten that they may be the starting-point of more serious troubles. If an abscess or ulcer form at their site, lymphangitis or spreading suppuration may follow their neglect or injudicious treatment, and in old people, with atheromatous arteries, cases of senile gangrene have been recorded which owed their commencement to this comparatively trivial cause.

Treatment.—It is essential for its cure that the injurious pressure which has produced a corn should be removed. Broad-toed boots with low heels are required in most cases, though sometimes the fault lies in the upper leather being too thick or badly-shaped. Each boot should be carefully designed to fit the individual foot. After soaking well in hot water, the hard epidermis, and especially the central core, should be carefully cut away with a sharp scalpel, and a circular ring of felt plaster should be worn to protect the site of the corn from pressure. Salicylic plaister (10 or 20 per cent.) or a saturated solution of salicylic acid in collodion frequently applied will act well in softening the corn, after which the central part may be more readily cut out. In using the knife only the thickened cuticle should be removed, and hence no bleeding should occur.

In cases of hammer-toe (where the second toe is hyper-extended at the metatarso-phalangeal joint and strongly flexed at the first inter-phalangeal one) a very troublesome corn is apt to develop over the latter articulation. The only effectual treatment in some of these cases is to amputate the offending digit.

Warts (verrucæ).—A cutaneous wart consists essentially in a pedunculated overgrowth of the epithelium, in the centre of which is an extension of the papillary layer of the corium containing a vascular loop and often a small nerve. The peduncle may be exceedingly slender, whilst the projecting part of the wart branches in a cauliflower-like manner. They vary much in consistence, the hardest ones generally occurring on exposed parts which are kept dry, whilst those situated on a region of the skin habitually moist are often very soft and delicate (Fig. 199).

It should be mentioned that warts may develop on mucous membranes, such as the lining of the lips, the soft palate, the conjunctiva, the vaginal or urethral walls.

It is practically impossible to draw any distinction between a wart and a papilloma, and the so-called papillomata of the larynx have a similar structure to many warts of the skin, allowing for slight differences due to their site. The colour of warts varies from greyish-white to pinkish-red, the latter being dependent on free vascularisation, and therefore being associated with a tendency to bleed readily if abraded.

Causes.—There is no doubt that local irritation, *e.g.* of some decomposing secretion, is the most frequent exciting cause in the formation of warts, and it is probable that microbes play a considerable part; indeed, a special bacterium has been described as present in many cases. In connection with this it is noteworthy that some warts appear to be due to inoculation, and their tendency to occur in clusters or groups is often very marked. Secondary syphilis is also a common cause; thus a neglected condyloma may pass into the warty condition and, apart from any mucous patch or condyloma, warts are liable to form on the dorsum of the tongue and elsewhere during the secondary stage.

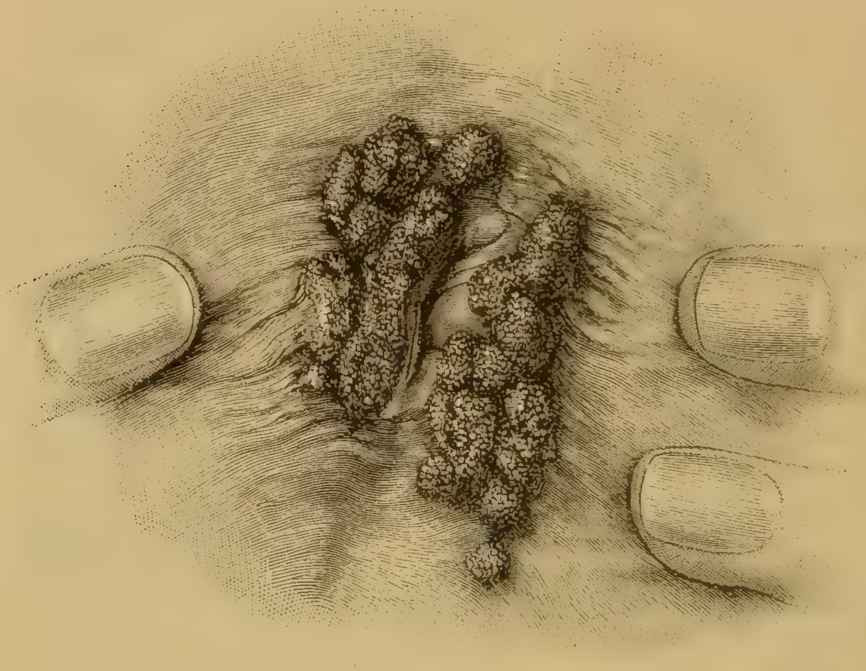

Fig. 199.—Warts round the Anus, showing clusters of "cauliflower-like" growths. (From a patient who had recently had both gonorrhœa and syphilis.)

Tubercle of the skin may also lead to warty growth: thus lupus when it affects the feet and other regions covered with dense epidermis is often warty in large part. The so-called *verruca necrogenica*, or post-mortem wart met with on the dorsum of the hand (especially over the knuckles), has been proved to contain tubercle bacilli, and is as difficult to cure as any form of lupus, although it rarely ulcerates. (*See* page 297.) From these considerations it is correct to consider warts as essentially inflammatory in origin, the cause being probably of microbic nature.

It must, however, be noted that some forms of epithelial cancer are at first warty in structure, as, for instance, many cases of epithelioma of the glans penis, the lower lip and the tongue. The older the patient and the longer the warty growth has persisted, the greater is the risk of it passing into epithelial cancer, especially if irritated by being continually "picked at," etc. Histologically, the stage of cancer is reached when there is not only epithelial downgrowth into the corium, but this downgrowth breaks up in an irregular manner amongst the cells of the latter.

The **multiple wart.**—We will now consider two varieties of warts in which it is difficult to assign any irritative cause, and which occur in a disseminated form. The first is the multiple wart met with generally in young male adults or children, and especially prone to occur on the backs of the hands or fore-arms. They may be flattened, but are more commonly branched or filiform, of a greyish colour (sometimes black from their tendency to collect dirt-particles), and have nearly always a narrow pedicle. They are unsightly, but cause no other trouble; and their most remarkable feature is the occurrence in crops and their occasional equally sudden disappearance. The latter has led to various popular nostrums being vaunted as curative of warts, and amongst the drugs which have been apparently successful at times are sulphate of magnesia and arsenic given internally. The surest way, however, of causing these warts to disappear is by local treatment, and one of the best applications is glacial acetic acid. This should be applied daily with a fine brush or pointed piece of wood, it is almost painless and causes the wart to whiten, shrivel, and drop off. The process should be repeated if there is the slightest sign of re-appearance. Pure liquor arsenicalis, nitric acid, ethylate of sodium, and other caustics are sometimes used, and a very common plan is to treat each wart vigorously with nitrate of silver. The latter leaves an ugly black stain for some time, and the others are decidedly more painful, and no more efficient than glacial acetic acid.

The **sessile wart.**—The second form is the sessile wart, occurring in a large proportion of old people, sometimes on the face or other exposed part, but especially met with on the skin of the trunk. They are nearly always multiple, flat-topped, often brown or brownish-red in colour, and they may be soft and somewhat greasy to the touch. They persist indefinitely, and are of some interest from the fact that epithelioma or rodent ulcer (on the face) may start in connection with them; this sequence is fortunately rare. In any case where the cancerous change is suspected the growth should be excised freely, and when, as sometimes happens, a sessile wart on the face gives trouble by repeated hæmorrhage on slight irritation, or is unsightly, it may be readily got rid of by cauterisation with a drop of nitric acid, nitrate of mercury, or glacial acetic acid.

Warts on the genitals.—Warts on the genitals of both sexes have already been mentioned (they are apt to occur here in dogs and other animals as well as in man), but it must be noted that on the vulva and glans penis they may develop to an extravagant extent. Generally they owe their origin to an attack of gonorrhœa, but retained secretion (under a non-retracted prepuce, etc.), or the irritation of sexual intercourse may produce them. On the vulva they may develop so freely as to entirely hide the vaginal orifice and the labia, and in these cases there is usually a most offensive odour present.

Mild cases require merely strict attention to cleanliness, aided perhaps by the use of a powder of equal parts of tannic acid and iodoform.

If any gonorrhœal discharge persist, of course it must be carefully treated; and where the warts fail to yield to the above treatment they should be nipped off with curved scissors. When excising large masses of warts it is necessary to give an anæsthetic, and to apply the actual cautery to the bases, which tend to bleed very freely. It is certainly important to cure these cases of extensive venereal warts in women, as the decomposing discharge which attends them is sometimes absorbed and causes marked depression in health, and even symptoms similar to those of pyæmia.

Summary.—To recapitulate, the chief varieties of warts met with may be grouped in the following manner:—

1. Warts on the genital surfaces, due always to some local irritation.
2. Warty growths developing in old neglected condylomata, or otherwise depending on secondary syphilis for their origin.
3. The verruca necrogenica (tubercular in nature?) met with on the hands of surgeons, pathologists, and butchers.
4. The sessile or flat-topped warts (widely disseminated, and occurring mainly in late adult life).
5. Multiple warts on the hands, etc., of children and adolescents.
6. Warts on the mucous membrane of mouth, larynx, urethra, etc., the cause of which is quite unknown. (*See also* Papillomata in Art. XXII. page 476.)

Horns.—In severe cases of congenital ichthyosis we occasionally see miniature horny growths composed of hardened epidermic projections which may be detached by vigorous rubbing, etc.; with this exception horns are usually single, and develop, as a rule, in adult life or old age. That their origin is always in connection with a sebaceous gland is doubtful. Often they commence in the following manner: a sebaceous cyst ulcerates and then pushes up layer after layer of epithelioid cells, which dry and harden into a conical, often spirally-twisted or curved horn. They have been met with most often where sebaceous cysts are common, *i.e.* on the scalp and face (50 per cent. of the cases), after this region comes in frequency the internal aspect of the thighs and (very rarely) the genitals. However large and hard a horn may be in the human subject its structure is entirely epidermic, and, if excised, the base is found to be comparatively soft, and, as already noted, to consist generally of the remains of a sebaceous gland or cyst, which it is essential to remove in order to prevent recurrence. Upward papillary projections containing vessels may occur in the base, but the hæmorrhage is only slight during their removal, which is the only treatment advisable. It should be remembered that occasionally a horny growth occurs with an epithelioma at its base, and this has been seen several times in a part of skin for long the site of common lupus. In such a case it is, of course, necessary to excise both horn and epithelioma (the latter having a curious structure somewhat suggesting that of a sebaceous gland), and if this is done freely the prognosis is fairly good. (*See* Art. XXII. page 476.)

Boils (furunculi) and carbuncles.—It is convenient to discuss these together, since it is impossible to draw a sharp distinction between the two. It may be said that whilst a boil is a small abscess developed around a hair-bulb and its glandular appendages, and discharging by a single orifice, a carbuncle involves a definite slough of connective tissue, and presents several openings for the discharge (page 136). The micro-organisms which play an important part in both are probably identical (the staphylococcus pyogenes aureus being the chief); the favourite sites for both are the back of the neck and trunk, the perineum and buttocks; and a similar condition of ill-health may predispose to the development of either.

Causes.—With regard to carbuncles, diabetes mellitus is occasionally found in their subjects, but this is quite exceptional, and we may roughly classify the predisposing causes of boils and carbuncles thus:—

1. The debility met with in convalescence from typhoid and other fevers.
2. Insufficient animal food, or a diet imperfect in other ways: thus excess of the nitrogenous elements may be to blame (butchers, for instance, are very liable to develop boils).
3. A sudden change of diet and habits, as, for instance, in rowing men or athletes who have just gone into training.
4. General privation, long-continued work in a close unhealthy atmosphere, chronic renal disease, etc.

Great importance, however, must be attached to local inoculation with the micrococcus mentioned above, which is identical with that which in young subjects may produce acute osteo-myelitis. And in many cases of the latter grave disease there is reason to think that the organisms have entered the circulation by means of some cutaneous boil. Conversely, experiment has proved that friction on the skin with a little pus from a case of acute diffuse periostitis will lead to a crop of boils, having their origin around the hair-bulbs. It must be remembered that many micro-organisms (including the staphylococcus pyogenes aureus) can be found at times on perfectly healthy skin, and the reason why boils and carbuncles are so common on the nape of the neck and buttocks is no doubt that these parts are peculiarly subject to friction and chafing from the clothes, and hence to inoculation. The common cases of boils in rowing men are more probably due to this cause than to any peculiar state of general health.

Manifestations.—The germs enter, in the examples just mentioned, as a rule, through a hair-follicle and the glands which open into it, and multiply in the cellular tissue just around. Inflammatory reaction leads to the formation of a raised itching and painful lump in the skin, often topped by a hair, which may be the centre of a small pustule. Resorption may take place if the boil be carefully protected from friction, etc., but the general rule is for suppuration to occur in from four to six days, and until the pus is let out the pain and tension may be very severe. This is especially the case in such situations as the auditory meatus, the nose, the perineum, and the buttock. The greatest possible relief is obtained by an early incision with a sharp narrow-bladed knife, followed by warm antiseptic poultices—*e.g.* carbolic lotion, 1 in 60, on boracic lint covered with oiled silk or guttapercha tissue. Granulation and healing will quickly occur in the case of a small boil, but a carbuncular slough may be of very large size, and the resulting excavation may take several weeks to fill up. This slough is of a greyish-white swollen aspect, adhering for some time to the surrounding corium by processes, traction on which is extremely painful. The orifices through the overlying skin may be very numerous unless an early incision has been made, and there is, as a rule, considerable undermining of the edges. Micrococci are readily found in the discharge, and occasionally long bacilli, which resemble somewhat those of malignant pustule or true anthrax. In the latter affection (which in England is only met with amongst those engaged in sorting or carrying hides of dead sheep or oxen, or who are otherwise brought into contact with them), no pus is formed, there is a small black eschar at the centre of a large patch of congested and swollen skin. (*See* page 311.) The constitutional disturbance also is much greater, the patient's temperature, for instance, with a malignant pustule may reach 104° to 106°, and the prognosis is infinitely more grave. Nevertheless, a carbuncle, if of large size, and occurring in an elderly subject with diabetes or in feeble health, may prove fatal, either through exhaustion or pyæmia, or by the supervention of erysipelas or diffuse cellulitis.

Carbuncles of the face, especially those of the lips, are perhaps more dangerous than any others, probably owing to the extreme vascularity of the part, and the readiness with which the poison enters the veins.

In the case both of carbuncle and boils there is a marked tendency for infection of adjacent parts of the skin, particularly when ordinary poultices are used; sometimes a regular crop of smaller boils form around the primary one. Hence care should be taken to renew the dressing frequently, to use one no larger than is strictly required, and to foment with a solution of carbolic acid or corrosive sublimate instead of the linseed or bread poultice.

The regions of the body most often affected have already been mentioned. It remains to note that the dorsal surface of the fingers (especially over the first phalanges) and the axillæ are also frequently the sites of furuncles. The coarse hairs and accessory glands present, and the free perspiration in the latter position no doubt explains this fact. The scalp, however, is but rarely affected by either boils or carbuncles.

No part of the body is wholly exempt, and in certain unusual situations, such as the penis or the toes, a boil may give rise to considerable difficulty in diagnosis. The slough due to a subcutaneous or cutaneous gumma may resemble that of a carbuncle, but the rapid and very painful onset of the latter always serves to distinguish it. It is asserted that carbuncles in diabetic subjects are more insidious and less painful than the ordinary form, but this is only true in some cases. It must be noted that the sloughing in such diabetic patients may be very extensive and deserve the term of gangrenous cellulitis, recovery being comparatively uncommon.

Treatment.—In the early stage of a boil remove all pressure or cause of friction from the part, protect it with a small antiseptic fomentation, or use a thick circular pad of felt-plaister. Painting the surface with tincture of iodine, or counter-irritation *around* the inflamed area with the same application, or injecting a few minims of carbolic acid dissolved in glycerine into the circumference of the boil, are methods recommended to prevent suppuration. They, however, often fail, and when pus has been formed, and there is much pain and tension, the surgeon should not hesitate to incise. The old-fashioned free crucial incision is probably now rarely employed. An ancient plan of treatment of carbuncle has, however, been lately revived, consisting in cutting with scissors and scraping away all the slough, in the belief that this shortens the healing process. An anæsthetic should be given in order to do this. Undoubtedly some time and, perhaps, considerable pain are occasionally saved by this plan, but on the other hand the risk of opening up veins and causing pyæmia appears to be distinctly increased, and many surgeons have on this account abandoned it.

A good wholesome diet should be enjoined, with some wine or stout if the patient's health be depressed. Tonics, such as quinine, are useful, and the state of the alimentary tract should be attended to. A brisk purge is almost always advisable in the stage of tension and pain, and after this salicylate of bismuth or salol is recommended with a view to intestinal asepsis. To promote healing after the slough has come away there are few applications better than boracic or carbolic fomentations, but for an alternative the unguentum resinæ is useful, as is also iodoform diluted with three parts of boracic acid.

Various internal remedies are vaunted as preventing the tendency to repeated crops of boils, which some patients suffer from. Chief of these are sulphide of calcium and yeast; they are probably equally useless. Careful attention to the patient's diet and general hygiene and, if possible, a change of air to a bracing seaside resort, are far more likely to do good.

It remains to be noted that in the case of carbuncle occurring in diabetics, a special diet, in which sugar and carbo-hydrates are largely excluded, may be advisable; albuminuria may indicate a milk diet, whilst in any case, if the pain and distress accompanying the sloughing be severe, opium or morphia should be given in appropriate doses. The older the patient the more is this drug indicated, and it may well be combined with quinine.

Molluscum contagiosum.—This rather rare affection consists in the occurrence of a number of small raised umbilicated tumours, which are solid and have a structure resembling somewhat that of a sebaceous gland. They occur, as a rule, on the face and neck of children, more rarely on the genitals, the trunk, etc., of adults. Their size varies greatly, from that of a pin's head to a pea, and they rarely exceed this limit, having a tendency to inflame and to drop off when they have attained a certain size. The accompanying woodcut (Fig. 200) shows a crop of molluscum contagiosum on the eyelid, cheeks, lips, and neck of a child, and in the larger ones the central depression can readily be seen. They are aptly compared to mother-of-pearl buttons; their sharp definition, pinkish colour, and central depression all favouring this comparison. They usually develop in a crop and are undoubtedly contagious in a mild degree.

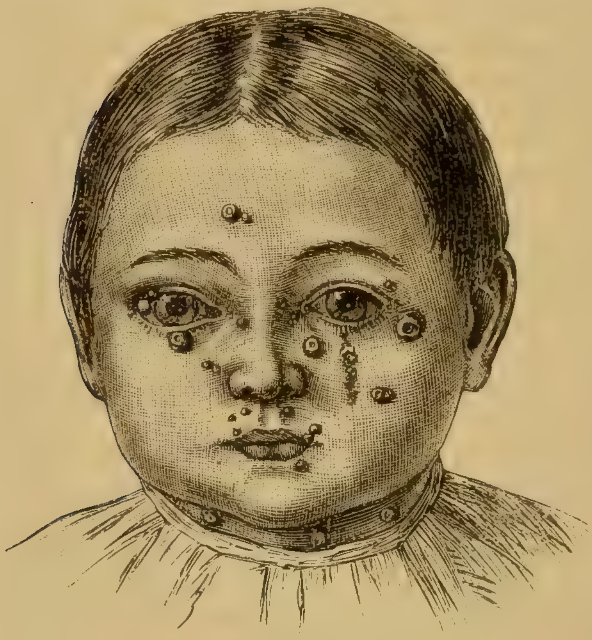
Fig. 200.—Molluscum Contagiosum on the Neck, Cheeks, Eyelids, etc., of a Child.

Conclusive evidence of this is obtained in some cases where a suckling woman develops them around the nipple, and the infant has them on the face; in adults a very extensive crop of molluscum contagiosum on the trunk, etc., may follow a Turkish bath with its attendant shampooing. The exact medium of contagion is doubtful, but probably consists in the central cells of the molluscum body, which are by some considered to be parasitic in nature. They have little importance except from the point of view of diagnosis; especially is this so when they occur on the penis and scrotum, when they are apt to be mistaken for venereal sores. Although somewhat resembling in microscopic structure a sebaceous gland, they certainly do not commence in the glands, but may start in any part of the skin surface.

Treatment consists in snipping them off with scissors, or in making an incision through them and squeezing out the whole molluscum body, which always presents a well-defined capsule.

Lupus.—This disease is to be regarded as a form of tuberculosis of the skin, extremely slow in its course and difficult to cure. It is divided into *lupus vulgaris* and *lupus erythematosus*, the former being much the more common. In both there is an infiltration of the corium with granulation tissue (masses of small cells, which may degenerate and lead to ulceration or organise into fibrous tissue). In both the disease tends to persist indefinitely, and to spread at its edge; in both a certain amount of evidence can generally be obtained proving their relationship to tubercular affections (*e.g.* history of phthisis in near relatives, occasionally the development of "strumous" glands or joints in the patients). In both, the face is the region of the body most often attacked. Finally, cases are sometimes met with which seem to combine the characters peculiar to common lupus and to lupus erythematosus.

Features of the two varieties.—The following are the chief distinguishing features:—1. Lupus vulgaris usually commences in childhood or early adult life, the erythematous form generally after thirty years.

Note.—Exceptions to both these rules are not infrequent.

2. Lupus vulgaris causes much more infiltration of the skin, and tends to ulcerate far more commonly than lupus erythematosus. The former may destroy or lead to the atrophy of deeper structures, such as the cartilages of the nose, the ears, the bones of the fingers.

3. In lupus vulgaris, beyond a tendency to the formation of "satellite" patches around a primary one, there is nothing special to be said about the grouping of the infiltration-nodules. In lupus erythematosus there is a very marked tendency to symmetry, and in particular the nose, both cheeks, and the hollows of the external ears are often invaded to precisely the same extent on both sides. The "bat's-wing" form of lupus erythematosus is very characteristic —the nose representing the body, and the patches on the cheek the two wings. The forehead towards the hairy scalp and the latter itself are also frequently involved, the backs of the hands less commonly. The disease is rarely seen except in the situations just mentioned, whereas common lupus may occur on any part, *e.g.* the feet, external genitals, arms, etc.

4. From the point of view of the microscopist, the cell-infiltration of common lupus is very prone to degeneration, is often accompanied by giant-cells; and by careful search tubercle bacilli (or micro-organisms indistinguishable from them) can be found in small numbers. The latter have rarely, if ever, been discovered in typical lupus erythematosus, in which, moreover, the infiltration is especially arranged around the sebaceous glands and the small vessels of the skin. Dilated tufts of the latter, often suggesting to the eye

nævoid structure, are one of the most characteristic features of lupus erythematosus. In common lupus a pinkish semi-translucent appearance may be present in the nodules, and has given rise to the comparison to "apple-jelly."

5. With the greater tendency of common lupus to ulcerate must be associated the occurrence of crusts or scabs, and sometimes of considerable pus formation.

6. Lupus vulgaris may attack mucous membranes, especially those of the lips, nose, palate, and eyelids.

It may be noted finally that both common lupus and lupus erythematosus are mainly diseases of temperate or cold climates, being rare for instance in Australia, and the cases are nearly always benefited by removal to warm and equable climates.

Stages of lupus.—The following stages of lupus vulgaris may be enumerated.

There is first a nodule of cell-infiltration of a pink or brownish-red colour; this spreads at its edge, fresh spots are formed in the vicinity, and they may persist in this form for many months or even years. As a rule, before long a pustule develops at the centre of the nodule, it bursts, and a small scab-covered ulcer results. The ulceration increases both at the edge and to a less extent in depth, and may occasionally cicatrise spontaneously; this, however, is rare, and even should it occur at one point the disease is probably continuing to spread at the edge. The scars left by common lupus, whether the result of treatment or not, are often coarse and thick, and prone to break down again: in other words, it is very difficult to thoroughly eradicate all the cell-infiltration, which is the essence of the disease.

A very common site for lupus to begin is the nose or cheeks, and from here it tends to invade the skin in the direction of the larger blood supply, *i.e.* towards the neck. Pain and itching are rarely marked, though the ulcers and scabbing may cause discomfort.

It is most important to recognise early and to treat vigorously every patch of lupus, since if neglected the scarring may lead to deplorable contractions and deformity; ectropion, narrowing of the nostrils or mouth-aperture, and contraction of the neck being each not infrequent in old and neglected cases. Further, if it once invades the mucous membranes of the mouth, etc., it is especially difficult to cure and has occasionally led to death by extension to the larynx.

Diagnosis.—The diagnosis, bearing in mind the features already described, is not as a rule difficult. If very superficial and attended with a thin dry crust instead of a scab, it may resemble a chronic form of *eczema*; but the frequent presence of scarring and the more marked infiltration in common lupus will usually decide the matter. The chief difficulty arises in distinguishing common lupus from *tertiary syphilitic* (acquired or inherited) *lupus*. The two diseases are totally distinct, and the proper treatment for one will be quite unnecessary or injurious in the other. The diagnosis is fully dealt with in the section on Syphilis. (*See* page 414.)

Complications.—Amongst the complications of long-existing lupus may be mentioned the following:—

(1) The occasional occurrence of tubercular (caseous) disease of the nearest lymphatic glands. This is, considering the true nature of lupus, surprisingly rare. It may be noted that experiments on rabbits, etc., have proved that if some of the lupus infiltration be inoculated into the aqueous chamber of animals it is followed by the formation of tubercle nodules in the iris, etc.

(2) The occurrence of a widespread outbreak of lupus nodules on many parts of the body, quite remote from the primary patch. This curious fact, observed most often in children, can only be explained by infection through the blood. These secondary patches may greatly improve or almost disappear after a time, or they may prove inveterate.

(3) Epithelioma is not an infrequent complication, occurring in elderly people, usually in the centre of a very chronic patch of lupus. It is generally of an exuberant or fungating type, and though certain to kill if left alone, may not recur if freely excised. Horny growths are also occasionally met with, and when lupus develops on parts with thick epidermic covering, such as the feet or hands, it has usually a warty appearance and hard consistence, very unlike the soft pinkish nodules common on the face.

(4) Atrophy of the digits, etc., when the hand is extensively involved, has already been noted. Here disuse and failure of growth must be assigned an important part in the production of the deformity, which occasionally affects a whole limb.

Treatment.—The treatment of common lupus consists in most cases in vigorously attacking the diseased patches, either by Volkmann's scoop and Paquelin's cautery, or by excision. Constitutional treatment (cod-liver oil, generous diet, etc.) is rarely of any use, and any local measures short of those above indicated are, as a rule, only attended with disappointment. If the lupus be very superficial a trial may be made of strong salicylic acid plaisters (*e.g.* 20 per cent.), and some cases are too extensive to treat at all; in the majority, however, much improvement follows scraping, cauterisation, or excision.

In the former method a small sharp scoop should be used, and the scraping can hardly be done too energetically; the soft infiltrated areas alone are affected by the scoop, and it is well to use a fine pointed cautery in the excavations made. Pure carbolic acid is a mild caustic of occasional value. Relapses are, unfortunately, frequent, and on this account many surgeons advocate excision, followed by grafting large thin epidermic layers from some healthy part of the skin (Thiersch's method). This is sometimes most successful, but is only suitable for certain selected cases where the disease is very limited. The grafts are cut by means of a razor, and may be applied either immediately after the excision, or later when healthy granulations have developed.

The treatment of lupus erythematosus is not very satisfactory. Perhaps the best results are obtained from cauterisation and scraping, but in many cases the patient prefers less heroic measures; then a trial should be given to a fairly strong tar ointment (half-a-drachm of liquor bituminis to the ounce of vaseline) or to the unguentum metallorum (B.P.).

Melanosis.—This term is applied to new growths—either sarcoma or carcinoma—in which dark pigment is present in the cells. It may commence in any part of the skin, not infrequently at the site of a congenital pigmented mole, or in the choroid layer of the eye. Occasionally it is met with in mucous membranes, *e.g.* that of the hard palate. It is a rare disease, but of great importance from its tendency to rapid dissemination in the viscera, especially the liver. The extent of these secondary deposits and the apparent rapidity of their formation bear no relation to the size of the primary growth. Occasionally one sees the liver increased to twice its normal size and coal-black from melanosis, resulting from an insignificant little mole, or what seems to be merely a small dark stain of the skin. In these cases, however, there is always a certain amount of growth, consisting of rounded or spindle cells, at the site of pigmentation. Sometimes this growth is not of sarcoma tissue, but of cancerous structure, starting from the deeper layers of the cutaneous epithelium and with a distinctly alveolar arrangement. Besides causing secondary deposits in the large viscera, a melanotic skin-growth is very apt to affect secondarily the nearest lymphatic glands, which enlarge considerably, and on section are found to be dark brown or black in hue. Finally, multiple subcutaneous tumours may form in the neighbourhood of the primary growth, also pigmented, and no doubt due to deposits through the cutaneous lymphatic system.

Melanotic growths occur with equal frequency in the skin and the choroid coat of the eye (page 470). Allusion has been made to their occasional development in and around a congenital mole, and the same fact is said to have been observed with regard to nævi (this is certainly very rare). When a mole becomes the seat of melanosis, besides the marked increase of pigmentation there is often an area of congestion, and, perhaps, of perceptible infiltration of the adjacent skin. Microscopically the pigment is found in the cells of the growth, sometimes as dark granules of "melanin," as well as free. Primary melanotic growths have been met with in all parts of the skin, for instance on the abdominal or chest wall, the nose, cheeks, and eyelids. They sometimes commence around and under the nails of either fingers or toes, and in this situation may readily escape diagnosis for some little time. It is stated that in a few cases the first growth occurs in the lymphatic glands.

The diagnosis of commencing melanosis is most important, since the only chance for the patient is an early and free excision. It must be remembered that the actual growth may be small, and indeed there may be apparently merely a dark stain covered, and to a certain extent concealed, by thick unaltered epidermis. If there is any suspicion of such a pigmented patch, or of the melanotic transformation of a mole, it is by far the wisest plan to resort to the knife. When once the lymphatic glands are involved, or when many secondary nodules have formed in the surrounding tissues, it is too late to expect much benefit from excision. Melanotic tumours, however, vary somewhat in malignancy. Thus I have known a case of melanotic sarcoma of choroid spread through the sclerotic into the orbit, prove impossible to remove, and six years later (although a black discharge persisted from between the eyelids) there was no sign of secondary growths.

As a general rule it may be said that in no form of malignant growth is resort to early and free excision of more importance to the patient than melanotic sarcoma or carcinoma. (*See also* Art. XXII. page 469.)

Diseases of the nails.—In the course of severe general eczema or psoriasis the nails are apt to suffer in their nutrition, to become thickened or occasionally to be shed. Less marked disturbance in their growth is sometimes met with after severe attacks of fever, such as typhoid.

In old people, especially if the care of the nails be wholly neglected, they may grow to an enormous size, becoming curved and of extremely hard consistence. The term *onycho-gryphosis* is then applied and, if much in the way, such hypertrophied nails are best removed.

A very troublesome and fairly common affection is **ingrowing toe-nail,** where the free border of the great toe-nail becomes embedded in the soft tissues; this is especially seen at the outer edge of the nail and is usually due to cutting the nail too short, or to lateral boot-pressure. Unhealthy granulation tissue tends to form over the edge of the nail, and there may be ill-smelling purulent discharge. The condition is, as a rule, exquisitely tender and painful, and may greatly hamper the patient's movements. Some relief is to be obtained by carefully thinning and scraping down the central part of the nail, by strict cleanliness, and the use of an astringent antiseptic powder to the inflamed part (*e.g.* equal parts of tannic acid and iodoform), and by the frequent insertion under the edge of the nail of a strip of linen or tinfoil. In severe cases which fail to yield to a careful trial of these measures (combined with the wearing of broad shoes or boots) an operation is called for. The patient should be anæsthetised (bromide of ethyl or nitrous oxide gas answers admirably), and the affected part of the nail should be cut off right up to the matrix with strong scissors and then pulled out. At the same time the overhanging soft tissues should be cut away with a scalpel. It is rarely necessary to remove more than one-third of the width of the nail. A dry antiseptic dressing should be bandaged on, and the patient should wear a soft slipper until healing has occurred. Relapse of the deformity is not very uncommon, and to avoid this it is absolutely necessary for the patient to wear broad-toed comfortable boots.

The nails occasionally suffer in both congenital and acquired syphilis. (*See* page 398.)

As the result of injury in children a curious disease is sometimes met with, known as **onychia maligna.** In this one of the finger-nails becomes loosened from its bed, and there is such inflammatory thickening of the matrix and surrounding soft parts that the digit assumes a clubbed form. The duration is practically indefinite unless proper treatment be adopted. This consists in the evulsion of the diseased nail, and thorough cleansing with a strong antiseptic solution, followed by the use of powdered iodoform, etc. The subjects of onychia maligna are frequently ill-nourished children, and attention to the general health and a good diet are advisable.

Peri-onychia (ungual whitlow).—As the result of inoculation with septic organisms (the access of which may have been made easy by some contusion or crush of the end of the finger), the bed of the nail or its root is liable to suppurate, a most painful and troublesome affection being thus produced. It is most often seen on the fingers of nurses, surgeons, and the like, and often takes the form of a serpiginous ulceration, which creeps round and may wholly detach the nail from its matrix ; this, however, is only effected after a long period of pain and disability, and the only speedy cure is to be obtained by removing the nail and applying antiseptic fomentations, etc. In a certain number of cases, however, the nail may be saved by persevering applications of carbolic solution, or of nitrate of silver (5 grains to the ounce). The latter is very painful for a time and blackens the nail, but certainly succeeds in checking the progress of the ulceration better than any other remedy. Even should this be successful the nail is apt to be distorted, and if removed a new one will probably grow. It should not be forgotten that primary syphilitic chancres of the fingers occur as peri- and sub-ungual sores, which may be very painful, like the preceding disease ; the indolent axillary gland enlargement is, however, an invariable sequel if the sore be a chancre.

Medicinal eruptions.—A few words must be devoted to this group, especially those eruptions which may follow the administration of drugs often used in surgery. By far the most universal form is an erythema or papulo-erythematous rash on trunk and limbs, which may, for instance, be produced by copaiba, cubebs, chloral, and a host of other drugs.

The *copaiba rash* is a very striking one, having a deep pink or red colour, confluent in patches, especially at certain sites, which are rarely exempt from the spots, namely, round the wrists and ankles. The extensor surfaces are involved more, as a rule, than the flexor ones. It appears rather suddenly, may develop in a subject who has been taking copaiba for weeks or months without previous trouble, and is attended with considerable irritation. The patient's urine will be found to become thick if floated on nitric acid, but there is no marked line just above the acid such as would reveal the presence of albumen. If the nitric acid be shaken up

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with the supernatant urine the cloudiness disappears and a quite characteristic lilac colour is seen for a time. The eruption quickly yields if an aperient saline be given and the balsam discontinued.

Cubebs may produce a very similar erythema, but ordinary doses of sandal-wood oil never cause it.

Iodide of potassium, besides causing nasal catarrh and lachrymation, and depressing the patient's mental and physical condition, may cause a pustular eruption resembling acne, and chiefly found in the regions affected by that disease. In some cases iodide (or bromide) leads to pus-secreting granulation growths, which may attain a most formidable size and appearance if the drug is pushed in ignorance of the cause of the eruption. It is especially common on the face; but it must not be inferred that iodide eruptions are really very frequent. Considering the enormous number of patients who are prescribed iodide of potassium, any skin eruption as a result is quite a rarity, and for this reason is especially liable to be mistaken in diagnosis.

XXIX. INJURIES OF BONES.

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CONTUSIONS OF BONE.

The result of simple contusions is more or less swelling of the periosteum—immediate from extravasation, which may occur also in the marrow and Haversian canals; later, from inflammatory exudation. As a rule, this traumatic periostitis soon subsides. Occasionally, tubercular or pyogenic organisms reach the injured spot, leading to tubercular osteitis in the one case, to acute suppuration, perhaps accompanied with symptoms of pyæmia, in the other. But between a blunt object and a bone the soft parts are often lacerated, as when a pistol ball strikes a bone obliquely: the injury is then compound.

Treatment.—Simple contusions are best treated by rest, elevation, and uniform compression, or by an ice-bag, until swelling has ceased; then moist warmth and, later, elastic pressure and massage will promote absorption. In compound contusions, seen early, careful disinfection and, if possible, closure of the wound must be practised; a reliable antiseptic dressing being used in any case. If the wound is, or can be rendered aseptic and closed, the probability of trouble is no greater than in a simple injury. If the wound cannot be closed, some separation of the injured bone is likely to result. If the wound suppurate, the clots in the veins leading from the dead area become infected with pyogenic cocci, a state of matters which formerly led only too frequently to embolic pyæmia. A suppurating wound with bone bare in its floor may be treated with iodoform and fomentations wrung out of 1 in 2,000 perchloride of mercury lotion. Symptoms of osteomyelitis or of general septic poisoning will necessitate removal either of the part, or of all the bone bare in the wound, until apparently healthy bleeding bone is reached in all directions.

WOUNDS OF BONE.

Incised wounds of bones are usually inflicted by sabres, axes, and the like; sometimes they are shallow or deep notches with cracked margins and a more or less widely comminuted floor; sometimes a flap of bone and of soft parts is raised, especially by sword cuts upon the side of the head, or a part containing bone may be completely cut through and severed from the body.

Treatment.—Asepsis is of first importance, so that the dangers to which the structure of bone specially exposes those suffering from septic injuries may be avoided; yet a wound containing injured bone, more or less cut off from its blood supply, must not be deluged with strong antiseptics. Whichever is selected should be used of strength proportioned to the probable infection, in small quantity and intelligently, so that each drop may do its work. Bleeding from a distinct vessel in bone is checked by plugging the orifice with a bit of catgut, or of aseptic wax; general oozing, by closing the wound and applying pressure, or if this is difficult to maintain, by draining for 24 hours. Only if sepsis is probable should loose fragments be removed. The wound should be closed like one of soft parts only; given asepsis, bone raised in a flap will probably live if the soft parts to which it is attached do so; it should be fixed by a few marginal catgut stitches, or by two or three tacks of silver wire driven through both pieces and buried.

A small completely separated part containing bone, like a last phalanx of a finger, has re-united, even after separation for an hour or more. Antiseptics should be used most sparingly in the conduct of such an experiment; periosteum should be united to periosteum, and skin to skin. A dry wool dressing should be used, and the part kept at rest. Bone exposed at the bottom of an open wound granulates and skins over like soft parts, a thin adherent and uncomfortable scar resulting.

FRACTURES.

Definition.—A fracture is a solution of continuity in a bone, suddenly made, either by contusion or flexure (or torsion). This, without the two last words, was the definition given by Richard Wiseman in 1676.

Statistics. *Frequency.*—Fractures are among the commonest injuries. P. Bruns shows, from over 300,000 cases of injury taken to the London Hospital during 33 years, that they constitute one-seventh of all injuries, are somewhat more frequent than sprains, and ten times more frequent than dislocations.

Seat.—Gurlt, using nearly 52,000 cases from the same source, calculates that fractures of the fore-arm bones formed 18 per cent. of the whole; of the leg bones, ribs, and clavicle, each 15 to 16 per cent.; of the hand bones, 11 per cent.; humerus, 7·8 per cent.; femur, 6 per cent.; foot bones, 2·9 per cent.; face bones, 2·4 per cent.; skull bones, 1·4 per cent.; patella, 1·3 per cent.; scapula, 0·8 per cent.; spinal column, 0·3 per cent.; pelvis, 0·3 per cent.; and sternum, 0·1 per cent. Shortly, fractures of the upper extremity are twice (52·60 per cent.) as frequent as those of the lower (25·88 per cent.). The trunk bones, among which he reckons the scapula (0·86 per cent.), constitute 17·66 per cent., and head and face bones only 3·86 per cent.

Age.—Using some 8,500 cases from different institutions, Bruns finds that the frequency-curve rises in the first, second, and third decennia, then falls steadily—reaching in the fifth the level of the first. In the ninth period nineteen times fewer fractures occur than in the first. But, in proportion to the number living in each period, the maximum number of fractures occurs between 30 and 40, the minimum between 0 and 10; and old people (70 to 90) are more liable to fractures than young (0 to 20).

Seat in relation to age.—Among children, fractures of both bones of the fore-arm, of the radius alone, of the lower epiphysis of the humerus and of the collar bone are most frequent; in the lower extremity, fracture of the shaft of the femur is most common. Among adults fractures of limbs are frequent, those of the upper being only slightly more common than those of the lower; to these are added fractures of the skull and bones of the trunk—especially of the ribs—while fractures of the patella reach in adults their highest point. In old age fractures of the lower extremity become specially frequent; that of the neck of the femur gaining an absolute majority in patients over 80 years of age. Fractures of the ribs, of the upper ends of the humerus, and of the radius are also frequent.

Sex.—Bruns, from 3,742 cases, finds that males are to females as 3·8 to 1. But as there are more females than males living in each decennium after the first, the correct proportion is M : F : : 4·5 : 1. Speaking of the absolute numbers of fractures occurring, and not allowing for the numbers living at each period, about twice as many fractures occur among males up to 20, about eight times as many from 20 to 50, about twice as many from 50 to 70, after which age about three times as many fractures occur among women as among men (women are in excess by 14 per cent.).

Season has not a very marked effect upon the total number of fractures occurring; each season has its special dangers connected with its special exercises and occupations. The influence of season is least marked in adults; children suffer most in summer, old people in winter.

Occupations naturally have a marked effect in rendering those pursuing them more or less liable to fractures; but extended statistics of different trades are wanting.

THE GENERAL ÆTIOLOGY OF FRACTURES.

Predisposing causes.—These either tend to weaken certain or all bones, or they bring about exceptional exposure of the bones to injury. The influence which *age, sex, season*, and *occupation* have upon fractures has just been pointed out. It is obvious that the position, form, strength, and elasticity of a bone are most important. Lastly, there are many pathological conditions which diminish the strength of bones.

Physiological conditions. (1) *Position.*—A limb bone, situate in an outlying part, used instinctively to protect the central parts, is far more liable to injury than a trunk bone; the trunk, again, is more difficult to protect, and is less sedulously protected than the head. A limb bone in the line along which force travels towards the centre is more liable to fracture than one not so placed—the radius is more often broken than the ulna, and the tibia than the fibula; though it is true that, in the latter instance, after the tibia has yielded, the fibula also often breaks, and that a twist of the foot, by diverting the line of force, may lead to a fracture of the fibula alone. The bones of the fore-arm and hand, leg and foot are more exposed to direct injury than are the proximal portions of limbs.

(2) *Form.*—A straight column resists crushing more strongly than a curved one, and all long bones are curved. Their structure becomes specially weak when an angle is developed, as between the neck and shaft of the femur. The longer and more horizontal the neck, the more easily does it break under longitudinal strain.

(3) *Strength and elasticity.*—Bones vary greatly in those properties which enable them to resist violence. The ribs, fibula, and clavicle are the most elastic, *i.e.* they may be bent farthest without breaking, and with subsequent perfect recovery of their form. The humerus among long bones and the cancellous bones are least elastic. The percentage composition of fully-formed bone, compact or cancellous, seems to be practically the same at different ages, and in the two sexes. Given the same form and size, greater weight means greater strength. Increase in length without proportionate increase in girth and weight, implies relative weakness. We see this in the great frequency of fractures between two and four years, when falls are common, and the length of the limb bones is greater in proportion to their girth than at any other period of life. So long as a bone is growing actively, it is relatively more vascular and, therefore, more porous and weaker than subsequently. On the other hand, in old age the bones undergo eccentric atrophy as the strain upon them lessens. Between the periods of growth and degeneration, when their strength is most needed, the bones are at their strongest. In spite of this, according to Bruns, 43·8 per cent. of all fractures occur between 30 and 60, while 26·1 per cent. occur between 1 and 30, and 29·8 per cent. between 60 and 90.

When the bones of a limb are arranged to form a single rod, this naturally breaks at its weakest point, *e.g.* the clavicle; but in many falls on the hand the limb is bent so that the brunt of the violence is borne by the more distal bones, especially the radius. Although we have now many careful measurements of the resisting power of the different bones at various ages to force applied in the long axis of the bone (crushing or tearing), at right angles to it (bending), and at the end of a short lever fixed at right angles to the long axis (torsion), our knowledge of the physics of the skeleton is far too imperfect to enable us to calculate the result of violence transmitted along a more or less bent limb, with all its muscles tense and acting like springs to ward off violence from the centre.

Pathological conditions.—These weaken all or certain bones, so that they break under the application of slight force. Fractures thus occurring are often spoken of as "spontaneous fractures"—a contradiction in terms; for "fracture" implies that some force—*e.g.* the weight of the limb—has acted; a bone eaten right through is not "fractured." Fractures of this kind are complications arising in the course of certain morbid processes, of which, sometimes, they are the first noticed symptom.

(1) *Atrophy of bone* owns many causes. It would be best to name the varieties from their causes; but in some the ætiology is unknown, in others it is complex. The latter seems to be the case in senile atrophy. In old age the vitality of the cells of the body becomes exhausted, with impaired functional activity and proneness to degeneration as consequences; the general feebleness and malnutrition of old age result. As muscular strength fails, the bones are subjected to less and less severe strains, and absorption of material no longer required occurs. Possibly degeneration and imperfect performance of function by the bone-cells increase the weakening. The size of a bone remains unchanged, but it becomes progressively lighter and more porous, owing to increase of all medullary spaces at the expense of the solid bone. Senile atrophy affects the long bones chiefly. It is more marked in women than in men, probably on account of their more sedentary lives. Its changes may appear early or late, in accordance with the health and habits of the individual; in some degree they may be regarded as physiological, though old bones are not uncommon which have escaped them entirely. But when they appear early in life, or advance out of proportion to the age of the individual, they must be viewed as pathological. The great frequency of fracture of the narrow part of the neck of the femur in old people, especially women, and from slight violence, is due to weakening of the cancellous tissue of the femoral neck by senile atrophy.

Atrophy from disuse is common. When, for months or years, a limb is kept at rest by paralysis, or disease of bone or joint, all the tissues waste and become fatty; the bones may do so to an extreme degree, being easily cut with a knife. Here the first element in the ætiology is impaired blood supply, due to non-performance of function by the muscles, each contraction of which causes an affluxus shared by the bones. When, as is commonly the case, disuse begins in childhood, impaired growth adds greatly to the effect of atrophy. It is possible that in these cases there may be some trophic influence at work also.

Thinning of a bone by the constant pressure of an aneurysm or simple growth may end in fracture.

Nerve influence, or the absence of it, is almost certainly concerned in producing the atrophy associated with tabes, perhaps, also, in that of chronic brain disease, especially general paralysis. The frequent occurrence of fractures, commonly multiple—36 in one case—in general paralytics has long been known. Formerly attributed to violence on the part of attendants, it is now recognised that in these patients the bones undergo eccentric atrophy, so that sometimes even limb bones may be cut with a knife or crushed by the fingers. The ribs are first and chiefly affected, then the limb bones and other bones of the trunk and head. The naked eye shows the compact layer to be porous and wasted, soft and dark; organic matter is in excess, and lime is deficient. The causes of fracture, when detected, have often been insufficient.

But the evidence in favour of atrophic influence is far stronger in tabetic cases, to which Weir Mitchell drew attention in 1873. Charcot in 1874 published the case of a woman who died at 61, having shown signs of tabes for 26 years. During the last 18 years she had sustained painless fractures, from slight violence, of both scapulæ, the left clavicle, both bones of both fore-arms, and the left femur; spontaneous dislocation of both hips and one shoulder also occurred. All the fractures had united with more or less shortening, but with an excessive amount of callus. Many cases have since been described; they are not very rare—many fractures in tabetics being missed, owing to the fact that they occur before ataxy develops. One painless fracture is frequently succeeded by others, and the cause is usually disproportionate to the effect; the lower limb bones, especially the femur, are affected more commonly than those of the upper limb. Union generally, but by no means always, occurs in about the usual time, and often with excessive callus; refracture may follow feeble union. Naked eye and microscopic examination have demonstrated in tabetic bones a simple eccentric atrophy, without sign of inflammation; but Regnard stated, as the result of his analyses, that the proportion of organic to inorganic constituents was more than reversed (76 to 24 instead of 33 to 66), which would not be the case in a simple atrophy. It is the occurrence of this atrophy in tabes, before muscular weakness, or even before inco-ordination appears, which renders a trophic influence of the central nervous upon the osseous system so probable. (*See also* page 897.)

(2) *Fragilitas ossium.*—This heading includes certain cases in which the bones are unduly fragile—in which an atrophy has been assumed, but has never been demonstrated. It is best to admit that we have no knowledge on the point. A good many cases are recorded, in which many fractures from trifling causes have occurred in members of three or four generations of a family, the predisposing condition of bones being inherited. In other instances, this liability to fractures has appeared, without heredity, in early childhood or later. The tendency has generally lasted throughout life. As a rule, firm union has occurred in the usual time; occasionally healing has been delayed. In these cases no general disease and no other skeletal defect has been detected. Example: A boy of 13 had sustained fourteen fractures from slight falls and twists—of the femur, of the fore-arm, of the ulna four months later, and, again, after four months, of the right humerus, radius, and tibia; all united in 3 to 5 weeks. He subsequently sustained six other fractures. This boy's mother had sustained five fractures of the left, and one of the right

thigh, all from slight violence; the last preceded by severe pain in the limb. Her brother, before he was thirteen, had broken one femur nine times, the other twice, his arm once, and he is said to have "dislocated his hip" once. (*See also* page 898.)

(3) *Inflammation of bone*, leading to necrosis, abscess, or caries, may so weaken it as to lead to fracture from slight violence.

Necrosis is the commonest of these predisponents. Almost all the patients are young—under 21—and give a history of acute infective osteomyelitis and periostitis, ending in the formation of a sequestrum, which, whatever its length, embraces the whole, or nearly the whole circumference of the shaft. The femur is in these cases by far the commonest seat of fracture, which usually occurs during the second month, when the connection between the living and dead bone is eaten through. If now the sheath, together with any living portion of the shaft which may remain bridging across the gap, is not strong enough to support the weight of the limb, a fracture may occur, even whilst the limb is supported by a splint; and is much more likely during a dressing, a passive movement (Fig. 201), or a sequestrotomy. Fracture of a fixed total sequestrum is very rare; as also is fracture of the sheath, so long as the sequestrum lies within it. But after the removal of the sequestrum, bending and fracture of imperfect sheaths are more common.

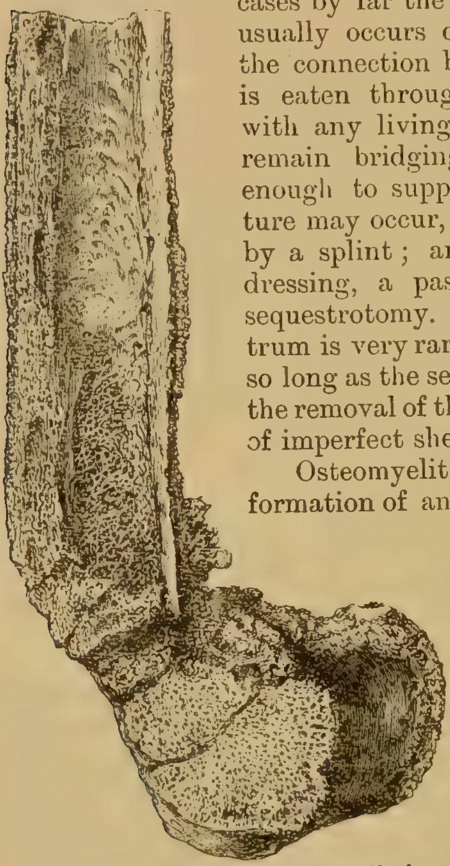


Fig. 201.—A longitudinal Section of the lower End of a Boy's Femur. He was admitted for necrosis of the femur and suppuration of the knee joint, and the tibia appeared to be dislocated backwards; but after amputation it was found that the femur had been broken, probably by passive movement, about 1 in. above the epiphysial line, and that the fragments had been united with angular deformity forwards. A considerable sequestrum of the popliteal surface, still adherent, is shown in section, its lower end lying in a cavity in the lower fragment. The bone is excessively porous, and deposit of new bone is wide-spread. (University College Museum, No. 277.)

Osteomyelitis ending in abscess without the formation of any, or any considerable sequestrum, rarely leads to a spontaneous fracture. The femur has most often been the seat of the injury owing to an abscess in the shaft, close to, but not involving the epiphysial line.

Tubercular caries rarely leads to fracture of the shafts of long bones, into which it seldom penetrates deeply; but Billroth saw the tibia thus eaten through. The ribs, the vertebral bodies, and the odontoid process suffer more commonly.

Syphilitic caries, the result of gummatous periostitis and osteomyelitis, is another cause of spontaneous fracture, which is, however, very rare in proportion to the wide distribution of syphilis. The bones of the upper limb, especially the clavicle, are the commonest seats. A history of syphilis, of bone pains preceding the fracture, of a periosteal swelling—though, according to Chiari, a central gumma may give no superficial sign of its presence—the existence of signs of syphilis, and the satisfactory results of antisyphilitic treatment, constitute the evidence upon which a diagnosis may be made. Gummatous infiltration of shafts of long bones near to, but not involving the epiphyses, may occur in infants the subjects of congenital syphilis, and may lead to fracture before, during, or after birth.

(4) *Rickets* is a very important predisponent to fracture in children. The practical result of the disease is that the material laid down by the periosteum and epiphysial cartilage undergoes true ossification irregularly or not at all; it forms a relatively weak, friable, wide-meshed, calcified tissue (spongioid) which encases the original well-formed bone of the shaft. The latter, however, undergoes progressive absorption in the formation of the medullary canal, and thus the bone loses its firmest portion. During the height of the disease, when the bones contain the largest proportion of this soft material, greenstick fractures are commoner than are complete, and arise from slight violence; but in the early stage and stage of recovery, when the proportion of well-formed bone is considerable, the fractures occurring are said to be more frequently complete. After recovery from rickets the bones are often unduly dense and strong, though small. According to Guersant, rickets is present in one-third of all fractures seen at the Hôpital des Enfants Malades in Paris; and many cases of infraction are never taken to a hospital. Multiple fractures are not uncommon; sometimes several occur in one bone, which may ultimately be greatly deformed. Union is delayed, and may not occur at all during the height of the disease; usually it is effected slowly, and with the formation of a large callus tumour, chiefly in the concavity. The quality of the callus is that of the bone of the same period. (*See* page 363.)

(5) *Osteomalacia* affects only adults and, typically at all events, only women—especially multiparæ. It is characterised by decalcification of bone and subsequent absorption of the animal basis by the hypertrophying marrow. The bones become thin-walled shells full of red marrow; indeed, all bone may disappear, and only the periosteum remain around the marrow. The change usually spreads more or less widely over the skeleton from the pelvic bones. So long as crisp bone remains, fractures are common and unite well; later, all kinds of bendings result. The pelvis, ribs, and limb bones are chiefly affected. (*See* page 898.)

(6) *New growths.*—Of simple growths, only chondromata have given rise to fracture. They are less commonly central than subperiosteal, but the central are more likely to lead to fracture. Thus, Gross found two fractures among ten central chondromata, but only three fractures among sixty-three subperiosteal growths. Very rarely, central chondromata seem to undergo cystic degeneration,

and the cysts cause "expansion" of the bone, with great thinning or complete absorption in places of the compact tissue—spontaneous fracture naturally resulting.

Malignant growths of bones are primary (sarcoma) or secondary (sarcoma or carcinoma). According to Gross, primary central sarcomata, affecting chiefly the cancellous ends of long bones, form more than half the cases, and spontaneous fractures arise in about 20 per cent. of all central primary sarcomata. They occur in only 5 per cent. of the primary peripheral growths. In both cases, round- and spindle-celled growths tend more strongly than giant-celled to induce fracture. In primary cases a tumour (Fig. 202) is always present before fracture occurs. Difficulties in recognising the cause of fracture arise from the thickness of the covering of the bone affected—*e.g.* about the hip—and from the possibility that displacement and hæmorrhage may have caused such swelling as is felt. The femur is most commonly affected, then the tibia, and then the humerus.

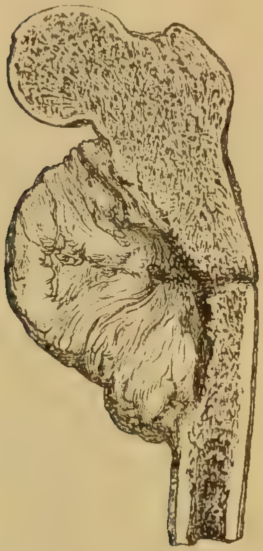


Fig. 202.—A Primary Sarcoma of the Femur of peripheral origin which has led to Fracture of the Bone. (King's College Museum.)

Osteomalacia carcinomatosa is the name which has been given to a very rare disease, clinically like osteomalacia, characterised by a diffuse malignant infiltration of the medulla, commencing usually in the vertebral bodies, and spreading thence over more or less of the skeleton without forming distinct tumours. The fractures and bendings are most common in the spine.

Secondary malignant growths, usually carcinomatous and central, cause spontaneous fractures more commonly than primary. Bruns found that, among 71 fractures from secondary growths, the primary growth had been mammary in no fewer than 59 instances—a predominance, probably, due to the long course of many mammary cancers. Of the 71 fractures, 34 occurred in the femur, 20 in the spine, 3 in both femur and spine, 11 in humerus, 1 each in humerus and spine, humerus and both femora, and clavicle. As a rule, no tumour which can be felt develops in these cases—the growths being small, central, and often numerous; or diffusely infiltrating the medulla. The diagnosis then depends upon the presence or history of removal of a primary growth, and the occurrence of a fracture from an inadequate cause.

Formerly cancer was regarded as a cause of fragilitas ossium, but research has shown that almost invariably a spontaneous fracture in cancer is due to a secondary growth. It would not be surprising if a condition like senile atrophy were induced by the long illness which sometimes precedes death from cancer, but indubitable cases are very difficult to find.

Improbable as it seems, there are four or five well-recorded cases of union occurring after fracture from malignant disease, the union soon giving way again before continued growth of the tumour.

Hydatid cysts in bones are a rare predisponent to fracture. Bruns collected 11 cases. Fracture seemed usually to have occurred early, before anything amiss had been noticed, though sometimes the inadequacy of the cause gave rise to suspicion. The diagnosis was generally made at an operation for pseudarthrosis or at the opening of an abscess. Among Bruns' cases the limb was saved only in three instances—twice by resection of bone and removal of the cysts, once by introduction of a seton, which induced suppuration, elimination of cysts, and union of fracture. In the rest death occurred from exhausting suppuration, or life was saved by sacrifice of the limb.

As traumatic separation of epiphyses will be included under fractures, it is right here to mention that spontaneous separation occurs commonly in the course of primary infective periostitis and osteomyelitis, as an occasional result of hereditary syphilis, and rarely in scurvy rickets. Scurvy, as at one time met with, was said to be a cause.

Determining causes.—A fracture is always due to the action upon the bone of force—either external violence, including the weight of the part, or the action of muscles. External force seems to be a far commoner cause of fracture than muscular action; yet, were it not for muscular action fixing bones, it is probable that many a fall which produces a fracture would fail to do so. Hence the remarkable escapes of drunken and insensible people from fractures of the limbs.

External force acts directly or indirectly, according as it produces a fracture at the point upon which it first acts, or at a distance from this point. Thus, when a cart-wheel passes over a limb, breaking the bones beneath it, the fracture is one from direct violence; but when the clavicle or radius is broken from a fall on the outstretched hand, the violence has acted indirectly.

Whether applied directly or indirectly, force exercises either traction or compression, torsion being a kind of traction. Pure *traction fractures* are rare, and include only the tearing off of processes by tendons and ligaments. Pure *compression fractures* or crushes of bone are not uncommon, but to obtain the effect of pure compression, the possibility of bending must be excluded; otherwise the fracture becomes in part a flexion fracture, in which compression is exercised on the concave and traction on the convex side of the bending bone. In *flexion fractures*, the break always occurs at the point of greatest tension, and begins on the convex side, at the apex of the angle. Numerous experiments and examinations of fractures have enabled P. Bruns to construct diagrams (Fig. 203) illustrating the more usual results of force so applied to a bone as to cause it to bend beyond its limit of elasticity. They show the production of infraction (A), transverse (B), and oblique fractures (C), and fracture with splitting off of a single wedge-shaped piece (D). The advantage of a long diameter in resisting flexion is obvious; the weights required to break the tibia by acting on its crest and on its inner surface are as 17 to 12.

The effects of *direct violence* depend upon the weight, momentum, consistence, form, etc., of the injuring body; upon the strength, mobility, protection by soft parts, and such like conditions of the bone. It compresses (crushes) a supported bone, bends an unsupported. The soft parts covering the bone are necessarily damaged—bruised, torn, stripped off, crushed, and killed—according to the nature of the accident, and the form and position of the bone.

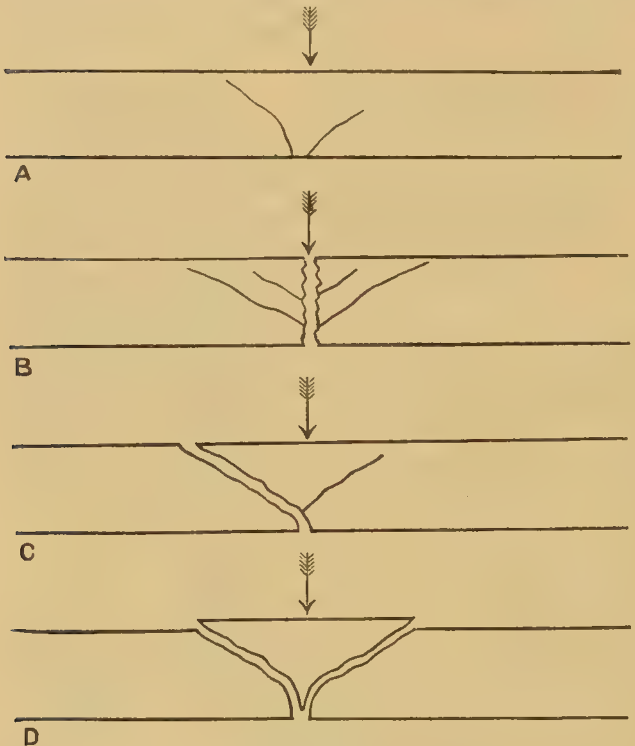


Fig. 203.—Diagrams illustrating the commoner Results of Flexion. (P. Bruns.)

The action of *indirect violence* is by no means so simple, and it is not possible clearly to explain why in similar accidents such dissimilar results ensue as a fracture of the os calcis, of the base of the skull, or of some intermediate bone, from a fall on the feet. The absolute and relative strengths of the bones concerned, the amount of force at work (body-weight × velocity as a rule), the line of force, the positions of the various bones with regard to this line, the effect of muscular action, the freedom of the various bones to move on each other, the resistance offered by the surface upon which the patient falls—these are some of the points about which exact information is indispensable before an accurate calculation of the result can be attempted; but inquiry along these lines will often lead to a helpful hypothesis.

Indirect violence may act by producing one or other of the following effects:—(1) Pure flexion, as when, the lower part of the leg being fixed, the body falls to one or other side. The mechanism is similar when a malleolus is broken off by pressure of the astragalus against its tip in extreme supination or pronation of the foot.

(2) End-to-end compression of a bone, causing increase of its natural curve and fracture at the point of greatest tension and weakness. Thus, in falls on the hand the radius is often compressed between the capitellum and the carpus; in falls on the foot, the tibia; in false steps, the femur—the neck being fractured. (3) Compression of a bone, flexion being prevented, results in the crushing of cancellous bones, and in the splitting up of large epiphyses by the driving into them of smaller and denser portions. Thus arise many fractures of the femoral neck and trochanter. (4) Force transmitted across a joint through tense ligaments may tear off the bone into which the ligaments are inserted. The anterior radio-carpal ligament is credited by some with this action in producing Colles' fracture. The malleoli, epicondyles, styloid processes, and the like are sometimes thus torn off. (5) Torsion fractures or, at least, fractures in the production of which torsion takes a share, do not seem to be rare. In falls, after a limb has reached the ground and has become fixed against it, the body frequently swings round towards one or other side. Occasionally, whilst the foot is immovably fixed, the body, to avoid a sudden danger, is turned violently round; or machinery may so twist a limb upon the body, or a surgeon may execute such a movement in endeavouring to reduce a dislocation.

The soft parts in complete fractures from indirect violence often escape without obvious damage; but the muscles usually, the skin not uncommonly, the vessels and nerves rarely, are lacerated by the ends of the fragments. Such damage as is done to these parts is inflicted from within and by the fragments—a pointed piece of bone being often found sticking through a hole in the skin. The contrast in this respect between fractures from direct and indirect violence is important, on account of the slighter injury to and less danger of sloughing of the soft parts.

Muscular action, voluntary or involuntary—*e.g.* epileptic—is said to be responsible for less than 1 per cent. of all fractures, but, as before said, it probably shares with external violence the credit of producing many more. Usually muscular action tears off a process or an epiphysis—as the coronoid process or the tuberosity of the calcaneum. A considerable proportion of fractures of the patella is due to sudden contraction of the quadriceps to prevent a backward fall; but in most of these cases the bone is probably bent and cracked across the condyles of the femur, *i.e.* it is a mixed traction and flexion fracture. The ribs have been broken in fits of coughing, also the sternum in a child of two years; several times the sternum has been torn in two by the action of the abdominal muscles in labour. The cervical spine (4th or 5th vertebra) has been broken five or six times by sharp extension, practised to avoid striking the bottom in diving into shallow water. The clavicle has not uncommonly been broken in powerful actions of the most varied kinds. But the most remarkable fractures from muscular action are those of the long bones. Gurlt collected 95 of these, and found that the humerus was the seat in 57, the femur in 25, the legs and fore-arm bones in 8 and 5 instances respectively. The humerus has usually been broken either in lifting a weight or in an action like that of throwing a stone or striking a blow which misses its object. In the latter case the shoulder muscles stop the upper end of the bone, whilst the rest of the limb moves on, a fracture resulting, which has been compared to that of a swinging stick just beyond the grasp of the hand. The fore-arm bones have rarely been broken by forcible pronation and supination, as in wringing clothes. In many of the fractures of the femur attributed to muscular action, the momentum of the body turning upon the limb has introduced the question of torsion; and it seems likely that the same may be said of the much rarer fractures of the tibia. Strong action of the biceps has occasionally torn off the upper fibular epiphysis.

All fractures such as the above should be carefully examined for some pathological predisposing cause; only such cases are classed as fractures from muscular action in which no such predisponent is found.

VARIETIES OF FRACTURE.

Classification.—The most important divisions of fractures—simple, compound, and complicated—are based upon the condition of the soft parts; less important varieties rest upon the condition of the bone. A fracture is *simple* when there is no wound of the soft parts admitting air to the fracture. The presence of a wound not communicating with the fracture does not render the latter compound, but practically it is impossible to draw the line when a wound exists over a fracture. A fracture is *primarily compound* when a wound, rendering direct infection possible, is inflicted at the same time as the fracture; it is *secondarily compound* when such a wound is formed later, as the result of late perforation of the skin by a fragment, of an incision, or of the separation of a slough. The later a fracture becomes compound—the more freely the injured tissues are granulating—the less dangerous is sepsis. Either a simple or a compound fracture may be *complicated* by great bruising and extensive stripping up of skin from the fascia, by unusual laceration of muscles, by wounds of many or important arteries or veins, by injury to nerve trunks, by tearing of ligaments, and by dislocation of the fractured bone.

No description of the naked-eye appearances due to the above lesions is necessary. Blood-stained fluid soaks far and wide, and, clotting on torn surfaces soon conceals their structure; but round about the broken bones the blood generally remains fluid for days. The conditions of the bone, upon which varieties are based, deserve careful study.

Condition of the bone.—The bone may be broken completely through into two or more distinct pieces (*complete fracture*), or the solution of continuity may be incomplete (*incomplete fracture*). A fracture is complete, even though the periosteum be untorn, and there be no displacement. Before an epiphysis has united, violence may detach it completely or incompletely, thus constituting the important group of *traumatic separations* of epiphyses.

Incomplete fractures are much rarer than complete; the greenstick fracture is the most important variety. It is seen typically in the shafts of long bones, as the result of bending. A partial transverse fracture starts on the convex side and traverses the compact layers, whilst the corresponding layers in the concavity are bent; longitudinal fissures often run up and down from the transverse fissures between the broken compact layers (Figs. 204, 205, and 237). Sometimes two oblique fissures start close together on the convexity, and diverge as they pass towards the concavity (Fig. 203, A); rarely the fractures start in the concavity. The periosteum is generally intact. Infraction is probably most common in the clavicle, then in the fore-arm bones; it is rare in all others. The great majority of cases occur between 3 and 10 years, especially in the seventh and eighth years. Indirect violence is the usual cause, but direct violence sometimes results in infraction. Deformity is the chief point upon which the *diagnosis* of greenstick fracture rests; the bone is seen and felt to be bent, and the results of the bending upon the shoulder or other part are evident. Movement is not free in all directions, and may be

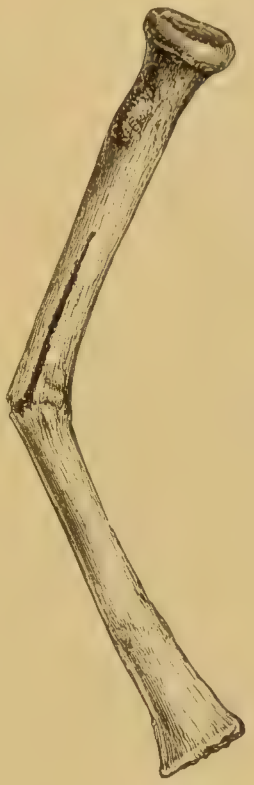


Fig. 204.—A Greenstick Fracture of the Radius of a Child. The fore-arm was bent towards the radial side; a transverse fissure divides the inner half of the bone about its centre, whilst the outer half is bent. The upper fragment is split longitudinally for nearly half its length, starting from the fracture. (King's Coll. Museum.)

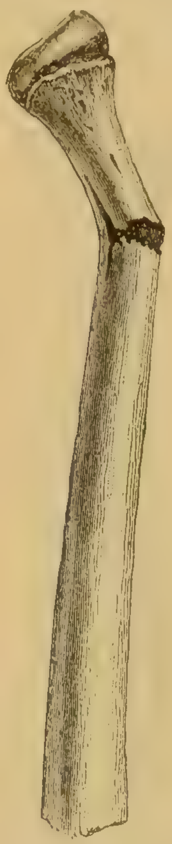


Fig. 205.—A Greenstick Fracture of the lower End of the right Ulna of a Boy, aged 14 years, due to a machinery accident. The shaft has been bent inwards 1½ in. above its lower end; a transverse fracture extends through the outer three-fourths of the bone, whilst the inner fourth is bent. The mode of formation of a longitudinal fissure is clear. The lower epiphysis was partly, and the lower epiphysis of the radius was completely, separated. (Charing Cross Museum, No. 355.)

absent; but it is generally obtainable in that direction which will increase the deformity and open more widely the fissure on the convexity. Crepitus is slight or absent; local pain, swelling, and loss of function are more or less marked. As to treatment, that for the corresponding complete fracture should be used. Deformity should always be corrected—an anæsthetic and a good deal of force being sometimes necessary—when it is such as to interfere with the utility of the part, but in a labourer's child a firmly-fixed infraction of the clavicle need not be forcibly undone, especially as the deformity tends to diminish with growth. Function is resumed earlier after incomplete than after complete fracture; yet Betz, who had an opportunity of comparing the two in the same child, forty days after the injury, found the callus much better developed and stronger in the latter than in the former.

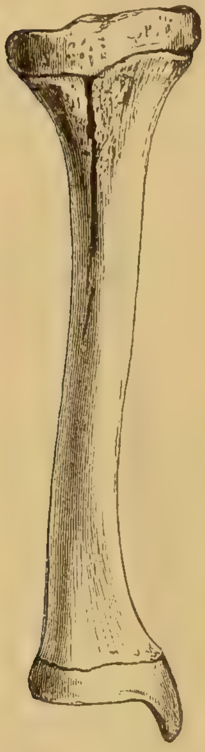


Fig. 206.—Tibia of Child showing a longitudinal Fissure in upper Half of Shaft, starting below the Epiphysis. No history. (St George's Museum, Series I. 208. Pick's "Fractures and Dislocations.")

Isolated examples of typical infractions due to machinery accidents have been described in the fore-arm bones of young men, and still more rarely of other bones of adults—*e.g.* of the acromion in a man aged 54 (P. Bruns).

Infractions and bendings of flat bones are seen almost solely in the bones of the roof of the skull. Spoon-shaped depressions form on the frontal or parietal bones, as a result of long pressure against the sacral promontory in cases of contracted pelvic brim; they are said to show bending without simple fracture. Similar depressions from blows after birth always exhibit radial fissures. The depressed bone is said gradually to rise. Very rarely in adults, when the diploë is thick, the external table has been depressed without affecting the internal.

Fissures not passing completely through or across a bone form the second variety of incomplete fractures. They are common in the flat bones, especially in those of the skull; sometimes as single clefts running in various directions, again as numerous cracks, radiating from a centre, or running more or less circularly round a depressed area. Incomplete fissured fractures of long bones may be divided into those which start from the joint ends (Fig. 315), perhaps extending into the shaft, and those involving the shaft only (Fig. 206), These fissures are impossible of recognition in simple injuries, so we do not know how frequently they occur. Usually a fissure is recognised only in compound injuries by the nail or some pointed instrument (when care must be exercised against mistaking a line of suture for a fissure), or its presence is inferred from the occurrence of bleeding, or of bruising at a point which has not been directly injured. Fissures of long bones probably heal well. In the skull it is not uncommon to find that sufficient callus has not been thrown out to close the narrow cleft, the edges of which are rounded off and perhaps connected by a delicate bridge here and there. That fissuring of a joint-end renders a contusion of a joint more serious, and opens the way to possible hindrances to complete recovery of movement, is clear.

Complete fractures.—In these the bone is completely divided into (A) two or (B) several fragments:—(A) In the first case, the line of fracture may be (1) transverse, (2) oblique, or (3) longitudinal; and into this group fall fractures of bony processes.

(1) *Transverse fractures* are those in which the line of fracture is, on the whole, at right angles to the long axis of the bone (Fig. 217); the surfaces are sometimes smooth, more often finely or coarsely toothed. This direction is much commoner than the longitudinal, but not nearly so common as the more or less oblique. Transverse fractures occur chiefly in flat and short bones, in the ribs, in the spongy ends of long bones, and in the shafts of the long bones of children. They are generally due to direct violence bending the bone, but may arise from end to end compression, causing the same result. Displacement of the fragments is less liable to occur than in more oblique fractures, but should it occur in deeply-toothed fractures, it may be impossible to reduce the fragments with accuracy. Close interlocking of the teeth may so limit movement of the fragments as to cause some difficulty in diagnosis. Union occurs readily.

(2) *Oblique fractures.*—The line of fracture crosses the long axis of the bone at an acute angle in any direction. Including, as it does, all fractures between the purely transverse and purely longitudinal, and occurring chiefly in the shafts of long bones, this group is by far the largest. The best examples are seen in the femur (Fig. 228) and tibia, and are due to bending, produced generally by indirect violence passing along the length of a bone, or applied at right angles to the shaft, one end being fixed.

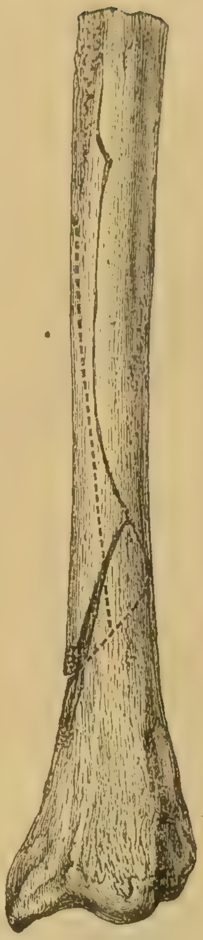


Fig. 207.—A spiral Fracture of the right Tibia seen from the Back and outer Side. Two fissures start on the posterior edge some 2 ins. from the malleolus; the spiral winds up across the inner surface (dotted line), then across the outer surface on to the posterior aspect; here, some 3 ins. above the starting point, it is joined by the nearly vertical fissure which lies entirely on the posterior surface. From the point of meeting a fissure 5 ins. long ascends on the posterior surface and another (dotted line) not quite so long ascends on the inner surface from the spiral fissure. No history. (University College Museum, No. 804.)

Oblique fractures of joint ends—especially of the lower ends of humerus and femur—arise from indirect violence, driving the articular ends together, so that its maximum effect falls on one edge or half, which is thus split off (Fig. 292). The more oblique the fracture and the smoother its edges, the greater the tendency to displacement. The direction of the fracture largely controls the displacement, one surface serving as an inclined plane upon which the other naturally glides. A common result of an oblique fracture is the formation of two fragments, each of which looks like the mouthpiece of a clarionet—this is the *fracture en bec de flûte*, and is specially frequent in the tibia (Fig. 314) and femur.

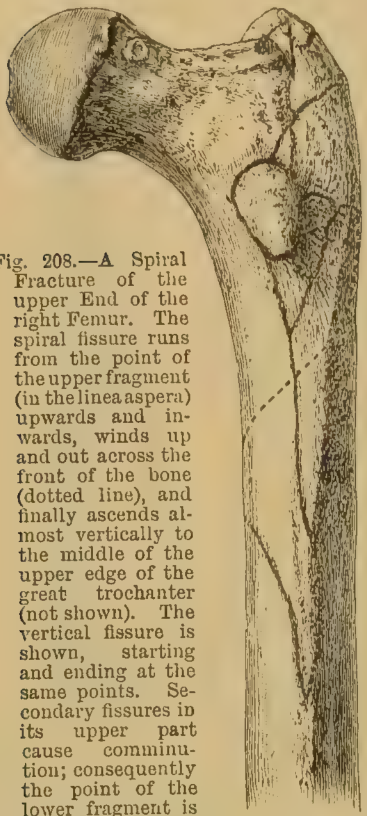


Fig. 208.—A Spiral Fracture of the upper End of the right Femur. The spiral fissure runs from the point of the upper fragment (in the linea aspera) upwards and inwards, winds up and out across the front of the bone (dotted line), and finally ascends almost vertically to the middle of the upper edge of the great trochanter (not shown). The vertical fissure is shown, starting and ending at the same points. Secondary fissures in its upper part cause comminution; consequently the point of the lower fragment is where the dotted line ends. (University College Museum, No. 263, *a.*)

The *spiral fracture* is a much rarer variety, and is seen chiefly in the lower half of the tibia (Fig. 207) and upper half of the femur (Fig. 208). A fissure winds round the shaft more or less obliquely, sometimes including six or eight inches between the point of starting and the point in the fissure which is vertically above it; these two points are connected by a practically vertical fissure. From one angle or the other, either the vertical or the spiral fissure is usually continued, often into a neighbouring joint. The long sharp points which result are very liable to pierce the skin and to cause serious injury to deeper parts. Bruns states that a number of the recorded cases, though simple, have been brought to light by the occurrence of suppuration, gangrene, or other accident. Koch and Filehne, by applying torsion to fresh bones, showed that a spiral fracture could thus be produced with great constancy, the fissure ascending to the right or left according as the forceps holding the free end were carried to the right or left.

(3) *Longitudinal fractures* are very rare in long bones, if the term be used to include only cases in which a bone is split from end to end by a fissure in or parallel to its median plane. There are many instances in which the patella, and a few in which a vertebral body and the sternum have been thus fractured; but Krönlein could find recorded only one case in which a long bone—the tibia—was split from end to end by the kick of a horse. In this case, after subsidence of early swelling, a longitudinal groove could be felt, and,

later, this filled with callus. A *post-mortem* two years later confirmed the diagnosis. A case of fracture of the clavicle from the acromial end to a point just external to the sternal end, recorded by Chassaignac, deserves to be mentioned. Krönlein saw the humerus longitudinally split in a man of 27, who was endeavouring to lift a heavy ladder. The subcutaneous fracture suppurated, necrosis of the fractured surfaces resulted, and the diagnosis was made through incisions at either end of the arm. Recovery was complete only after two years, the shoulder and elbow being completely ankylosed. Krönlein recorded also three cases of longitudinal fracture of the proximal phalanx of the fourth and fifth fingers; union was slow, and a good deal of stiffness remained. A few almost complete longitudinal fissures of long bones have been figured. This fracture has not been produced experimentally. The points which would probably characterise a longitudinal fracture are:—Early swelling along the whole length of the bone; possibly the detection of a longitudinal fissure in a subcutaneous part; no angular or rotatory deformity, no shortening; crepitus throughout the length of the bone on pressing the fragments together, on moving them upon each other, or in rotating a distal portion of the limb; swelling and tenderness of the joint at each end of the broken bone. The prognosis is necessarily less favourable than in less extensive fractures.

Separation of small fragments of bone.—The breaking off of a bony process undoubtedly constitutes a complete fracture; but it does not destroy the continuity of the weight-bearing or force-transmitting portion of the bone, and this has often led to the placing of these fractures among the "incomplete."

The amount of bone broken or torn off varies from a thin surface layer to which a tendon is attached, to a whole process with more or less of the adjacent bone. The cause may be direct violence—as when the acromion is broken by a fall on the point of the shoulder, the coracoid process by a wheel passing over its point, or the spine of a vertebra by a blow. Or the cause may be indirect violence, of which the following are examples—fracture of the head of the radius, or of part of the lower end of the humerus from a fall on the hand; or tension of a ligament, stronger than the bone to which it is attached, which it then tears off, *e.g.* fracture of the epitrochlea in dislocations of the elbow, fracture of the styloid process of the ulna in Colles' fracture, and many other small fractures accompanying dislocations; or, lastly, fracture of the tuberosity of the os calcis, or of the great tuberosity of the humerus, from tension of muscle, probably always due to contraction.

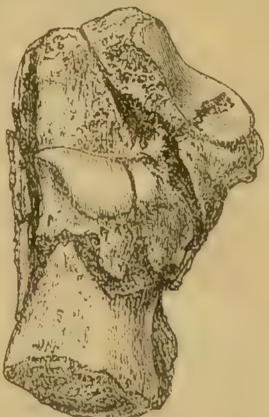


Fig. 209.—Extensive Comminution of Os Calcis, from a lunatic who jumped from a third floor window and alighted on his feet. The fracture was simple. (Charing Cross Museum, No. 421.)

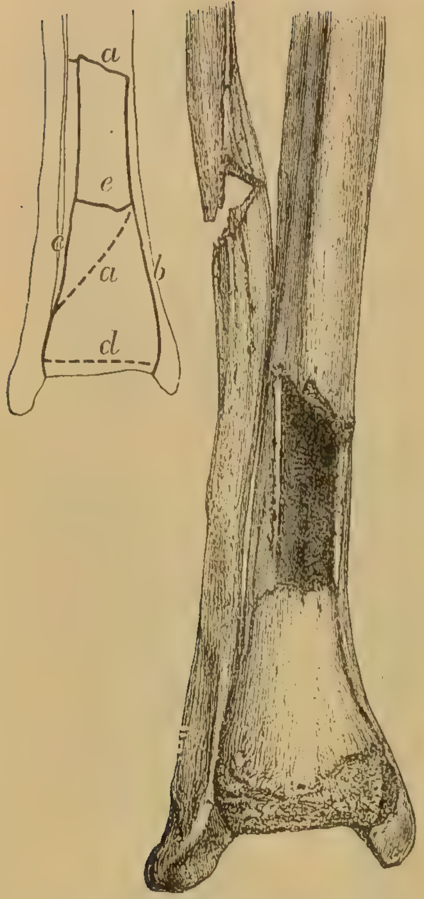


Fig. 210.—Leg Bones (viewed from the Front) after Amputation for a compound comminuted Fracture of both bones.

A spiral fissure (*a*) winds from the fibular fossa up and in across the hinder aspect of the tibia on to the inner surface, where it forms the upper edge of the gap shown. Two vertical fissures, *b* and *c*, are seen; they are joined at *d* on the astragalar surface and again at *e*. The lozenge-shaped fragment between *a*, *e*, *b*, and *c*, has been lost. (University College Museum, No. 316.)

(B) A fracture is said to be *comminuted* when either a whole bone is broken into bits (Fig. 209), or when, as is more common, splinters are broken off from the primary fragments (Figs. 208 and 211). The addition of a third small fragment is hardly in accordance with the idea suggested by the term "comminuted"; but we have no other general term. Comminution is a frequent result of direct violence, such as the impact and wedge-action of a bullet, or the passage of a wheel over a part; but it is often caused by indirect violence also. Thus, in fractures due to bending there is a tendency towards the formation of a wedge-shaped fragment on the concave side (Fig. 203, D), which again may be broken into many pieces; in spiral fractures a lozenge-shaped fragment is sometimes separated between the spiral fissure above and below, and two vertical fissures, one on each side (Fig. 210); and another example is yielded by the splitting up of the lower end of the radius by impaction into it of the shaft. The splinters may be *in sitû*, or more or less widely displaced into the surrounding parts; they may be more or less completely stripped of periosteum. Sometimes, especially over spongy bones, the periosteum is but little torn. Usually the injury to soft parts is proportionate to the comminution; but the skin may preserve its continuity over pulped soft parts and innumerable bony fragments (Fig. 209). On the whole, comminuted fractures of the shafts of long bones are

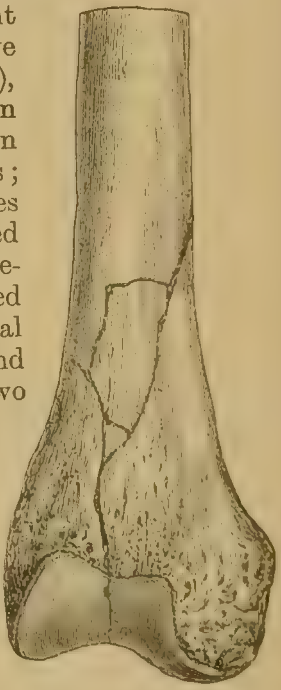


Fig. 211.—A Y-Fracture of the lower End of the Femur. The foot of the Y separates the two condyles, while the two arms embrace the wedge-shaped lower end of the shaft. This end is slightly comminuted. The fracture was simple, but the knee-joint suppurated. (Charing Cross Museum, No. 390.)

most commonly due to direct, of the epiphyses and spine to indirect violence.

The Y- and T-shaped fractures of the lower ends of the femur (Fig. 211) and humerus (Fig. 255) appear to arise from both direct and indirect violence; in some cases it is thought that the shaft is driven downwards as a wedge, and splits the joint end into two pieces (indirect violence); in others (direct violence) that the patella or olecranon acts as a wedge, and starts the fracture by a fissure from the joint surface.

Double fractures of bones—*i.e.* two distinct fractures, separated by a considerable piece of unbroken bone (Fig. 212)—are rare; they occur chiefly in the lower jaw, ribs, and long bones, and almost always from direct violence. Naturally, the difficulty of keeping the fragments in position is exceptional. The passage from a comminuted fracture to a double fracture is obviously gradual. More than two distinct fractures in the length of one bone are very rare.

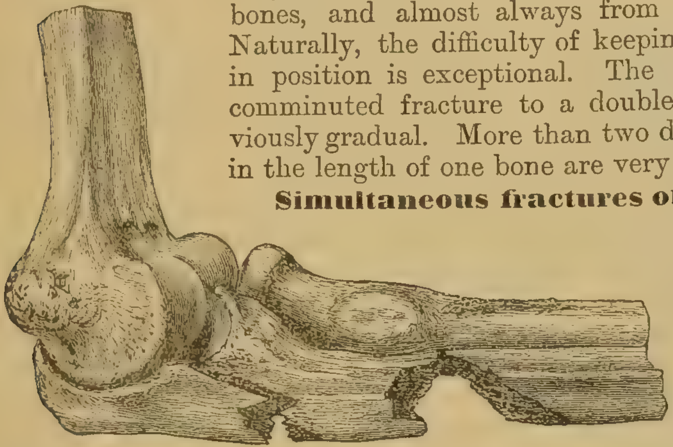


Fig. 212.—A double Fracture of the Ulna. The upper fracture runs down and back from near the centre of the greater sigmoid notch, detaching the olecranon and subcutaneous surface. The second fracture, an inch lower, was comminuted. The upper fragment was fully extended by the triceps; the middle fragment was drawn to the front of the humerus by the brachialis anticus, and the head of the radius accompanied it. (University College Museum, No. 205.)

Simultaneous fractures of many bones in the same or different parts are common. Especially is this the case with bones of the same part, which are frequently exposed to the same violence. In these injuries the diagnosis is generally rendered easier; the prognosis, as regards function of the part, and, in the case of the ribs, as regards life, more serious. Simultaneous fracture of several bones in different parts of the body results only from the most severe injuries, is often associated with lesion of important organs, and a very distinct mortality arises directly from shock. Should these dangers be escaped, the various fractures usually heal well.

Traumatic separation of epiphyses.—Though not strictly fractures, the two injuries are closely allied, being due to similar violence, presenting similar signs, and requiring similar treatment; often, indeed, they are combined. Yet, as separations of epiphyses present certain clinical peculiarities, they must be specially treated of. They are no longer regarded as very rare accidents; but, owing to difficulties in diagnosis, we are unable to say more than that they are much less common than fractures.

Epiphysial discs have been found in long bones as late as 30, but no certain cases of separation seem to have been recorded after 21, at which age all important epiphyses have usually united.

Age.—As the violence necessary to separate an epiphysis is considerable, and as young children are more carefully looked after than older ones, these accidents are commoner after than before the age of 10, being most frequent between 16 and 17 (Bruns); but in one case compound separation of the lower tibial epiphysis was produced in a five months' fœtus by the mother's falling from a height on to the abdomen; and there is a considerable group of cases, chiefly affecting the upper epiphysis of the humerus, due to manipulations during delivery.

Seat.—As to the frequency with which different epiphyses are affected, the best criterion is probably afforded by the statements of the number of cases which have come under the notice of Mr. J. Hutchinson, junr., at the London Hospital. Mr. Hutchinson during some years saw 17 cases of separation of the lower radial epiphysis, 14 or 15 of the lower humeral, 13 of the upper humeral, and 10 of the lower femoral. All are agreed that these are the epiphyses most commonly separated; but as to the exact order of frequency, authors, basing their statements on collections of recorded cases, differ. Thus Mr. Hutchinson's collection of over 350 cases gives the following results:—Lower femoral epiphysis, 75; upper humeral, 66; lower radial, 54; lower humeral, 52; internal epicondyle, 38; lower tibial, 27; upper femoral, 26 (?); great trochanter, 11; upper tibial, 10; tibial tubercle alone, 6; lower ulnar, 6; upper fibular, 4; clavicular, 3; upper radial, upper ulnar, and lower fibular, 2 each; small trochanter, 1.

Condition of the parts.—The force required completely to detach an epiphysis is considerable, though no doubt slighter violence often produces incomplete detachment or separation without displacement—the real nature of the injury being missed. The periosteum is the main obstacle to separation, the force required to tear off an epiphysis after careful division of the periosteum being about one-fifth of that required when this membrane is intact. The periosteum of a growing bone is specially thick near, and has a close attachment to, an epiphysis. When the latter is detached from the shaft, the periosteum may be merely loosened round the latter; but if the epiphysis be much displaced, the end of the shaft tears a hole for itself in the periosteal tube on the side towards which it is moving, and, after this, the epiphysis carries the periosteum with it, stripping it off the shaft (Figs. 213 and 268). This extensive denudation is peculiar to the injury, and is due to the toughness of the periosteum and the looseness of its attachment to the shaft. Still more important is the exact plane in which the separation takes place. The main part of the epiphysial disc almost always adheres to the epiphysis. Bruns collected 61 cases in which statements were made on this point. In 5, all occurring in the earliest years, the cleft traversed the disc about midway between the shaft and the epiphysis, the surface of each being smooth and cartilage covered. In 23 the cleft traversed the zone of calcification between the cartilaginous disc and the shaft, for the disc almost invariably adheres to the epiphysis more strongly than to the shaft; both fragments then show elevations and depressions, and present small calcified particles. Detachment in this plane is the rule in early childhood. These two varieties may be spoken of as *pure* epiphysial separations. The remaining 33 cases, all between 10 and 20, were *mixed* epiphysial separations and fractures—the cleft lying to a greater or less extent in the shaft parallel to, but just beyond the epiphysial disc—so that both fragments are coated with bone; or in the calcifying zone of the epiphysis at first, thence passing obliquely into the tissue of the shaft (Fig. 291). The "mixed" variety would appear to be the commoner, but the numbers are too small to decide the relative frequency of these varieties. Whether or not separation of an articular end will open into a joint varies with the epiphysis, and even with the age. Thus, at birth, separation of the upper epiphysis of the femur, which includes head, neck, and great trochanter, would be extracapsular; three or four years later there are separate nuclei in both head and great trochanter, the neck is fully ossified from the shaft, and a separation of the upper epiphysis (head only) is entirely intracapsular.

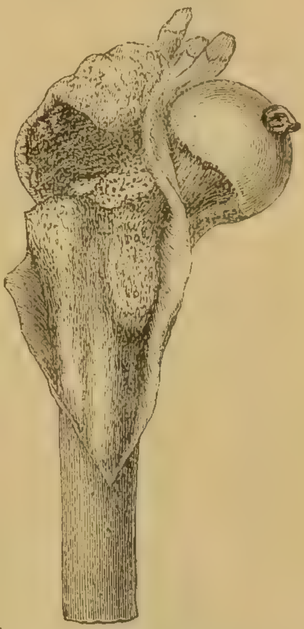


Fig. 213.—Separation of Epiphysis of great Trochanter with wide Stripping of the Periosteum from the Shaft. (London Hospital Museum.)

Signs.—These vary with the degree and plane of separation, but the age must always be such as to render the accident possible. In well-marked cases, deformity, abnormal mobility, and crepitus are present. Crepitus is soft when the cleft lies wholly in cartilage, rougher when it traverses the calcifying zone, best marked when it invades the shaft. In pure cases the plane of separation corresponds with the position of an epiphysial disc. The end of the shaft may have a characteristic form, and is usually convex, the epiphysis being correspondingly concave—points easily concealed by thick covering or swelling, and lost in mixed cases.

Diagnosis.—These accidents are distinguished from dislocations just as are fractures of articular ends. Dislocations of the elbow are most frequent between 5 and 15; otherwise separations of epiphyses almost replace dislocations up to 20. From fracture of the articular end, separation of the epiphysis is distinguished by the plane of separation, the form of the fragments, perhaps a characteristic deformity, and the quality of the crepitus. The age may decide in favour of fracture.

Separation of an epiphysis, without displacement or tearing of the periosteum, can be characterised only by slight mobility and crepitus. Partial separation is unrecognisable unless accompanied by a greenstick fracture and deformity. Such injuries are usually taken for bad sprains, and are probably sometimes followed by impaired growth.

The **prognosis** has some special points in it. Generally the *functions of a joint are threatened* by permanent displacement or by callus, which tends to luxuriance owing to the extensive stripping up of periosteum.

Suppuration and *necrosis* have occurred with undue frequency as compared with fractures—a fact connected by some with the wide separation of periosteum, which, at all events, implies a wide area of injured tissues, in which pyogenic organisms would be likely to settle and grow. Again, the deep layer of the periosteum, like the junction of epiphysis and shaft, is actively growing, and very vascular, and we know from experience of acute infective osteomyelitis and periostitis, that these tissues are specially liable to the attack of pyogenic organisms. But these considerations afford a by no means complete explanation of the occurrence of suppuration; for, as Hutchinson points out, no suppuration occurred among twenty-four cases of detachment of the lower humeral epiphysis, whilst it followed in six out of ten cases in which the great trochanter was concerned. Possibly this may be accounted for by the fact that the great trochanter is generally detached by direct violence, from which widespread injury of soft parts results. Abrasion of the skin would, of course, increase the liability to infection.

All authorities seem now agreed that marked *impairment of growth* is only an exceptional result of these accidents. Experiments on animals, the prolonged observation of cases occurring in man, the rarity of shortening after Ogston's operation and "redressement forcé" for genu valgum, the failure even of suppurative epiphysitis always to arrest growth, coincide to show that the epiphysial disc resists injury strongly. J. Hutchinson, junr., has followed up six cases of separation of the lower radial epiphysis, in only one of which did marked shortening occur. Even resection of a protruding and irreducible end of a shaft does not necessarily stop all growth. And we may recall here that detachment of epiphyses is most common at an age when the growth to be expected from all epiphyses, except those of the first importance, is so slight as to be negligeable.

The most important factors in producing arrest of growth are:—(1) The plane of separation. Probably the danger is greatest when the "preparatory zone" of proliferating cartilage cells is torn through. (2) Permanent displacement of the epiphysis; in animals displaced epiphysial discs undergo rapid atrophy (Hutchinson). (3) Any inflammation, especially suppurative. (4) Mechanical destruction of growing cells by rough manipulations, which will also excite inflammation.

placed in modes and in directions which do not admit of classification.

Causes of displacement.—The direction and nature of the line of fracture, though not causes, have a most important influence. The more nearly transverse the line, the more irregular the surfaces, the less the tendency to displacement, and *vice versâ*. Also, any lateral displacement is usually determined by the direction of the broken surfaces, which glide on each other. For instance, in the upper third of the femur the usual line of fracture is from above, downwards and forwards, and the lower fragment glides upwards and backwards under the influence of the hamstrings; as it does so it hitches against and pushes forwards the upper fragment, thus helping the psoas to flex it (Fig. 284). If the fracture line tend inwards or outwards in its course, the lower fragment, as it ascends, will push the upper fragment inwards and outwards; and lastly, if the line be unusual—*i.e.* from above downwards and backwards—the lower fragment rises in front of the upper, and by its weight annuls the effect of any contraction of the ilio-psoas which may occur; it thus prevents the usual deformity (Fig. 285).

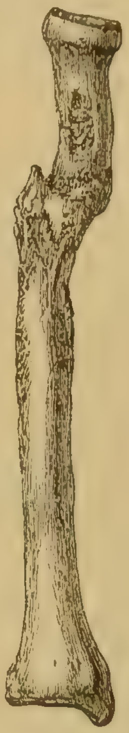


Fig. 215.—A left Radius repaired after Fracture in the upper Third of its Shaft. The upper fragment is fully supinated; the lower is drawn up and in by the pronator teres. (University College Museum, No. 209.)

The actual causes of displacement are:—(1) The fracturing force which, continuing to act upon the fragments, drives them before it. Certain displacements—*e.g.* impaction—are always due to this cause. (2) Elasticity of the soft parts. (3) Muscular contraction, formerly assumed to be constant or "tonic." But this is not the case. The muscles contract intermittently under nervous stimulation, voluntary or involuntary, and produce an effect upon the fragments, which is the resultant of the various forces, and the resistances which they have to overcome. Frequently fragments are displaced by muscles which have no opponent attached to the fragments—*e.g.* the upper fragment of a radius, broken above the pronator teres, is completely supinated by the first contraction of the biceps; there is no muscle on the fragment which can pronate it, so we have no choice in our treatment but to carry the lower fragment to the upper. Again, gravity is the only force which would naturally tend to undo shortening produced by muscular contraction, but its power to act is often annulled by the horizontal position. Muscular contractions are, therefore, a very important cause of displacement. (4) The weight of the part. Almost invariably the lower fragment of a fractured spine drops backwards, and, unless carefully supported, the distal portion of a broken limb tends to dangle from the upper when the latter is raised. It is gravity which causes eversion in fracture of the thigh. (5) External force. Unskilful lifting or handling of the part, or the bearing of weight on a broken limb may produce disastrous results, protrusion of a fragment through the skin being common. Faulty apparatus used in the treatment is another common cause; thus the tendency of all leg splints, with a vertical foot-piece, is to hold the foot vertical while the upper fragment rotates out. The tendency of Liston's long splint is to produce angular deformity of the femur outwards, or forwards and outwards (Fig. 286), and rotation of the lower fragment.

Displacement due to the fracturing force is often spoken of as *primary*, while that due to all other causes is *secondary*—a distinction of little value; for, in point of time, the other causes may act simultaneously with the fracturing force, and muscular contraction may be the sole cause of displacement. The terms *early* and *late* displacement are more useful, as distinguishing between displacement found soon after the occurrence of the fracture and that which may subsequently occur, gradually or suddenly, owing to unlocking of fragments or yielding of restraining soft parts.

THE SIGNS OF FRACTURE.

In making an examination for fracture a regular order should be followed, the object being to effect an accurate diagnosis without inflicting more pain upon the patient, or more damage upon the tissues, than is absolutely necessary; consequently, crepitus is not the first sign which should be sought for, and movements of the fragments should be practised only with care and gentleness. The chief points to which attention must be paid are:—The detection of a fracture, its position and direction, the number and relation of the fragments to each other, the condition of the soft parts near the fracture and of the joints in which the injured bone takes part. The best plan is, first, to inquire into the history; then, to expose the injured part and that which corresponds to it, place them as nearly as possible in similar positions, and carefully compare them by inspection and measurement; lastly, to palpate the part, and endeavour to move the fragments on each other. Thus we may hope to detect the signs of fracture present in any case.

The signs of fracture.—These are:—(1) Pain and tenderness, (2) impairment or loss of function, (3) deformity from swelling or displacement of fragments, (4) abnormal mobility, and (5) crepitus.

In the *history*, inquiry should be made as to:—The time and exact mode of occurrence of the accident, to determine the parts likely to be injured, and the cause—whether muscular, indirect, or direct violence; how it acted, and what kind of fracture is likely to have resulted; whether any "crack" was heard by the patient or bystanders (a similar noise may be heard when strong tendons or ligaments are ruptured); the immediate symptoms (pain and loss of power), endeavours to use the part, details of immediate treatment and mode of transport. If muscular violence seems to have been the cause, and in any case in which the cause seems to have been inadequate, or the pain unusually slight, questions should be put to detect the presence of any cause of spontaneous fracture. It is always worth while asking if the limb or part was normal up to the time of the accident.

(1) **Pain and tenderness** at a point which has been directly injured are of no diagnostic value; but when only indirect violence can have acted upon the part, the presence of sharply-localised pain and tenderness may be of much importance—sometimes, especially in fractures of ribs and fibula, they are the only symptoms which render the existence of a fracture probable. Pain is complained of; tenderness may be discovered by palpation, by percussion, or by moving the fragments on one another, direct pressure on the painful area being avoided. If, after a wrench of the ankle, the patient complains of pain three inches above the external malleolus, and it is found that pressing the two leg bones together at the middle of the leg increases pain at the suspected point, there is good reason to fear that a fracture has been sustained. Similarly, pain elicited at one point in a rib by pressure on another sufficiently removed is often of value. Pain and tenderness may be astonishingly slight, or even absent, without evident reason. Their absence is characteristic of tabetic fractures.

(2) **Loss of power,** more or less complete, naturally results when a part is deprived of its rigid support. The impairment of function is least marked when the fracture does not destroy the continuity of the weight-bearing portion of the bone, when a certain continuity is re-established by impaction, when only one—and that the least important—of two parallel bones is broken, and when a strong ligament serves as a *point d'appui* for the lower fragment, as in certain intracapsular fractures of the femur, or when the fragments are held together by untorn periosteal and fibrous coverings (patella, olecranon). In some of the above cases loss of power is very slight; on the other hand, a severe contusion or other painful injury of a part, without fracture, may produce absolute helplessness—so impairment of function is not a sign of first importance.

(3) **Deformity** may be due to swelling from extravasation, inflammatory or reparative changes, or to displacement of the fragments. It is detected by inspection, measurement, and palpation.

Deformity from early swelling may be localised or diffuse, superficial or deep. This is of no diagnostic value as a sign of fracture if the part have been directly injured; it is of much value in opposite circumstances, coupled with circumscribed pain and tenderness. The detection of a "callus tumour" (*deformity from late swelling*) is most important when an examination is made some time after an accident to determine whether a bone has been fractured.

Deformity from displacement may at once strike the eye, or careful and repeated measurement and palpation may be required, or it may be concealed by early swelling and thick coverings. It is most striking in the shafts of long bones. The displacements described earlier (page 745) may cause:—(1) shortening of a part; (2) very rarely lengthening of a part, though lengthening of a bone by separation of its fragments—*e.g.* patella transversely fractured—is common; (3) increase in girth of a part from overlapping and separation of fragments; (4) angular deformity—this, when absent with the limb at rest, often develops when the part is raised actively or passively; and lastly (5) rotatory deformity. The total deformity may be made up of two or more of the above varieties. In examining by eye, by measurements, or by touch, it is essential that the injured part, and that which corresponds to it, shall be satisfactorily exposed, and placed as nearly as possible in corresponding positions. The eye will in most cases then detect any deformity, and some place more faith in it than in measurement.

Measurements are undoubtedly open to certain sources of error—*e.g.* quite a distinct difference in length of limb may exist naturally, the possessor of the limb having no knowledge of it; it is often difficult or impossible to place the two limbs in truly corresponding positions; the presence of swelling upon the injured side renders a tape measurement upon its surface too long; and lastly, the bony "points" from which measurements are taken are often of considerable extent, and when they are thickly covered, anything but corresponding points may be selected from or to which to measure.

In the upper limb the best of these "points" are:—The ends of the clavicle, the acromial angle—meeting of the outer and posterior borders; the inner and outer epicondyles, the head of radius or the cleft between it and the humerus, the tip of the olecranon, the tip of the styloid process of the ulna, the cleft of the first carpometacarpal joint, and the base of a phalanx.

In the lower limb the best points are:—The anterior superior iliac spine, points on the edge of the patella, the edge of a femoral condyle (especially if the knee be flexed), the ilio-tibial spine on the outer anterior angle of the head of the tibia, and the tips of the malleoli (especially the internal). Measurement is more difficult in the lower than in the upper limb, owing to greater vagueness of the points—especially of the anterior superior iliac spine—and to the variation in apparent length of the limb, as measured from this point, according as the hip is more flexed, adducted, or abducted than its fellow. With the two limbs lying flat and parallel to the vertical axis of the trunk, one may appear to be shorter than the other; but when a thumb is placed upon each anterior spine to mark its position, it may become evident to the eye that the line between them is oblique to the vertical axis of the body—one spine is raised, the other is lowered, and the limb in connection with the first is adducted and shortened, whilst the other is abducted and lengthened. Or, again, measurement shows one of the two parallel limbs to be shorter than the other; the anterior spines may be on the same horizontal line, but that on the short side is generally lower and a little more prominent than its fellow, the fold of the groin is deeper, and there is lordosis of the lumbar spine. This shortening is due to flexion.

To avoid these sources of error, measurements should be taken with a graduated rod, like the instrument with which a shoemaker takes the length of a foot. Thus an error from swelling is avoided. Corresponding points over the bony prominences between which it is desired to measure should be marked with a pencil. To measure the whole length of the lower limbs, the rod should be 4 ft. 6 in. long; 8 in. from the upper end, 15 in. of brass riband should be attached at right angles to it, and there should be two travelling points on the rod. The upper 8 in. of the rod should be laid accurately in the mid-line of the abdomen; the pelvis should be so placed that the lower edge of the brass riband corresponds to the lowest perceptible point of the anterior spine on each side. It should be seen that there is no lordosis, that the limbs are lying flat and symmetrically on either side of the rod. The sliding points should now be placed in contact with corresponding points on the inner malleoli, and the difference, if any, read off.

The position of the great trochanter shows whether shortening is due to injury (or disease) above or below it; two plans are used to determine it. Nélaton's line, which the top of the trochanter should touch, may be drawn from the anterior superior spine to the most prominent point of the tuber ischii; the latter is especially vague, and it is often undesirable to move a patient about sufficiently to draw the line with any accuracy. Bryant's triangle is less open to objection; with the patient on his back, a vertical line is dropped from each anterior spine, from this a horizontal line is drawn down to the great trochanter—if possible, to the tip behind; the completion of the triangle is unnecessary. The two limbs being placed symmetrically with regard to the mid-line, the relative lengths of the vertical lines will show whether one or other trochanter has been driven forwards or has fallen backwards, and the relative lengths of the horizontal lines will show whether one trochanter is raised. By this method disturbance of the patient is avoided, and the vertical line gives a more accurate point to measure from than the iliac crest, and renders the error due to swelling less; it is the only line along which the distance of the trochanter from the anterior spine should be measured.

The detection of deformity is often sufficient to render the diagnosis of fracture certain. In greenstick fractures, and fractures with firm impaction, we have to depend more or less entirely upon this sign. Deformity may, however, be the result of a former injury or disease, or even of some congenital peculiarity; or it may be the result of the injury, although no fracture has occurred, as in the complete eversion of the lower limb, which may follow a bruise of the hip. On the other hand, in a larger number of fractures there is no discoverable deformity.

(4) **Abnormal mobility** in the length of a bone is a certain sign of fracture; conceivably it may be due to an old ununited one.

but practically this may be set aside. It is sought for by gently though firmly grasping the limb above and below the suspected point, and endeavouring to produce angular movement, and shifting the hands up and down until the whole bone has been examined. As a rule it is easily obtained in parts containing one bone, or two if both are broken; not so when only one of the two has suffered, especially if it be the radius or fibula, to which the ulna or tibia acts as a splint. Each bone must then be carefully examined along its length, the fingers grasping it rather than the limb; the same applies to a metacarpal or metatarsal bone. With the fibula, and to a less extent with the radius, it will be found useful to support the fingers or thumbs, as the case may be, of the examining hands upon the tibia or ulna, and to press the suspected bone towards its fellow; if broken, it will be felt to yield unduly, and to lack its usual spring, and the point of yielding will soon be ascertained.

When a bone capable of rotation is broken so high, and perhaps under such deep covering, that angular movement is difficult to detect, the shaft may be rotated with one hand, whilst the other, if possible, grasps the upper end firmly, to determine whether the two move together; if they do not, immobility of the upper end is conclusive evidence of abnormal mobility in the length of the shaft. This can be well carried out in the humerus and femur, up to the great trochanter; it is far more difficult to be certain whether the head of the radius moves or not, for it presents no irregularities, can be felt only posteriorly, and is covered by muscle even there. When the neck of the femur is broken, we cannot feel that the head does not accompany the shaft in rotation; but a hand on the trochanter may feel that its movement is abnormal, for, instead of describing a considerable curve with the neck and head for its radius, it passes through a smaller and smaller arc in proportion as the fragment of neck in connection with it is shorter, until, in fractures of the base of the neck, it merely rotates like the shaft on its own axis.

But though the detection of abnormal mobility is a certain sign of fracture, the absence of this sign does not negative fracture; it is absent in many incomplete and in firmly-impacted fractures, and may be so slight in deeply-toothed and locked fractures as to defy detection—especially when the bone is one of two, and deeply placed. It is evident, too, that in such a fracture the head would rotate with the shaft. Sometimes, with processes torn off, or in fractures of short bones, it is impossible to seize or so to act upon the fragments as to produce and detect abnormal mobility.

(5) **Crepitus.**—The grating of one broken surface against another is felt rather than heard, though it may be audible at some distance. It is obtained by the same manipulations as have been described for the detection of abnormal mobility—rotation being specially useful. The conditions of its production are the existence (*a*) of abnormal mobility, and (*b*) of contact between the fragments. But a single, certain click is often obtained in fractures of the fibula, ribs, or other bones when we cannot detect the abnormal mobility, and may

enable us to feel quite sure of our diagnosis. It is absent in incomplete fractures, in which there is no gliding of one fragment upon the other; in cases of firm impaction and interlocking, though it is often obtained when mobility cannot be discovered; in cases of intervention of soft parts, of diastasis of fragments, and of their separation by overlapping. In the two latter instances it will be obtained on pressing the fragments together, or on extending them until they are drawn to the same level, and then moving them upon each other. Crepitus is obtained most easily between irregular freely movable ends of compact tissue placed superficially; comminution is favourable. Crepitus is less distinct with surfaces of cancellous bone, diagnosis in fractures of joint ends and of short bones being decidedly more difficult than in the case of shafts. It is least distinct, and is spoken of as "soft crepitus" in separations of epiphyses. Crepitus is the sign of fracture the eliciting of which causes most pain, and is often unnecessary to the diagnosis; yet it is the most satisfactory evidence that the fragments are actually in contact, and should it be absent in circumstances which would seem favourable to its production, it is wise to seek for the explanation, lest delayed or non-union should result from some remediable cause. Narcosis greatly facilitates the obtaining of mobility and crepitus; but the patient's struggles and the necessary endeavours to control them may be productive of much harm. In the case of a rib, crepitus may be heard by the ear applied to the chest when it cannot be otherwise detected.

It is necessary to learn by experience to distinguish between crepitus and the crepitations produced by tenosynovitis, emphysema of connective tissue, and blood-clot; and to be on guard against mistaking grating in a rheumatoid joint for crepitus of bony fragments.

When there is reason to fear a fracture, but its existence can be neither proved nor disproved, one must be assumed to exist, and proper treatment adopted.

REPAIR OF FRACTURES.

Naked-eye changes.—On examining a recent fracture we find the sharp fragments, more or less displaced, lying in the midst of a collection of fluid blood, which clots slowly and varies much in amount and in the extent to which it infiltrates the medulla and the surrounding connective tissue. The periosteum is generally torn through all round, often very irregularly, and it is sometimes widely stripped from the fragments. The soft parts round about, especially the muscles, are torn and bruised in varying degree.

At the end of the *first week* they are covered with clot; the fluid portion of the extravasation is becoming absorbed or is already gone; the clot is becoming peripherally decolorised. The injured soft parts to which it adheres are swollen, and their special structure is obscured; they present the homogeneous, translucent, pinkish grey appearance of soft parts infiltrated by an inflammatory exudation; and all—periosteum, muscles, fat, connective tissue—are welded into an irregular mass.

By the end of the *second week* (tenth to fourteenth day) clot has disappeared or remains only here and there, especially near the fissure; but there is a widespread staining. The mass of swollen soft parts is now more regular in outline, and in it can be detected a spindle-shaped swelling about the fragments, limited by a membrane, which is continuous with the periosteum above and below, and which separates the embryonic tissue for the repair of soft parts from that for the repair of the bone. The bruised yellow marrow above and below the fracture has been replaced by vascular granulation tissue, which has blended across the cleft, and a similar tissue is creeping from without between the broken ends. The ends of the fragments are now united by this new tissue, much as if they had been pressed into a mass of hot sealing wax (Billroth). The new tissue is called "callus"—"provisional" or "temporary," because more or less of it is destined ultimately to disappear. That which lies between the periosteum and the bone is called "external" or "periosteal"; that in the medullary canal "internal," "endosteal," or "myeloid"; that between the fragments "intermediary" (Fig. 221). The periosteal callus is the most freely developed, though it rarely, if ever, forms so complete a sheath around the fragments in man as it does in animals (Fig. 216). It is most strongly marked in angles due to displacement of the fragments and accompanied by stripping up of the periosteum (Fig. 217), and it extends for some distance upon the fragments, ending gradually. At first the whole callus is soft, translucent, red and vascular in the living, and pinkish-grey in the dead state; but it gradually becomes firmer. It now ossifies either directly or after becoming fibrous or cartilaginous. In animals more or less cartilage usually appears particularly in the neighbourhood of the cleft (Fig. 216); but in man it is only occasionally seen.

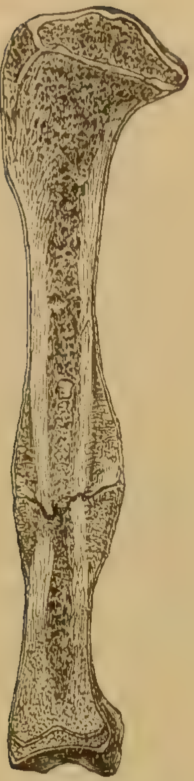


Fig. 216.—A longitudinal Section of the Tibia of a young Dog killed ten days after a transverse Fracture without Displacement. The line of fracture is clear; no tissue can be distinguished between the fragments. These are held together by an oval mass of external callus, most freely developed on the posterior, muscle-covered surface; and by a plug of internal callus in the medullary canal which does not extend so far as the external up and down from the fissure. The external callus consists of finely porous bone which has evidently appeared first in the angles between the bone and the raised periosteum, and has spread thence in fibro-cartilage towards the fissure—most rapidly in actual contact with the shaft. Between the two wedge-shaped masses of bone on each side, a triangular area of fibro-cartilage remains at the level of the fissure. In the internal callus at this level only a few flecks of fibro-cartilage remain. The periosteum is thickened over the callus tumour. The production of bony callus is much earlier and freer than in the following specimens from man.

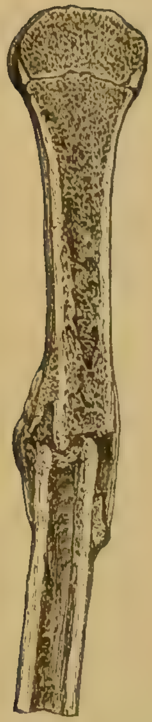


Fig. 217.—A longitudinal Section of a Humerus nineteen Days after transverse Fracture with slight lateral Displacement above the entry of the medullary artery, from a boy, aged 10, who died from a fracture of the skull. Callus formation is poor and late as compared with that in Fig. 216. The dry periosteum indicates the extent of the external callus. This formed most freely on the fragments where the periosteum had been most widely stripped; here wedges of porous bone are seen. Internal bony callus has formed most freely above the fracture; but it has closed the medullary canal of each fragment, and has narrowed the medullary channel in the neighbourhood. (Univ. College Mus., No. 41.)

About the *twelfth to fifteenth* day the deposit of lime salts can be recognised in the angle between the bone and the raised periosteum; it advances towards the cleft most rapidly in the layers of callus next the bone (Fig. 217). We first notice slight grittiness, opacity, and increased firmness; then a hard red-brown mass, wedge-shaped and granular on section, becomes plain in the callus, and spreads till the whole is converted, and the masses above and below the cleft blend across it, fixing the fragments together, much as the clay around a graft unites the scion and stock. Ossification of the medullary callus occurs similarly, but a little later, beginning also in contact with the bone, and away from the fracture; a ring of bone is thus formed within each fragment, which thickens as it approaches the cleft, at the level of which it usually closes the medullary canal, and blends with its fellow across the cleft.

Finally, periosteal ossification creeps into the intermediary - callus and joins the medullary callus. The fragments are thus united by bone (Fig. 220).

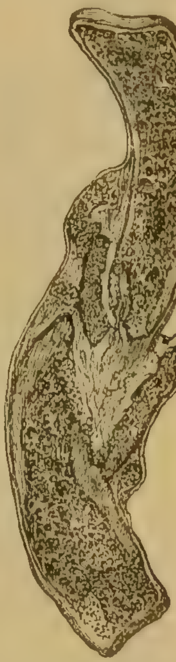


Fig. 218.—A longitudinal Section of a Clavicle twenty-three days after a comminuted Fracture. From a potman, aged 23, run over by a 'bus; he died of bronchitis with delirium, and was from the first difficult to control. The main fragments are united by a large mass of fibrous-looking, blood-stained callus, in which are embedded two large splinters. At three or four spots beneath the swollen periosteum posteriorly, considerable masses of granular new bone are seen; but ossification is decidedly late. Univ. College Museum, No. 88a.

The superficial layer of callus has assumed the appearance of periosteum, and remains unossified; it is closely connected with the surrounding injured tissues, and both it and the periosteum for some distance from the fracture are a good deal swollen. Often a thin layer of new periosteal bone can be traced for some distance from the seat of fracture, but much and widespread callus suggests unusual irritation, probably from a compound fracture (Fig. 224). The newly-formed bone is very porous, the vascular channels being both large and numerous, and so arranged as to be at right angles, or at least oblique and to the surface, to the chief Haversian canals of the old bone. At first, and for some time, the stalactitic needle-like tubes, plates, and irregular masses of callus can be easily stripped off, leaving the bone apparently unaltered; but, later, its vascular foramina and grooves are obviously enlarged where the callus has been separated, and their further enlargement renders the surface quite irregular (Fig. 219).

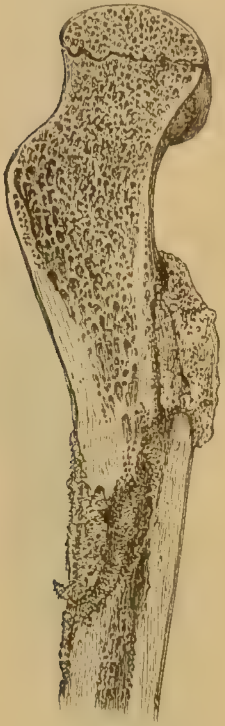


Fig. 219. — Section of Femur of Boy of 14, who died of enteritis four weeks after an oblique fracture near junction of upper and middle thirds. The lower fragment has risen a little in front of and inside the upper. A fairly complete ring of porous callus unites the fragments, though not very firmly. The callus is in great excess anteriorly in the angle due to displacement. The line of fracture is quite distinct. The medullary canal was probably closed by new bone. The compact bone, where covered by callus, is superficially toothed and more porous.

This rarefaction of the compact bone renders it more like the porous callus; a fine injection proves that the vascular spaces of old and new bone are continuous (Fig. 222); and later specimens show that fresh bone is deposited round the vessels in these spaces—partly upon the old, partly upon the new, increasing the density of both, and establishing absolute continuity, so that the callus can no longer be chipped off. This condensation goes on until the bone about the fracture becomes more dense than normal; but the endosteal callus does not as a rule become so dense as the periosteal and intermediary. Nor does all the callus undergo this condensation; much is soon absorbed. Callus is usually formed in excess, but perfect fixation tends to limit the amount of provisional callus. The firmer callus becomes, the less of it is wanted; the peripheral portion and any parts which bear no strain are absorbed; the removal of these leaves the more central portion, especially that situate in angles due to displacement, to bear the whole strain, and its density and strength increase. Increase in density normally keeps ahead of absorption, and for months or years there is excess of callus of too great density. Gradually all that is unnecessary is removed, and a return to or towards the normal external form is effected under the modelling influence of surrounding parts.

The callus acquires everywhere a compact surface-layer like that of normal bone. Within, cancellous tissue develops in accordance with the laws governing its development in normal bones; consequently, its main cancelli are in the lines of greatest pressure and tension, and their arrangement in cases of displacement may be as complex as it is in the neck of the femur (Fig. 220). In the interior, the plug of medullary callus becomes first cancellous, and is then gradually removed, the continuity of the medullary canal being thus restored. Red marrow replaces it, then this gives way to yellow marrow. When there has been little or no displacement, the seat of a fracture becomes unrecognisable in a rabbit in twenty-six to twenty-eight weeks; in man the same occurs, but only after a much longer period. Even the interstitial callus, in which at first the course of the vascular channels is similar to that in the periosteal callus, becomes so modified by those interstitial changes of absorption and deposit which continue throughout the life of a bone, that it is indistinguishable from the rest of the compact tissue. When there is marked lateral displacement, the ends of the fragments remain sealed with compact tissue, covering a little cancellous bone. The compact walls in contact with each other may remain liable to severe strain, in which case there is no attempt made to restore the medullary cavity; but in other instances, bridges of callus filling up angles tend more and more to convey weight and strain from one fragment to the other, with the result that compact portions of the shaft become functionless, and are consequently rarefied, or even entirely removed.

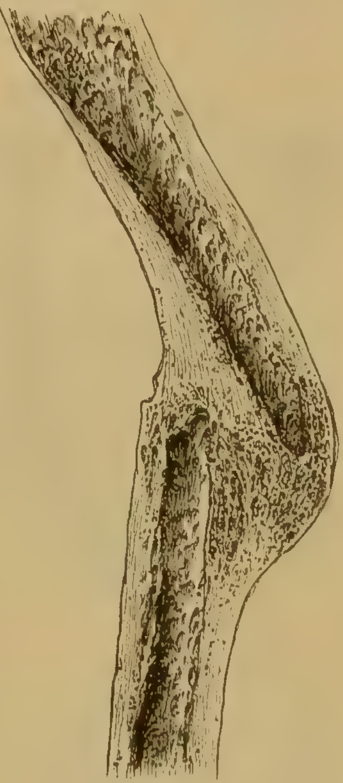


Fig. 220.—Section of the Shaft of a Femur united after Fracture with angular Deformity to show the complex arrangement of cancelli between the anterior wall of the lower fragment and the lower end of the upper, and the buttress of dense bone in the angle posteriorly. (University College Museum, No. 269.)

Nor are the changes after fracture with *permanent dislocation of fragments* limited to the seat of fracture. Altered curve alters the statical conditions under which the repaired bone must work, and gradually its architecture is changed. Some of the alterations—as change in form and position of a joint surface—may be striking.

In the repair of *comminuted fractures*, the loose fragments are set, as it were, in the callus tumour (Fig. 218); and long after this has ossified, dense pieces of shaft will be seen on section, contrasting with the looser new bone around. The ultimate fate of these fragments has not been worked out. Frequently, too, fragments are so dislocated as to project like spines into the soft parts; these may serve as points of attachment of muscle—and show little tendency to absorption (Fig. 251).

Fissures of flat bones are often poorly repaired. In the skull, where practical immobility is secured, little or no callus forms; the outer edge becomes rounded, and thin union is effected just external to the dura mater, so that the inner surface is rendered smooth and continuous; but permanent gaps may remain. The scapula (Fig. 242) and ilium (Fig. 272) are repaired more freely, because they are thickly covered, and mobility of the fragments keeps up irritation.

The fragments of *fractured spongy bones* are not, as a rule, much displaced. Little external callus forms, but the pieces are welded together by callus between them, which first becomes abnormally dense, and then assumes more and more nearly the type of the bone of which it forms a part.

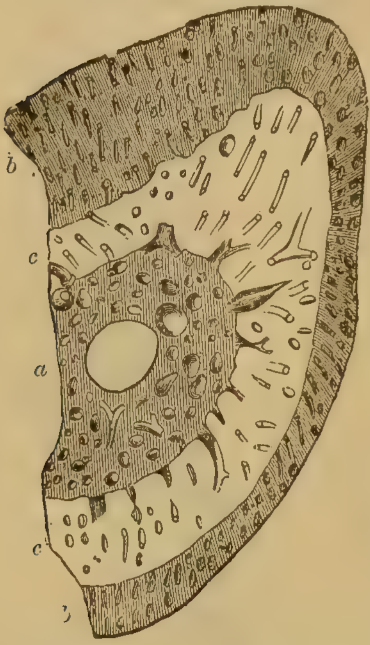


Fig. 221.—A transverse section of the Tibia of a Dog close to a Fracture eight days old.
a, Internal callus; *b*, external callus; *c*, cortex of tibia. × 20. (Billroth.)

Articular cartilage is repaired by fibrous scar, which replaces the early blood-clot, and is in its turn slowly replaced by true cartilage. Bits chipped off either remain loose or become encapsuled and attached to some part. *Rib cartilages* are united by fibrous callus, of which the subperichondral portion ossifies, forming a bony cylinder, clasping the ends together.

Little is known of the reunion of *detached epiphyses;* we should expect that when the detachment occurs in bone, union would occur as in fracture, and when in cartilage, by fibrous tissue. Danger to growth lies in replacement of the growing cartilage by either bone or fibrous tissue. The new vessels communicate.

Microscopic changes.—Cell-infiltration of soft parts round about a fracture begins at once after the injury; by the second day all are swollen, their structure is obscured, and they are blending in one mass. The torn periosteum is at first lost in the early swelling, but as the callus acquires firmness, a section shows upon its surface, a fibrous streak uniting the periosteum on the two fragments; this gradually assumes the structure of periosteum, but it is some time before it becomes distinct from the scar in the soft parts around the fracture. New vessels develop rapidly, and establish connections between the vessels of the stripped-up periosteum and those of the bone on the one hand, and of the soft parts on the other. Thus a mass of vascular granulation-tissue forms round the bones and, more slowly, also in their medullary cavities and Haversian canals, and forms a soft bond of union between the fragments, obtaining a hold upon them by absolute continuity with their soft parts and vessels. Some authorities regard the infiltrating cells as leucocytes, but most think that proliferating connective tissue corpuscles take their share. The vessels running through the cell-mass beneath the raised periosteum, though freely intercommunicating, naturally run more or less vertical to the bone (Fig. 222).

The granulation tissue becomes more densely cellular and firmer, fluid being absorbed. Blood-clot is infiltrated and decolorised by removal of its pigment; except as a framework into which cells can wander, clot takes no part in the repair, and soon disappears.

In the angle between the raised periosteum and the bone—the point of least irritation—there early become visible, both in longitudinal and transverse sections, slight, faintly-granular opacities, due to deposit of lime salts; they include cells in their substance, and are usually of needle-like form; from their bases on the bone their points rise more or less vertically, and between each pair is a vessel surrounded by cells. Evidently the deposit of lime salts occurs midway between two vessels, and the needles are sections of the walls of calcareous tubes which lengthen, branching as the vessels branch, and forming a very open trelliswork (Fig. 223). Bundles of osteogenic fibres develop, either in a ground substance, secreted by the cells which cluster round the calcareous needles, or in portions of the bodies of the cells themselves, and seem to serve as guides for the further deposit of salts. On the wall of each tube the peripheral cells, which have become large and angular, like osteoblasts, arrange themselves in a layer, and the central vessel is covered with an epithelioid layer. The osteoblasts at once begin to lay down lamellæ of true bone, some osteoblasts being included between them as bone-cells. Haversian systems are thus developed, the callus becomes truly bony, and as the space round the central vessel becomes constricted, the bone becomes more and more dense. The dark granular calcified portions are soon absorbed and replaced by bone. Meanwhile the Haversian canals of the fragments have been enlarged to a considerable extent by the ordinary process of erosion; when this ceases, osteoblasts deposit layers of bone here also, continuous with those in the callus, and thus absolute continuity is established.

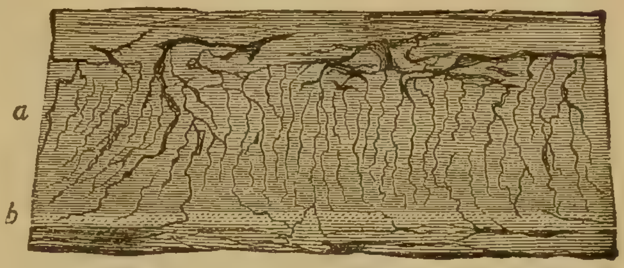


Fig. 222.—A longitudinal Section of Injected external Callus from a Rabbit's Tibia in the vicinity of a Fracture five days old. × 20. *a*, Callus with vessels running more or less vertically to those in the bone, *b*. (Billroth.)

This is direct ossification of callus, and it seems to be the only mode followed in the union of compound fractures in man. But in simple fractures, according to some observers, the callus becomes cartilaginous. Judging from specimens derived from animals, this occurs in the neighbourhood of the fracture, where, presumably, the irritation from movement is somewhat greater than at more remote parts. Cornil and Ranvier state that a clear matrix, not staining with carmine, appears between the round embryonic cells of the callus about the eighth to tenth day; the tissue thus assumes the appearance of embryonic cartilage. From the tenth to fifteenth calcification and ossification begin, following the physiological plan. The cartilage cells multiply and fill large spaces; these open into one another, giving rise to still larger spaces—primary areolæ—the walls of which calcify. Beneath the periosteum, and close to the bone, a layer of vascular medulla remains, and this bursts through at various points into the primary areolæ. The cartilage cells disappear before the medullary. Osteoblasts speedily line the cavities, and proceed to lay down bone in the usual way. The periosteal and intermediary callus become cartilaginous, but in

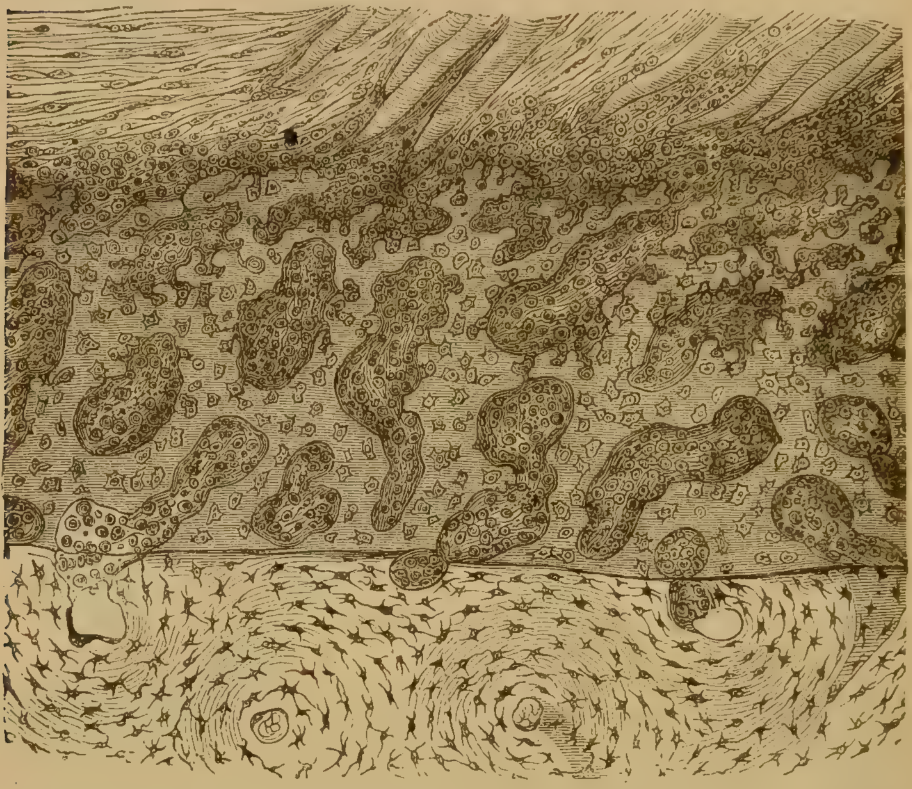


Fig. 223.—A transverse Section of Callus upon the compact Wall of a long Bone Below is seen the compact bone; its Haversian spaces are becoming enlarged, and the line between it and the subjacent callus is thus rendered irregular. The callus shows many large spaces, containing vessels (not shown), and numerous osteoblasts, in a calcified matrix. No Haversian lamellæ, regularly deposited on the walls of the cavities, are shown, and the bone cells scattered through the matrix exhibit no regular arrangement. The round-celled infiltration on the surface is pushing between the connective tissue and muscle fibres, and is being invaded on the other hand by bony spicules. (Billroth.)

The earlier the injury, and the more important the epiphysis, the greater will be the shortening. Cases of five and six inches' shortening in the humerus, after early separation of its upper epiphysis, have been recorded, the arm being otherwise well developed. When growth is arrested in one of two parallel bones, however, the result is more serious, for the other necessarily curves as its length increases.

Separation of an epiphysis may be *compound* or *complicated by the various injuries to soft parts* given under fractures. Irreducibility is generally due to the shaft being caught in the hole in the periosteum, through which it has protruded, or to the slipping of a tendon between the fragments. In compound cases it has occasionally been found necessary to saw off the lower end of a protruding shaft; in simple cases, otherwise irreducible, an open operation would often be indicated, either to avoid arrest of growth, improve function, or relieve surrounding parts from pressure, the epiphysis being pinned to the shaft if necessary.

Displacement of the fragments in fractures is very variable and important—for displacement gives rise to deformity, one of the best signs of fracture. It may prevent the eliciting of crepitus and abnormal mobility in the length of a bone; and it may prevent union by separating the broken surfaces. Unreduced displacement may impair or annul the utility of a part.

There may be no displacement; this is most common in incomplete or transverse toothed fractures, in fractures of one of two parallel bones, and of bones covered with dense fibrous tissue, which sometimes escapes untorn.

As a rule, the fragments are displaced in one or more of the following directions:—

(1) *Angular displacement* is very common, and does not cause any noteworthy separation of the fragments. It is the only form possible in incomplete (Fig. 205), subperiosteal, and deeply-toothed fractures; it is the usual displacement in simple transverse fractures, and, coupled with overlapping of the fragments, is frequent in oblique fractures.

(2) *Lateral displacement* alone is somewhat rare, and occurs in transverse or nearly transverse fractures (Fig. 217). When incomplete, the broken surfaces are not entirely separated; but when complete, longitudinal displacement, separating the raw surfaces, is almost certain to be added (Fig. 214). Some lateral displacement accompanies the longitudinal displacement usual in oblique fractures: whether the broken surfaces are separated in this case depends on the amount of shortening.

(3) *Longitudinal displacement* includes several varieties:—(*a*) Overlapping of the fragments—which lie side by side instead of end to end—is the commonest. It is usually present in oblique fractures (Fig. 288), also in transverse fractures after complete lateral dislocation (Fig. 214). The surfaces are more or less completely separated, and the limb shortened. Sometimes, in comminuted and in Y-fractures, the

localised suppurations result. The presence of these severe and prolonged inflammations is necessarily accompanied by increased vascularity of the part; the periosteum receives its full share of the increased blood supply, and lays down new bone abundantly (Fig. 224). Thus we always find a larger amount of callus in a septic compound fracture than in a simple fracture. New bone may be deposited in considerable amount even on an uninjured bone parallel to the broken one; other evidence of this increased vascularity is to be found in the extensive growth of hair upon the part. In the most favourable circumstances, inflammatory matting of soft parts to each other, and to the fracture, must be greater than in simple injuries, and it may be very widespread. The muscles degenerate and waste, mainly as a result of the inflammation, prolonged rest, and compression during treatment; portions of them or of their tendons may slough. Lastly, the presence of a septic wound opens the door to all the infective diseases of wounds—especially erysipelas, septicæmia, and pyæmia—which latter is always favoured by the presence of injured bone in the septic wound. Secondary hæmorrhage and late accidents to vessels are almost always of septic origin.

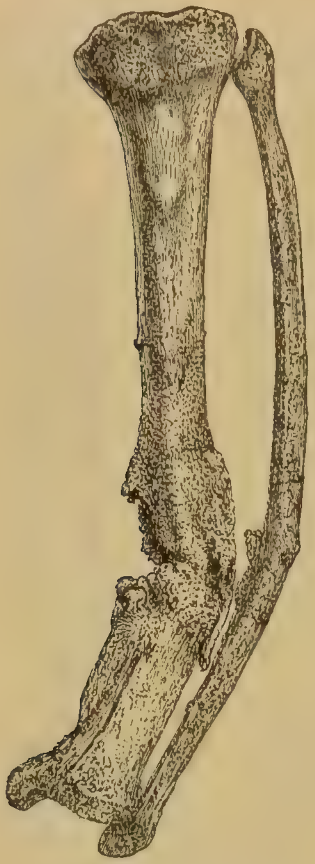


Fig. 224.—A compound comminuted Fracture of the Tibia and probably a simple Fracture of the Fibula at the Junction of the lower and middle thirds. The fibula is united and presents a long uniform curve outwards, consequent upon a shortening of the tibia due probably to the removal of bone. The ends of the tibial fragments are swollen by callus in which a single splinter is caught on the outer side. Most of the tibia and even of the fibula shows a thin layer of new bone.

Theory of the formation of callus.—The majority believe that the formation of bony callus is a reparative, not an inflammatory process, and that it occurs only in the presence of medullary cells, which alone can become osteoblasts. Transplantation experiments have shown that the cells of the deeper layer of the periosteum and, in a much feebler degree, those of the medulla, possess the power of forming bone—apart from bone itself; they apparently do so in fractures, producing the external and internal callus, of which the former is by far the more exuberant, for any effect which the bone and the cells in the Haversian canals may have should tell equally on both sides; lastly, Ollier carefully removed a zone of periosteum, and broke the bone—no callus formed till the edge of the periosteum was reached. This has not always been the result, but the evidence is sufficient to show the great importance of the periosteum in the formation of callus; and though it is impossible to demonstrate the multiplication of its cells, or to trace their progeny, yet the view above stated receives support from all observations on repair of tissues since Senftleben's experiments on the corneal cells, for all tend to show that the cell-elements of a tissue effect its final and perfect repair. Cohnheim, Maas, and others, on the other hand, regard the process of bone-repair as inflammatory, and the cells in callus as leucocytes.

There can be little doubt but that the early infiltration is inflammatory, and the cells chiefly leucocytes; but where is the evidence that leucocytes can form bone, or even the cartilage met with in callus? No scar, away from bone, shows such changes, whereas a scar in bone ossifies with practical constancy. To talk of the "influence of the mother tissue" does not render things clearer; there is no reason to think that bone can exert an influence save through its cells, which have throughout life the power of selecting lime salts from the nutrient fluid around them, and of laying them down in the form of bone. It is, therefore, *à priori* likely that the essential cells of callus are of medullary origin.

The formation of callus seems clearly to depend upon irritation; it develops very sparingly in fissures of the skull, but largely in extensive injuries, with much displacement, much mobility (ribs), and much septic irritation (compound cases).

The formation of *cartilage* in callus has always been connected by English pathologists with mobility; it occurs in the ribs, and occasionally in other simple fractures of man, and in most fractures in animals. The irritation of suppuration does not tend to the formation of cartilage. But v. Winiwarter in the 15th edition of Billroth's "Pathology" describes the formation of cartilage in simple fractures in man as usual, and so also do Cornil and Ranvier in their "Pathological Histology." In the absence of direct statements it is always difficult to determine how far Continental views are based upon observations on animals. Cornil and Ranvier, however, base their statement as to man upon the fact that they have always found cartilage in the callus of a considerable number of simple fractures due to senile atrophy, which they have examined. Exceptional mobility would very likely have been present in these cases, and, if it was, they would not prove the rule.

The time required for union of various bones.—This varies a good deal. The following times may be taken as averages. They will not enable us to dispense with a careful examination before permitting use of a limb which has been fractured, or with the need for caution in beginning to make use of the part.

Phalanges are said to require 2 to 3 weeks; metacarpal, carpal, metatarsal, tarsal bones, and ribs, 3 to 4 weeks; clavicle, fore-arm bones, and fibula, 5 weeks; humerus and tibia, 6 to 7 weeks; both leg bones, 8 weeks; femur, 10 to 12 weeks. By these times the bones mentioned are generally, after simple uncomplicated fractures, united with sufficient firmness to allow of use being commenced. Fractures unite more quickly in children than in adults.

PROGRESS AFTER FRACTURE.

Course of a simple fracture.—This should be uneventful. *Shock* is usually slight in limb fractures, but may even prove fatal in cases due to great violence, and in the old and feeble. *Pain* is not, as a rule, severe or lasting, but it may be both. Whenever severe it is probably due to a complication. During the first few days, starting pains—due to imperfect fixation—may be very troublesome, and together with a constant ache, may disturb sleep greatly. Much discomfort may be experienced from necessary restraint of apparatus, and acute pain in the back is not uncommon for a day or two when the dorsal position must be maintained. Generally pain soon disappears after the satisfactory setting of a fracture. *Tenderness* subsides steadily, and is little marked after ten to fourteen days.

Swelling may be rapid and great, or slight, and slowly increasing for twenty-four to forty-eight hours. The early swelling is due to hæmorrhage, and is proportionate to the number of vessels torn, those of bone always bleeding persistently; later, swelling is due also to inflammatory exudation from injured vessels, and to impaired lymphatic and venous return owing to rupture, compression, and thrombosis of these vessels. The swelling is diffuse, for the fluids spread along the areolar planes; it may be deep or superficial. In the latter case it pits, and all the colours of a bruise appear. Gradually absorption sets in, and the bones are felt, yet not clearly and sharp as at first, but more or less concealed by the callus-tumour, which steadily increases in size and definiteness. It is perceptible at the end of the first week, and fully developed and firm in the second to third week. When firm union has been effected, the finger traces the absorption of the callus, and the gradual return of the bone towards its normal form. Unreduced displacement of the fragments, and its effects upon the limb—shortening, bending, etc.—are, however, permanent.

Absorption of the extravasation is accompanied by certain general and urinary symptoms, viz. fever, very rarely an icteric tint, and the occurrence of bilirubin, fat, albumen, and casts in the urine.

Fever.—Among 168 cases of simple fracture at University College Hospital, 92 per cent. became febrile (Horsley), the temperature being sometimes only slightly raised, again reaching 101° or even higher within twenty to sixty hours, and falling to normal in three to fourteen days. The fever does not affect the general well-being, and, with marked exceptions, is higher the more extensive the bruising, and in those under than in those over forty.

This pure "traumatic" fever is not peculiar to fractures, but occurs after severe contusions, and is probably due to absorption of pyrogenous stuff from extravasated blood (fibrin ferment) or torn and inflamed tissues. Other causes are suggested—*e.g.* the effect of irritation of sensory nerves on thermogenetic centres, though experimental irritation is known to cause depression of temperature; fat-embolism and tension (Horsley)—both very doubtful.

A hæmatogenous jaundice connected with the absorption of blood pigment has been observed.

Urine.—Bilirubin, derived from blood pigment, is said to be frequently found; it yields a yellow-green fluorescence when the urine is shaken with $ZnCl_2$ and NH_3.

Fat-embolism.—According to Scriba and Riedel, fat in very variable quantity may be found in the urine after simple fractures, in as many as 42 per cent. of the cases, appearing two to four days after the injury, as a whitish layer on the surface, and disappearing in about twenty days. It is due to absorption of oil-drops from crushed fat and marrow cells; they are carried to the lungs (fat-embolism), where they may be demonstrated in the smaller vessels by staining sections with osmic acid; gradually they are swept on into the systemic circulation and to the organs supplied by it, and thus reach the kidneys and pass through the urine. Horsley and myself examined between us the urine of a large number of cases of simple fracture, but we never found fat; Horsley detected it in the sputum in two cases. Riedel further stated that albumen and casts (hyaline and brown granular) might be detected twenty-four to forty-eight hours after simple fractures, disappearing by the fourth to sixth day. In rare instances I was able to confirm this observation. Albumen and casts in the urine have been produced in animals by experimental fractures, injections of blood into the peritoneum, or by injection of fibrin-ferment.

Fat-embolism, to a greater or less extent, occurs in most fractures and contusions. It has been regarded as the cause of death in some obscure cases. The symptoms are said to be dyspnœa, with râles, low temperature, and death in coma. Cohnheim disputed the likelihood of the explanation, because large quantities of fat can be injected into the circulation of animals without causing serious symptoms, and even ligature of one pulmonary artery causes only momentary elevation of the blood pressure.

Suppuration of a simple fracture and *necrosis* are very rare. Suppuration has generally occurred after injury to the skin from direct violence, or in alcoholic, diabetic or albuminuric patients, or in connection with some boil, septic sore or ulcer; but in some cases no source of infection can be found.

Delirium tremens is less common than formerly, but is still not infrequent in alcoholic patients who become the subjects of fractures. They do not sleep from the first, often reply to questions in a quick jerky way, and their movements are tremulous; in two or three days the characteristic delirium appears. The great points are to procure sleep, to treat the patient, if possible, so that he shall not be kept in bed, but shall be able to get some exercise, and to put up the fracture so that the limb as a whole can be freely moved.

Acute mania may follow a fracture, as it may any other injury.

Wasting and stiffness of a limb are common on abandoning splints, and are easily explicable. Wasting is due to more or less complete rest of the part, and to the constant pressure of restraining apparatus. Stiffness to matting of parts round about the fracture, especially of tendons in their sheaths; to adaptive shortening of muscles, best seen when a foot has been allowed to drop; to adhesions in joints, even when they have not been injured; to permanent displacement of fragments, forming part of joint surfaces; to the formation of irregular masses of callus; or later, to the development of rheumatoid changes. Rarely, true anchylosis occurs (Figs. 327, 328).

Œdema.—As a result of the injury to and thrombosis of vessels, of the prolonged diminution of pressure upon their walls, and of the imperfect nutrition resulting from elevation (when a limb is slung or raised) and fixation, swelling of the limb—especially the lower—is usual on beginning to use it, and often very enduring.

Rheumatoid changes are often excited in the joints of those who are predisposed to rheumatism, by fractures near to or involving joints; and in such subjects a limb which has been the seat of a fracture may remain a more or less trustworthy weather-prophet.

The rule, however, is that, unless crippling displacement persists, recovery from a fracture is complete, and the limb in a few months is as strong and useful as ever.

Course of a compound fracture.—The course of an aseptic compound fracture is just as uneventful as that attributed above to simple fracture; except that the presence of a wound has its inconveniences and difficulties, it makes no difference to the result, unless, perhaps it delays healing a little. It is far otherwise with a septic compound fracture. The occurrence of suppuration almost certainly induces more fever than is usual in simple fractures; and retention of pus and spreading suppuration cause prolonged and severe fever. The exhaustion due to this, and to frequent anæsthetics and incisions for the evacuation of pus; the increased difficulty in fixing the part which results from the presence of a wound or wounds, which must be dressed; the more prolonged fixation which is necessary, and the consequently greater wasting of parts—for muscle does not benefit by increased blood supply, unless stimulated to contract; the widespread matting of tissues which follows upon the presence of a suppurating wound; and the fact that the presence of a septic wound exposes the patient to all the infective diseases of wounds—render a suppurating compound fracture an injury which may greatly impair the usefulness of the part, or may cost the patient his limb or his life, or both. It will be understood, of course, that suppuration of a compound fracture does not necessarily bring about the above consequences; the ill-results may be slight or most severe.

TREATMENT OF SIMPLE FRACTURES.

First help.—When a patient is knocked down and sustains a fracture of the lower limb, it happens only too often that the injury is rendered compound or complicated by an endeavour to rise and bear weight on the limb, or by improperly-managed lifting—the portion of the limb beyond the fracture being allowed to hang unsupported. In the case of the upper limb this danger is much less. Theoretically, then, a person whose lower extremity has been injured should be prevented from rising until it has been ascertained that at least the tibia and femur are sound. Should there not be time for this, the patient should be lifted to a place of safety—*one person taking charge of the injured limb only.* If the leg be injured it is best lifted by a person standing on its outer side, grasping it firmly above and below the point of injury, and raising it in one piece, at the same time maintaining slight "extension"; his elbows, bent at suitable angles, should, if possible, rest against the body; the lifting should be done by straightening the hips and back, and the extension by endeavouring to separate the hands grasping the limb.

Should a fracture of the femur be found, one person should support the whole of the limb beyond the fracture, raising it *pari passu* with the body to prevent angular deformity, and maintaining some extension by traction from the body.

Upper limb fractures are more easily dealt with, the limb being smaller and lighter; exactly the same rules apply when lifting is necessary. Shortly, the form of the limb should, as far as possible, be steadily preserved.

Means of transport must often be found. Most patients after fractures of the upper limb can walk, supporting the limb with the other hand, or having more or less of the fore-arm supported by a pocket-handkerchief sling, or, in cases of fractured humerus, having the injured arm fixed closely to the side by the coat buttoned over it or laced up with string, the hand being brought out and supported between the buttons. But those with fractures of the lower limb should be carried. If anything like a stretcher can be arranged, it will be sufficient, even when the femur or tibia is broken, to fasten the broken limb to the sound with handkerchiefs, above and below the fracture, to prevent movement of the fragments. Should a temporary splint be thought desirable, something efficient can generally be found by one who knows what is required to be done. Stout newspapers folded, corrugated paper, trelliswork flower-pot holders (Fig. 225), an umbrella frame, straw bottle-cases, and bundles of straight slender branches are some of the many things which may temporarily replace more orthodox splints. They must be applied firmly or they will be useless; some will require padding with any available soft stuff. Cold must be specially guarded against during transport after an accident.

Deliberate treatment.—The indications for treatment are:—

(1) to "set" the fracture; (2) to apply, if necessary, such apparatus as will maintain the fragments in position. There are a few preliminaries of general importance to be attended to. For a fracture of the pelvis or thigh the bed should be prepared by placing fracture-boards across beneath the mattress to prevent it from sinking under the weight of the pelvis. Clothes must be removed from the injured part with all gentleness, seams being cut up, boots being fully unlaced and drawn off whilst the lower part of the limb is most thoroughly steadied, etc. Till a fracture has been examined it should be assumed to be all but compound. The limb should next be well washed with flannel, soap, and hot water; if hairy, shaving will mitigate itching, and a wash with 1 to 1000 perchloride of mercury lotion will help in the same direction, by checking decomposition of sweat. Everything being in readiness for the application of retentive apparatus, the fracture should now be "set."

(1) **The setting of a fracture** means the removal, so far as is possible, of deformity due to displacement of the fragments. It should follow immediately on the complete examination, that the necessary pain may be got over in one bout. It is a principle in the setting and treatment of fractures that force should not be used to overcome resistance which may be conquered by art—*i.e.* by the relaxation of parts.

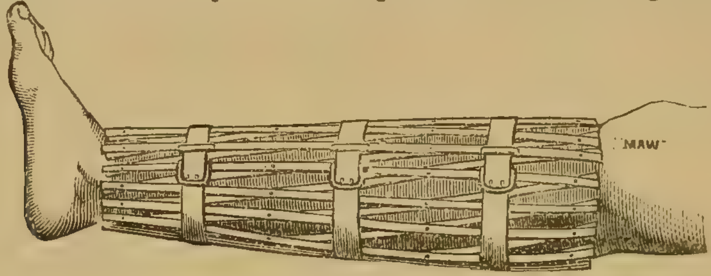


Fig. 225.—Wyeth's Universal Trellis Splint. It is the type of many easily extemporised splints.

Let us take, for example, the ordinary oblique fracture of both bones of the leg, with displacement upwards and backwards of the lower fragments, maintained by the great calf muscles, which contract directly an attempt—probably painful—is made to reduce the deformity. Here, with the patient on his back, the hip and knee should be flexed to 90° by an assistant grasping the thigh just above the knee, so that his thumbs press firmly on the quadriceps tendon. His hands should form a fixed point of counter-extension. Another assistant, grasping the foot, has raised it and the lower fragment correspondingly. Under the direction of the surgeon, the latter makes extension, and performs angular or rotatory movements of the foot and lower fragment. When the surgeon is satisfied with the result, as tested by eye, finger, and tape, the assistants maintain the *status quo*, whilst he applies the splints. Straightening of the knee should be very gradual, and may be altogether inadvisable. Fig. 254 gives another example of extension and counter-extension.

(2) **The application of splints.**—While the setting of a fracture fulfils the first indication of treatment, the application of some kind of apparatus is often necessary to fulfil the second ; and in the great majority of cases this apparatus is some form of "splint."

Movable splints are those which, being applied with ordinary bandages, are easily removed and reapplied ; they permit examination of the fracture, and can be tightened or loosened as swelling subsides or increases. They probably do not limit muscular action and blood supply so much as the immovable varieties. On the other hand, they must be tightened up every few days, as the bandages loosen and get untidy ; the disadvantage of this is much reduced by the use of webbing straps and buckles round the splints instead of or beneath the bandage. Movable splints are made of wood, iron, tin, perforated zinc, sheet lead, wire, poroplastic, guttapercha, sole leather, plaster of Paris. Wood is the material most commonly used, and there are few fractures which cannot be treated with fair result by anyone possessing a knife and a deal plank 4 in. wide by $\frac{3}{8}$ in. thick. Another 3 in. by $\frac{1}{4}$ in. is convenient for children. Gooch's "kettle-holder" splinting (Fig. 226) is often very useful for certain fractures. Special - shaped splints — such as Carr's, Cline's, Hodgen's, MacIntyre's—are used with more or less advantage. They are made in three sizes as a rule, and the aphorism that "shaped splints never fit anyone" is somewhat pessimistic.

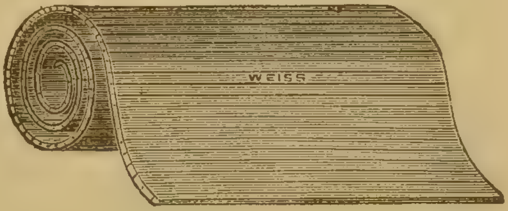


Fig. 226.—Gooch's "Kettle-holder Splint" Material

Most of the above forms of splints require padding. Pads are made of tow (which is cooler than wool and less liable to become lumpy), covered with linen ; but cotton is often used. The tow should be pulled into long wisps placed together, till the required dimensions are reached ; the edges of the covering are then turned in and oversewn. The pad should be a little larger than the splint it is to cover ; it is fixed to the splint by a zig-zag of stout thread passing from edge to edge across the back of the splint. A linen towel makes a capital pad ; so, too, does folded boracic lint. In addition to the principal pads, minor ones are often required, especially to prevent pressure upon prominent points. Wool or lint is often used for this purpose, and it should never be placed between the prominence and the splint, where it will only increase the pressure on the point it is desired to protect. To protect a bony prominence—*e.g.* the internal epicondyle—make a "ring pad," *i.e.* a thick strand of wool twisted into the form of a ring, the small central hole of which is to receive the epicondyle, while the ring of wool projects beyond it all round, like a corn plaster, and receives the pressure. A little extra padding round about may be necessary to distribute the pressure.

Shaped splints may be moulded for any case in wire, poroplastic, guttapercha, leather, or plaster; and very simple ones may be made of sheet lead.

If *wire* be used, the outline is made in stout telegraph wire, the ends being lashed with fine wire or, preferably, soldered (than which nothing is easier) and filed smooth. The interval is filled in with a trelliswork of light wire.

If *guttapercha, poroplastic*, or *leather* be used, a pattern in stout paper is made, the paper being laid on the part and cut to the required shape, pinched up and cut off where excessive in rounding convexities, notched where it must gape in passing concavities. A gap of at least 1 in. should be left between the edges where it grips a limb. From this pattern the splint is cut (Fig. 227), at first in general outline.

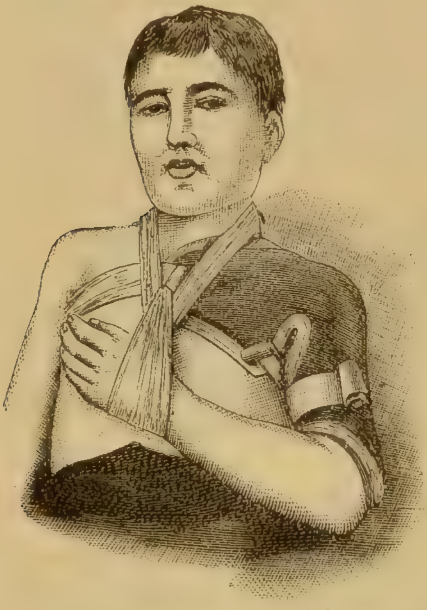


Fig. 227.—A Shoulder Cap cut from pattern and fitted.

Guttapercha is softened in hot water; if made too soft it gets sticky. It remains soft for some time, so there is no need for hurry in handling it. The limb must be thoroughly oiled, the guttapercha laid upon it folded over where it is in excess, carefully stretched where it is insufficient, and bandaged on. When it has acquired some firmness the bandage may be cut up, and the splint placed in cold water to hasten rigidity; or, if possible, the part and splint may be placed in cold water. Angles are now dealt with by the pressure of a hot iron, continued till the surfaces are smooth; where the material has been stretched, pieces of guttapercha must be stuck onto the outer surface. When firm, numerous holes should be punched through the splint, which is then lined with chamois leather. Such splints are not very rigid, and retain the sweat disagreeably.

Poroplastic may be softened in hot water, but rotation on a string in front of a fire, or, if large, heating in an oven, gives better results. It hardens quickly. The limb should be bandaged with thick flannel before the splint is moulded on. Before heating, excessive material must be cut out—too little rather than too much being removed. When soft, the edge of the material may be stretched a little, where a concavity has to be passed; but generally non-professional workers have to cut notches here which greatly weaken the splint. The bandage used to mould the splint must be applied very quickly, uniformly, and firmly. On removal, necessary modifications are made; intervals where pieces have been cut out are closed by sewing with stout twine; an endeavour may be made to strengthen the splint opposite concavities by sewing suitably-shaped pieces into gaps which have been cut; and lastly, the edge all round is shaved quite thin from the outer surface to prevent the fixing bandage from driving the edge into the skin. Numerous holes are punched through the splint, and it may be lined.

Leather splints are cut like poroplastic, except that, by patient stretching, there is somewhat more chance of escaping without a gap opposite a concavity. When cut out, the edges are trimmed, and softening is effected by twenty-four hours' soaking in cold water; or, if time is lacking, in water to which vinegar has been added, but this tends to render the leather hard. The splint, freed from superfluous water, is applied directly to the part and fitted accurately, a few stitches being inserted where pieces have been cut out. Over hollows, where a bandage cannot press, pads of cotton wool must be placed, and then a firm and careful bandage is applied. The part must be fixed for some hours, until the splint is dry; then the intervals are properly sewn up and, if necessary, bits inserted in gaps. The splint should be lined with chamois.

Webbing bands and buckles can easily be attached to the above splints. There is so much manipulation in fitting them, that they are not often used early in the treatment of fractures. Turning round a concavity is the difficulty with all three materials, of which leather is the most comfortable and best, unless quick hardening is essential —then poroplastic or plaster of Paris is needed.

Immovable splints are made of materials which harden; they do not become loose, except by shrinking of the part within them; they are more troublesome to put on and to take off than are the movable splints; when accurately applied they prevent movement very completely. The latter point was regarded as a chief merit, but there is reason to think that it is often a disadvantage.

Many materials are used, but we shall describe the application of three only—plaster of Paris, silicate of soda, and starch.

Plaster of Paris splints.—Plaster of Paris can be used in two different ways. (1) With roller bandages. These are prepared as follows:—A quantity of quite dry plaster of Paris is placed on a tray; a roller of "crinoline" or still more open muslin is thrown across the tray, drawn by one end through the plaster, which is well rubbed into its meshes and rolled up again as loosely as possible. In addition to the bandages there will be required a basin of cold water deeper than a bandage is wide; strips of tin 18 in. by ¾ in. punched through at every 2 to 3 in., the holes being left rough; a pair of strong shears to cut the tin; some rollers of boracic lint of suitable width. The tins are used to strengthen the splint without adding much to its weight; they are cut of suitable lengths, and bent to fit the positions which they are destined to occupy; the rough edges of the holes are turned outwards, and fix the tin by catching in the bandage; the corners are rounded off and the ends slightly turned up from the limb. The prepared limb, held carefully in position, is enveloped in boracic lint, no reverses being used, and every pains being taken to avoid thick folds and creases—especially over thinly-covered bones, where they are often a source of pain and sores. One bandage at a time is placed upright in the water, and allowed to remain there until all bubbling of air has ceased; it is then turned on to the side, each end is grasped by a hand, and it is lifted out of the water and pressed fairly dry; the end of the bandage is found, and thus it is handed to the surgeon. If the bandage be squeezed by one hand a great deal more plaster escapes. The surgeon applies the bandages lightly from just above the lower to just below the upper edge of the boracic lint, without reverses, and carefully avoiding the folds which result from the figure-of-8 bandage, as ordinarily used. This he does by making no endeavour to bandage continuously from below up, but rather allowing the bandage to follow its own bent. So soon as the lint is covered, the tins are placed in position, and caught by the next turns of bandage. Two or three thicknesses are required. Many now take a little plaster cream, and rub it on the surface; but, with well-prepared bandages it should be unnecessary, and it adds to the weight—a chief objection to plaster. If there be much tendency to displacement, the part should be held in position till the plaster is set; it should be left exposed till dry. Such a splint can be cut down fairly easily between two tins with a strong knife, a Hey's saw, or a pair of vine shears.

(2) The splints necessary to surround the limb are cut out of house flannel, coarse sackcloth, canvas, or some thick open material, and thoroughly soaked in water; two layers are generally sufficient, tins being placed between. When wet, the splints are carefully fitted to the limb. Opposite a convexity they are notched to the necessary depth, and the flaps are fixed in position by a stitch or two; opposite a concavity bits are sewn into the gaps. A thick plaster cream is now prepared by mixing equal parts by measure of plaster and cold water, the plaster being strewn into the water without stirring. In this the splints are kneaded, till they have taken up as much as they can hold; they are then placed in position, pulled quite smooth, and fixed on with an ordinary or a plaster bandage. This constitutes an immovable splint, but division of the bandage along a line of meeting of the splints permits their removal and reapplication with another bandage; the opposite line of meeting (if there be two) serves as a hinge. Another method, which is highly recommended, is to build up first one splint and then the other of long wisps of tow soaked in the cream; other materials may be similarly used. In one or other of the above ways, single shaped splints may be moulded to any part.

Some apply plaster of Paris directly over well-greased skin, others soak only the outer layer and rub cream into the outer surface of the inner. In slight fractures of the lower limb treated among out-patients, the patient's long stocking makes a comfortable covering for the limb, especially if it be woollen. A covering similar to the woollen vest generally used under Sayre's corset can often be found for other parts.

Silicate splints.—Waterglass or silicate of soda has the advantage of being much lighter than plaster, but it takes twenty-four hours to harden. The limb is similarly prepared. Two to four thicknesses of open-wove bandages soaked in a solution of silicate of soda are used in the way above described for plaster of Paris.

Starch splints.—The limb is surrounded with some antiseptic wool, ¾ in. thick by 4 to 5 in. wide, made into rollers and applied like an ordinary bandage. Outside this come splints of bookbinder's mill-board, cut of suitable shape, well soaked in hot water, thinned all round the edge by rubbing away with the finger the outer surface for an inch or so, and finally saturated with starch; a large iron tray takes most splints, but a bath is needed for those reaching from hip to foot. Thus prepared, the splints are placed in position and held by bits of bandage, which are removed as the starched bandage approaches them. Stout unbleached calico bandages are now pretty firmly applied after the following preparation:—They are soaked in water, wrung dry, drawn through hot starch, and then rolled up. It is a common practice to apply the bandage dry, whilst an assistant rubs in hot starch; the result is less perfect. Three or four thicknesses are generally used for the lower limb. The splint takes forty-eight hours or more to dry; it is fairly light and rigid; it maintains uniform elastic pressure on a part; it can be cut down, removed, and reapplied, wool lining being added should the part have shrunk, or some of the edge being cut away. Eyelets can be inserted if it is desirable to lace the splint.

In the hands of those who know how to use it, plaster of Paris is by far the most valuable of these three splints; its weight is the chief objection to it, and this may be greatly reduced by the use of light tin, trellis wire, or veneer strips. Some employ plaster in the treatment of fractures from the first, using only a light splint—easily removed should swelling require it, and easily reapplied. But the more usual practice in England is to wait until the early swelling has subsided, and until the tendency to shortening has disappeared, then to apply one or other of the above splints, and leave it on until union may be expected to have occurred. It has been charged against this treatment by Lucas-Championnière and others, that the long absolute rest thus enforced is responsible for the wasted and œdematous condition of the limbs, and for the painful adhesions which have to be dealt with on removal of the splint. This question will be again referred to under "massage" (page 774).

Other apparatus.—Splints are not the only apparatus employed in the treatment of fractures. In some no apparatus is of service, in others a sling or a few bits of strapping give such rest and contact to the fragments as is possible; in others, again, suitable position upon cushions or upon an inclined plane suffices; whilst still others benefit by the position given by a Salter's cradle or some other form of swing. Lastly, there are cases of simple fracture, in which the results of ordinary treatment being unsatisfactory, it is justifiable to inflict an aseptic wound for the purpose of directly acting on the fragments, by means of hooks, pins, sutures, or screws. This mode of treatment is most valuable, and should never be forgotten in cases of difficulty —cases of displacement, which cannot be dealt with by position and the use of apparatus, but which, if unrelieved, would perhaps become secondarily compound, or would recover with markedly impaired function or even with disfigurement. There must be good reason for converting a simple into a compound fracture, for it cannot be done without risk; the risk may be very slight in the hands of those who rarely have a failure from sepsis—it is incalculable in opposite circumstances, for not only the limb, but the life may be lost. The direct method of dealing with the fragments is adopted chiefly in fractures of bones which do not, as a rule, unite by bone; in cases in which a fragment is so displaced that it cannot be replaced, and will become a source of trouble; and in cases of very oblique fracture or comminuted fractures, in which other treatment has failed. It is a great advantage of this method that, once the wound has healed—sometimes even earlier—fixation can be abandoned in many cases, and the patient is able to use his muscles and joints. The results are often admirable, but sometimes, without failure of antiseptics, a joint which has been irritated by surgical manipulations becomes the seat of very troublesome adhesions.

Treatment by massage.—It is a fact that, after treatment by prolonged immobilisation, patients recover with wasted and œdematous limbs, often with adhesions in joints and tendon sheaths which have been injured, and which have been the seat of extravasation; also, that the early swelling is the cause of a good deal of tensive pain. It is stated by Lucas-Championnière and others that all these ills may be avoided by the proper use of massage, which has been widely adopted in England for sprains, but does not seem to have been much employed for fractures until after they have united, when the above-mentioned results must be dealt with. According to the above authorities, splints are an evil and are to be used only when displacement would result from their omission; in many fractures less restrictive methods may with advantage be adopted. The teaching with us has always been that perfect fixation is necessary to perfect union—that mobility is the great cause of non-union. As we have seen, in fissures of the skull, perfect fixation leads to the formation of very little callus and possibly to imperfect union; whereas callus always forms richly about a fractured rib, and non-union of a rib is practically unknown. But there is a difference in degree between the mobility in this case and in that of an untreated fracture of a limb, which sometimes results in non-union. Again, it is undoubted that massage will widely diffuse extravasations and effusions, and promote their absorption, and that it increases the circulation and nutrition of the tissues of a part; in these circumstances, callus production is asserted to be rapid and free. But, can massage be carried out without causing pain, excessive mobility, or displacement, which it is the object of splints to prevent? As to pain— the first point is to ascertain the seat of the strictly-localised fracture pain, and to avoid it in performing the massage. As to movement, the part is laid comfortably on something giving uniform support—*e.g.* a sandbag or, in some instances, the surgeon's thigh covered with a towel; little movement of the fragments then results from the massage. Under these conditions, it is said that comfort and relief of pain are always experienced from a *séance;* that the opposite effects may be taken as showing that the massage is too forcible, trenches too closely on the fracture, or is in some way improperly performed. The existence of a tendency to displacement is a serious difficulty; it is met by performing massage at once to diffuse the early extravasation, and then applying splints, which are removed after three or four days for a repetition of the massage, whilst the limb is held in position by assistants. The tendency to spasm and shortening is said to subside soon, and then regular *séances* can be undertaken.

The movements to be practised are of the simplest kind. With the limb well supported, light upward pressure is to be made with the thumbs, starting from the tender point; the fingers rest on the side of the limb, and both they and the thumbs are well greased with vaseline or olive oil. Even in the same *séance* it will be found that firmer pressure can be borne. At the end of each *séance* the patient should be encouraged to perform all the movements of the limb which are possible without producing displacement, the hands of the surgeon supporting the fracture; and where a patient fails through lack of resolution, the surgeon will execute passive movements. Each *séance* lasts a quarter to half-an-hour; it is repeated, at first, daily, or even twice a day until the early swelling is gone—then at longer intervals. Longer or shorter *séances* should be continued more or less frequently, so long as any part is painful or stiff. It is obvious that treatment of this kind must be carried out by a surgeon; it cannot, without danger, be handed over to "specialists in massage." A. Landerer quite recently has claimed that massage, begun so soon as there is no more fear of bending of callus, shortens the period of treatment to half or one-third, and enables the patient to use the limb properly in half the usual time, while wasting, œdema, and stiffness are avoided.

TREATMENT OF COMPOUND FRACTURES.

First help.—The first help to be rendered in cases of compound is similar to that recommended in cases of simple fracture, with the addition that no unnecessary meddling with the wound is to be permitted; if the part be not injured past recovery, the point of first importance in prognosis is the absence of infection of the wound. Bleeding must be dealt with by elevation or by pressure above the wound; pressure on the wound so often leads to infection and wide extravasation into the tissues. If transport to proper assistance will be short, it is probably best to leave the wound untouched, and not to reduce a fragment should it project and become soiled; it will act as a plug, which can be easily cleaned. If aid cannot speedily be procured, every endeavour should be made to render the wound dry. Unless most thoroughly washed, fingers should not be introduced into the wound, for they are great collectors of pyogenic organisms. If water can be boiled, and clothing can be sterilised by moist, or even by dry heat, the cleansing of the wound and the preparation of a clean dry dressing becomes simpler. It is certain that no thought or care expended upon the first treatment of a wound can be regarded as excessive.

Deliberate treatment.—In the deliberate treatment of compound fractures, the first question to decide is whether the case is one for primary amputation, for conservative treatment, or simply for care until death arrives.

The question of amputation.—Experience affords the only ground for forming an opinion as to whether a patient will or will not bear an amputation; and even the most experienced are often wrong. When in doubt, the treatment is that of shock, coupled with measures to prevent loss of blood and infection of any tissues which it is desired to save. If the patient rallies, amputation is done aseptically, infusion of saline solution being generally indicated. Statistics show that, in spite of "last chances" being given freely, primary amputation has become more and more successful. For example, Erichsen, some years ago, stated that he would never again, in an adult, amputate at the hip for injury. Of late years many successful cases have been reported. The great cause of death in these cases is shock.

When life is not threatened by shock, the question is, "Can the patient recover with a useful limb?" No doubt will exist when a limb, at or below a certain point, is crushed so that circulation in the distal part is impossible. A similar difficulty may arise from rupture of the main artery, a difficulty greatly increased by subcutaneous bleeding, compressing all the tissues through which collateral supply might be established, or by simultaneous injury of the main vein. In each case, gangrene is more probable in the lower than in the upper limb. The surgeon must consider each case on its merits, and decide whether or no something must be done; if he think that by incision and ligature of the wounded vessel, with removal of the main mass of extravasation, he can reduce the conditions to something approaching those prevailing after simple ligature of the same vessel, he will carry out this operation, but in such a way that amputation, should it become necessary, may be conveniently performed. But if the patient be old or feeble, the arteries rigid, the heart unsound, the extravasation when seen widespread, or the probability of deep-seated infection great, primary amputation at the seat of fracture will be the wisest course. Extensive comminution of a bone and fissures extending into joints necessitate amputation only when sepsis is added; in old days it was a frequent indication—especially in gunshot injuries,—experience showing that primary amputation gave the best results; but antiseptics, even in warfare, have changed this. Under these conditions careful disinfection will be given a trial. Great loss of soft parts, muscle and skin, or skin only, may necessitate amputation; but the success of extensive grafting operations must here be borne in mind. Two or more of the above conditions may be combined, the need for primary amputation being proportionately increased. Clear evidence of infection is always an important element in the decision.

Conservative measures.—If conservative treatment be decided upon, it is to be carried out somewhat as follows :—

Disinfection.—Immediately it is found that the limb is fractured at a certain point, and that a wound, possibly communicating with the fracture, is present, the wound is well douched with 1 in 20 carbolic lotion or other reliable antiseptic, and covered with a thick "guard" steeped in the same, and fixed by a turn or two of bandage. The splints which may be required, and the other things mentioned below, are got ready. The clothes are entirely removed from the injured limb, which is laid upon a mackintosh and towel wet with 1 in 40 carbolic lotion. The limb is thoroughly washed with a piece of gauze, soap, and hot 1 in 40 lotion, up and down from the guard; when the rest of the limb is clean, the guard may be replaced by one covering the wound only, and the washing carried out as far as is possible without washing anything into the wound. It is well then to shave a good area from the wound, and finally to sponge the limb with 1 in 20 carbolic lotion. The clean limb is now laid on another mackintosh and wet towel, and the wound is subjected to further disinfection, which must be thorough in proportion as the risk of infection —evidenced by time which has elapsed since the injury, presence of dirt and clothes in the wound, history of fingering of the wound, etc. —has been great. When a fracture of the tibia has been rendered compound by an endeavour to stand, and the point of the upper fragment has protruded through the skin, and has remained plugging the wound till the patient is seen, the chance of infection is slight, and a careful cleansing of the fragment and skin around with 1 in 20 carbolic lotion will probably be sufficient, without interfering with the depths of the wound; the fracture being reduced only after the point of the upper fragment has been cleansed. But when there is reason to fear infection of the depths of the wound, surgeons endeavour, by the introduction of antiseptic solutions into the cavity, to destroy any germs which may exist within it, and there can be little doubt but that it is the correct practice. For, although it appears to be true that organisms are absorbed with great rapidity from the surface of a recent wound, and that disinfection of a wound even immediately after its inoculation with a lethal organism will often fail to save the life of an animal, yet it is certain that pyogenic and septic organisms do not excite suppuration and putrefaction in a wound unless they remain in it enveloped in clot, etc. Probably suppuration is frequently prevented by the absorption and destruction by the tissues, or the elimination of germs falling into a wound. Accordingly, all obvious *foreign bodies and dirt*, blood and clot, should be removed from the wound, which is enlarged sufficiently to permit exploration of its recesses; for sepsis, with all its dangers, would certainly result from the retention of a bit of cloth. Then an antiseptic of suitable strength should be applied to the edges, sides, and depth of the wound. The stronger solutions are best applied with a bit of sponge or wool held in sinus forceps; the more dilute, in quantity by means of an irrigator or Higginson's syringe with a long fine nozzle—such as a glass female catheter. The pressure should be low, and there should always be ample space for the escape of fluid, lest it be injected into connective tissue planes. Sometimes parts which are very deeply infected are best treated by excision—*e.g.* the end of a protruding fragment or crushed edges in cases due to direct violence. An anæsthetic is often necessary for thorough disinfection.

The occurrence of *emphysema*, even widespread, does not involve extensive infection, for the air is filtered by the tissues of the wound, through which it is forced by movements of the part.

When the *bone is comminuted*, fragments found absolutely loose are removed, those still attached to soft parts are left. Theoretically, in an aseptic wound, loose fragments should not require removal, for it is unnecessary in simple cases, and pieces of bone can be transplanted. Nevertheless, such fragments often die, and either come away or require removal later. Even the ends of the main fragments, stripped of periosteum, not uncommonly die, and are cast off like any other sequestrum; so some would excise more or less of the end of a fragment from which the periosteum was widely stripped. There is a clear objection to the practice; a gap in a bone is likely to lead to weakness, deformity, or non-union, especially if it occur in one of two parallel bones, the other preventing shortening (Fig. 224). Where there is no alternative, then the second bone should be shortened by excision of a sufficient piece. The variation in frequency of necrosis in different statistics is so striking that one cannot help concluding that it is the less frequent the greater the success of antiseptic surgery in the clinic.

Another difficulty may have to be dealt with, viz. *hæmorrhage*. Oozing sometimes goes on even for days without there being a vessel of any size wounded; this is best met by elevation and uniform pressure over a gauze and wool dressing, covering a considerable area round about the wound. The case would seem suitable for the administration internally of chloride of calcium, as recommended by Wright. If the bleeding be too free to be regarded as oozing, and is not checked by the above remedies, search must be made for its source, the wound being opened up if necessary, and the fragments turned out as carefully as possible, so as not to increase the detachment of periosteum.

Reduction is effected as in a simple fracture, but assistance may often be rendered by a finger in the wound; difficulty in reducing a protruding fragment may necessitate enlargement of the wound, or even removal of the protruding portion.

When, in a compound fracture, there is *difficulty* in *maintaining reduction*, one or two holes should be drilled through the fragments, and wire sutures or plated screws inserted. Indeed, this is probably always worth doing when the fragments are so freely exposed as to be easily got at, the subsequent treatment is so much facilitated; but it is doubtful whether, without further reason, one would be justified in converting an uninfected puncture into a considerable wound, which, should it not heal *per primam*, would for a considerable time expose the patient to the possibility of infection.

Dressings and splints.—After the above treatment, the fracture is held in position whilst the necessary dressings and splints are applied. It is a general rule that splints should be placed outside the dressing, unless they can be entirely included within it. To fix a leg on an Arnold's or a MacIntyre's splint, and then apply a dressing about a wound, including the splint, is to court infection; for the discharge will almost certainly come through between the limb and the splint where it is not to be seen, remain moist, and liable to putrefy. When a solid gutter splint is used, even outside the dressing, there is always danger that discharge coming through towards the splint may be unnoticed and remain moist. Sometimes short metal side splints can be included from end to end in the dressing, and may be used; but if the dressing has to be changed, the splints come off with it, so they have no advantage in this respect over such splints as those above named applied outside the dressing. At each change of dressing the wound is exposed, the limb lifted by the surgeon (page 767), the old dressing is replaced by a new one and any re-padding of the splint done, and then the limb is laid down upon it. If well managed, everything being ready beforehand, the disturbance and pain are generally slight. But when there is tendency to displacement, when pain and nervousness result in spasm directly the limb is touched, it is a clear advantage to use apparatus which need not be removed, especially in the case of the heavy and powerful lower limb. Extension between sandbags is often useful. Thomas's knee splint without the leather apron, but with a plaster bandage round the bars and the limb running up and down from the dressing, is most valuable. The shoulder strap is used, and extension is generally useful. If a wide gap must be left between the plaster bandages, short lateral splints or a trellis splint can be buckled round the dressing. But, generally, a thick wool dressing and a firm bandage round limb and bars afford ample support. This is the most secure form of bracketed plaster splint. Hodgen's, Smith's anterior splint and the trellis splint are often useful. Many compound fractures are treated by the application of an ordinary plaster splint outside the dressing; discharge may come through the plaster and dry if not too profuse. Each surgeon will use that form of antiseptic dressing upon which he has learnt to rely. There is none better than the double cyanide gauze and salicylic wool applied in mass and firmly compressed; discharge may come through this dressing very freely before it requires changing. After twenty-four to forty-eight hours in an aseptic case, discharge has generally ceased, and dressings may then be left on for long periods.

Results of antiseptic treatment of compound fracture.— Makins and Abbott have given a very valuable summary of the results obtained at St. Thomas's Hospital in 322 compound fractures of the six long bones. The cases were under the care of many different surgeons, house surgeons, and dressers during the years 1881–1890; the point common to all was the attempt to carry out treatment based on the antiseptic principle. Obviously, similar results may be obtained by all who will take the necessary trouble. Of the 322 cases, 92 were of the upper (humerus, 54) limb, and 230 (tibia and fibula, 166) of the lower. Direct violence was the cause in 164, indirect in 107, whilst in 51 there was no cause stated. A joint was clearly implicated in 36 (9 per cent.), but the writers think this complication was more frequent. Of the 322, 12 (3·726 per cent.) died within 24 hours, 65 (20·18 per cent.) were treated by primary amputation, and 245 (76·08 per cent.) conservatively.

Of the 65 cases of primary amputation, 19 (29·23 per cent.) died—5 of 11 cases of fractured femur, 13 of 35 cases of fracture of both bones of the leg, 1 of 14 cases of fractured humerus. Most of the deaths were from shock and concurrent injury, but an amputation at the hip died from sloughing of the flaps, and two cases of leg amputation died of sepsis and pneumonia respectively. The high mortality, and the fact that only 3·7 per cent. died without operation, show that amputation was done whenever there was any hope of recovery.

Of the 245 cases living over 24 hours and treated conservatively, 17 (5·27 per cent. of 322, 6·93 per cent. of 245) required secondary amputation. The indications were general septic infection, 2; tetanus, 1; cellulitis, 2; suppuration of soft parts, 6; and of neighbouring joint, 2; necrosis, 2; and secondary hæmorrhage, 2—failure to obtain or to maintain asepsis was the fundamental cause in all cases. Of the 17 cases 3 died (17·65 per cent.).

There remained 228 cases (70·80 per cent. of the whole), which were treated conservatively throughout; of these 7 (or 2·86 per cent.) died. If two deaths from broncho-pneumonia—not septic—and ordinary pharyngeal diphtheria be subtracted, the mortality is reduced to 2·04 per cent. One death was from tetanus, and the four others from ordinary sepsis. When it is remembered that in pre-antiseptic days a large number of these conservatively-treated cases would have been subjected to primary amputation, that the mortality among those treated conservatively would have varied in different institutions between 25 per cent. and 70 per cent., and that a considerable number of those recovering would have lost the injured limb by secondary amputation, the above results show clearly the improvement effected by antiseptics. Of every 100 cases thought fit for conservation, treated as they were at St. Thomas's Hospital, 3·26 per cent. would die, and 5·71 per cent. would recover after amputation; more than 90 per cent. would regain a sound limb. It is worth noting also that of the 36 cases in which joints were implicated, 7 were treated by primary amputation, and of these 3 (knee cases) died; 2 needed secondary amputation, and 3 were excised. These, and all other cases treated conservatively, recovered.

Lastly, it is interesting to examine the complications which arose in the 228 cases treated conservatively. Pyæmia, septicæmia, and tetanus each proved fatal once; the septicæmic case had also delirium tremens. Erysipelas occurred 11 times (4·82 per cent.), with no death; cellulitis, 1; suppuration of a knee joint, 1 (fatal); secondary hæmorrhage happened 4 times; necrosis, 13 (5·70 per cent.); and gangrene of skin, 1. Every one of these complications is due to failure in the antiseptics; unfortunately it is not stated how often suppuration occurred. Non-union probably occurred in four cases, and an arterial hæmatoma in one.

Treves has recently recommended a very simple method of treatment, which has yielded surprisingly good results. The points in it are the following:—The limb is cleansed, the fracture reduced, and splints applied as soon as possible. The splints are wooden, well padded, and fixed with webbing bands and buckles, without bandages. The limb is always kept exposed to the air, to avoid contact with the hot, moist and foul air of the bed. The wound is dressed with a little heap of dry antiseptic powder—iodoform or creolin, the latter being preferable; more is dusted on as the powder becomes soaked, but the original crust is not disturbed for a week or more. In six years (1886–1892) Treves thus treated 61 cases—being all which did not die within twenty-four hours, which were not amputated at once, or which were not irrigated. Mr. Treves tells me that only cases in which he should otherwise have amputated at once were irrigated. Among the above 61, bone was sawn off in three cases, and in four instances delirium tremens developed. Of the 61 cases, 49 (80·32 per cent.) recovered without suppuration; 12 (19·67 per cent.) suppurated, 5 (8·2 per cent.) so badly that secondary amputation was done, without a death. All cases which suppurated were due to direct violence. One patient, a man of 69, died on the 28th day apparently of some chronic septic poisoning, his wound having healed *per primam.* Erysipelas occurred twice (3·27 per cent.); necrosis three times (4·91 per cent.). The numbers are small; but of every 100 cases thus treated, 1·64 per cent. would die, 80·35 per cent. would recover without suppuration, 19·67 per cent. after suppuration, and of the latter 8·2 per cent. would have undergone secondary amputation.

The method appears to depend for its efficiency upon—(1) the drying up of discharge into a scab or paste too thick to serve as a soil for bacteria; (2) upon the antiseptic power of the powder used; (3) upon the immobility secured, and the drainage—more or less free, according to the size of the wound—which is permitted. With regard to (2), we know of no reliable examination of the antiseptic value of creolin powder, but we do know that living germs may exist in numbers in either dry or wet iodoform. It seems likely, therefore, that dryness is the chief factor in the success achieved, as it was when Neudörfer similarly used plaster of Paris, and the surgeons of the Strasburg Hospital used sachets of castor sugar. Simplicity, absence of disturbance, and ease of examination are important advantages.

Treatment of septic cases.—If a compound fracture "goes wrong," treatment must be in accordance with the general principles of surgery. The most perfect fixation, the most complete rest, the best position to facilitate drainage, secure ease and diminish the blood supply to the part must be arranged for. If the skin-wound be small, the extravasation wide, and the infection severe, no time should be lost in cutting freely down to the fracture, and widely into the area of extravasation; large tubes or iodoform gauze plugs should be inserted, and, after bleeding has quite ceased, the inflamed part should be surrounded by a thick fomentation, wrung out of boiling 1 in 2,000 perchloride of mercury lotion, changed every three hours. The objects of this are (1) to ensure that there shall be no retention of infected fluids in the tissues; to provide that it shall be easier for such fluids and the germs present in them to run out into the dressing than to pass by lymphatics and veins into the circulation, causing general poisoning, or into adjacent connective tissue planes, there to excite suppuration—to obviate which evils drainage can hardly be too free; (2) to prevent further change in the discharge whilst in contact with the wound; (3) to dilate the superficial vessels over a large area, and thus reduce pressure in the inflamed area; and (4) to encourage granulation. Continuous irrigation where it can be established with a free flow—at least a pint a minute—of water at about 60° F. is another excellent plan of treating septic cases. It acts by washing away decomposable discharges, preventing the settlement of germs, and keeping down the temperature.

The temperature chart will be anxiously watched, and the limb be frequently examined for any indication of abscess or spreading œdema; sometimes suppuration goes on most insidiously, and considerable abscesses are unexpectedly discovered. With immovable splints it is difficult to make these frequent examinations; with movable ones, it may be hard to control the parts. The more widely our incisions are distributed, the more difficult is it to apply efficient dressings and splints. But, whilst the inflammation is spreading, our first object must be to control it, and deal with its results.

Ultimately, in bad cases, prolonged spreading suppuration, hectic fever, frequent incisions, and anæsthetics have so worn out the patient's strength and courage, and the limb has been so damaged, that both patient and surgeon are glad to end the case by a *secondary amputation.* In the course of such a case an uninjured joint may suppurate; but, generally, when a joint suppurates it has been involved in the fracture—perhaps by a fissure extending a long distance through the bone.

Septic osteomyelitis and septic arthritis are extremely grave complications, quite likely to be associated with pyæmia or septicæmia. Our chief hope lies in the most perfect drainage, not only of the fracture and fissure, but also of the joint; continuous irrigation may be of much value.

If the articular end of a bone has been fractured, and the state of the patient permit it, resection of the joint may be performed—for displaced and, perhaps, necrosed fragments are mechanical irritants, and septic discharge readily bags among them; but, in many cases, a careful consideration of the condition of the part, and of the patient's strength, will incline the surgeon to amputate.

The occurrence of pyæmia, septicæmia, or of acute spreading traumatic gangrene, must be met by high amputation; acute tetanus is probably best treated similarly. Erysipelas, by producing exhaustion by fever, by causing diffuse suppuration and sloughing of connective tissue and skin, perhaps by inducing suppuration of a joint over which it passes, may be a very serious addition to the patient's ills. Rarely secondary hæmorrhage occurs, and must be treated by ligature of the bleeding point if possible. Even in a sloughy wound, with our perfect means of hæmostasis, it is possible to follow up a bleeding vessel until a ligature will hold; but amputation will be preferred if the limb be much damaged, or if the ligature would seriously endanger the blood supply of the part beyond.

Necrosis, of greater or less extent, is the rule in suppurating cases; separation of the sequestra may be very slow; even repeated well-conducted operations may fail to find all, and those left may cause so much trouble in the way of abscesses, prolonged suppuration, fever, and albuminoid disease, of matting and wasting of muscles, that the limb has, after a long struggle, to be sacrificed. Vicious union and pseudarthrosis are unduly frequent results of compound fractures.

INJURIES OF VESSELS AND NERVES IN FRACTURE.

Wounds of arteries.—Recognisable injuries of considerable vessels are rare complications of fractures; yet, large arteries and veins are liable to injuries in both simple and compound fractures. In the latter, more or less of the blood from any bleeding vessel escapes externally, and the presence of a wound does away with any hesitation one may feel in simple cases when the question of ligature arises; otherwise—putting sepsis aside—the results of injuries of vessels in the two classes may be described together.

These lesions are three to four times more common in the lower than in the upper limb, owing to the greater frequency of comminution and laceration in the lower extremity, to the close relation of the femoral artery to the femur in its lower third, and of both the tibial vessels to the tibia—the anterior being sunk between the bones; and to the greater number of arteries in the leg of size sufficient to give rise to symptoms. Whilst the leg vessels suffer more often than the femoral (about 1 to 1·5), and the femoral is rarely injured above the lower third of the thigh, the axillary is involved as often as the brachial, and the fore-arm arteries very exceptionally.

The conditions met with are complete division, wound, rupture of the inner coats and consequent thrombosis, and simple compression, leading to thrombosis, by or between dislocated fragments or splinters. Each may be brought about by either direct or indirect violence, though rupture of the internal and middle coats is usually caused by direct violence crushing a vessel against a bone. The wounds present sharp or torn edges, according to the nature of the violence and of the fragments; in complete lacerations the ends may be sealed by the pulled-out external coat, just as when parts are torn off. Complete division and wound are about equally frequent—the chance that bleeding will be arrested being less in the latter case. Rupture of the inner coats from contusion and compression is relatively rare. The popliteal and the anterior tibial are almost the only vessels compression of which has been noted. The anterior tibial has occasionally been torn by stretching where it passes through the interosseous membrane. (*See also* p. 500.)

Few cases of compression or *wound of veins* only are recorded as complicating fractures; venous anastomoses are too free as a rule, the blood pressure too low to cause serious symptoms. But the addition of an injury of a main vein to an injury of the main artery may be very serious.

The results of the above injuries to vessels are:—(1) extensive extravasation into the tissue, with (2) more or less external hæmorrhage in compound cases; (3) the formation of an arterial or venous hæmatoma; (4) gangrene; (5) venous thrombosis and embolism.

When an artery has been wounded or divided, and the wound remains open, blood is pumped into the tissues, and forced to a greater or less distance up and down the limb—the distance varying with the blood pressure, the laxity of the areolar planes, the size of the wound, and the tendency of the blood to coagulate and form a wall to the extravasation. Under unfavourable conditions the limb becomes more and more swollen, especially in the neighbourhood of the fracture, its surface cold and pale or bluish, mottled by vessels containing stagnant blood, or by actual extravasation; if there was a pulse beyond the fracture at first, it disappears as the tension of the limb increases; colour beneath the nails disappears slowly under pressure, and returns even more slowly; the early tensive pain dies away, and is replaced by increasing insensitiveness. Moist gangrene is imminent. Under opposite conditions the swelling spreads and increases slowly, and is ultimately brought to a standstill; the circulation in the distal parts, though much impeded continues sufficiently to assure the surgeon that recovery is possible; gradually the circulation improves, and the general swelling subsides; then, perhaps, a circumscribed fluctuating swelling is discovered in the region of the fracture, and in this thrill, expansile pulsation, and systolic bruit may become evident. This is an *arterial hæmatoma.*

Between these two examples there are cases in which the most experienced will be in doubt as to whether circulation in a limb can be restored. The diffuse extravasation above described sometimes develops slowly, or rapidly after some days, owing, probably, to shifting of a clot or of a plugging or compressing fragment. Again, there may be no excessive primary swelling, but after some weeks, generally on the removal of the splints, a swelling is found at the seat of fracture, which may have all the signs of an aneurysm; but it may have none, the skin over it may be thin and red and fluctuation distinct; more than once a knife has been put into such a swelling in the belief that it was an abscess. In these cases of circumscribed arterial hæmatoma of late formation we can only surmise as to why there was little early extravasation or why the extravasation became so perfectly circumscribed: thus, a late wound or the shifting of a clot is possible at a time when the tissues are matted about the fracture and clot fills their interstices, when the limb is constantly at rest, uniformly supported, perhaps suspended, and arterial supply is at a minimum.

If the fracture be compound, but the wound small and the track to the wounded vessel long and oblique, there may be little *external hæmorrhage;* or it may be free at first, and cease under simple treatment, or none, as the wound fills with clot; the wound may heal, or more or less severe bleeding may recur at intervals; and, in either case, one or other of the above conditions develops from diffuse or localised hæmorrhage into the tissues. On the other hand, if the fracture be freely compound, the blood escapes externally instead of into the tissues.

Where arteries become closed as a result of laceration or rupture of the inner coats or of compression, and little bleeding occurs, the pulse will be missed below; collateral circulation may be established, just as after ligature at the point; but gangrene may occur after ligature, and is obviously more probable with laceration of surrounding tissues and compression by extravasation of collateral vessels. (*See also* page 536.)

The dangers of sepsis in these cases are many and obvious; *secondary hæmorrhage* from sloughing of the artery is specially likely in cases of undiscovered and unrelieved compression.

Wounds of *large veins* have caused huge hæmatomata, threatening or inducing gangrene; in one case (Erichsen) compression of the subclavian by a fragment of the clavicle caused gangrene.

Treatment.—In a freely *compound fracture with a bleeding vessel*—artery or vein—at the bottom of the wound, no one would hesitate to tie both ends above and below the opening. The treatment should be exactly the same when, in a compound fracture, the diagnosis of hæmorrhage into the limb from a considerable vessel is made. Enlargement of the existing wound will serve for more thorough disinfection, examination, and suturing of the fragments as well as for the securing of the vessel. If it become evident that the vessel can be better reached through another incision, this will scarcely add to the danger of sepsis. In such cases the circulation should be arrested, all clot should be removed from the wound by rubbing with sponges and scraping with a spoon, and as much fluid as possible should be expressed from the opened areolar planes to relieve collateral vessels from pressure.

In the case of a *simple fracture with free hæmorrhage* going on into the tissues, unwillingness to render the injury compound is natural, yet, as we can with great probability of success guard against sepsis in a wound inflicted by ourselves, and as this wound will very likely enable us to confer some advantage upon the patient besides that of securing the vessel, it is wise when the diagnosis of progressive hæmorrhage from a main artery can be made, to operate so soon as we can convince ourselves that the bleeding is not becoming limited in a natural way. To wait till gangrene is imminent, and till the limb is tensely filled with blood and clot, is to render the discovery of the vessel at fault and the restoration of the circulation most unlikely. When a case is first seen in this condition the surgeon may feel inclined to attempt to save the limb. He will probably fail, gangrene supervening even should he secure the bleeding vessel; very likely he will not find it if the fracture is in a part like the upper third of the leg, where either of three or four vessels may have yielded the extravasation. Early and accurate observations on the point of appearance of swelling and the pulse beyond in the different vessels may be of much help in determining which vessel is injured —hence the rule to feel the pulses beyond a fracture in all cases.

Whilst observing a case in which deep bleeding is feared, the limb should be elevated; some have recommended digital compression of the main artery for half to one hour in the hope that the bleeding vessel may be closed by clotting. When tension in the tissues is not already great, moderate uniform pressure by bandage over thick cotton wool, leaving the fingers or toes exposed, might limit the extrasavation; but there is an element of risk in it, and where tension is already great, no outside pressure must be used. The condition of the circulation in distal parts, and frequent comparative girth measurements of the limb are the points to which attention should be paid. When absence of distal pulse is noted without excessive swelling about the seat of fracture, exact setting of the fracture must first be seen to, to eliminate (as far as possible) compression. Compressing splints and bandages should, if possible, be avoided for a time. The limb should be wrapped in cotton wool and kept slightly elevated, to facilitate venous return; this may be aided by occasional massage in the upward direction. Should gangrene result from simple closure of an artery it appears early.

A circumscribed *arterial hæmatoma* may seriously impair the blood supply to parts beyond, it may increase and tend to burst, or suppuration may occur in the tissues round about it. It should be treated in these circumstances by free incision, whilst the circulation is controlled, turning out of blood and clots and double ligature of the supplying vessel. But should it not be a source of danger, the fracture should be treated, and, during the necessary rest, the hæmatoma may consolidate. Should it not do so, central digital compression of the main artery may be tried, with fair hope of success. This failing, the operation last mentioned should be carried out after union of the fracture is complete. Ligature of the main trunk at a point of election has succeeded, but the danger of gangrene is probably greater than in cases of true aneurysm; the cure of the hæmatoma is doubtful; there is in these cases no disease, but only an injury of the artery, and we can now so thoroughly control the circulation in most parts as to be able to perform deliberately operations which formerly were often matters of desperate haste.

Secondary hæmorrhage from a compound fracture which is not septic is almost as rare as late deep bleeding in a simple fracture. The sloughy condition of the wound constitutes the special difficulty in finding the bleeding point, and in getting a ligature to hold; this is best met by passing a probe along the vessel, and dissecting along it till a sound spot is reached. If the limb be already seriously damaged, this complication will raise the question whether it is worth preserving.

Venous hæmatoma should be treated on the same lines as the arterial form when it threatens mischief, though it must be admitted that the difficulty in dealing with one from the subclavian vein would be great. Compression by a splinter of the clavicle could certainly be directly dealt with.

Gangrene after fracture.—It will be convenient to summarise what has been said of gangrene in connection with fractures.

Gangrene may result (1) from the original injury to soft parts; (2) from constriction and compression of the limb by bandages and splints; (3) from inflammation; (4) from injuries to large vessels.

(1) It is not always possible to tell whether a part will recover, and it is right to give it the chance of doing so; in such a case the patient or his friends should be told the state of matters.

(2) The application of a bandage directly to the limb, or of some apparatus constricting the limb circularly, especially a plaster splint, either too tightly at first, or so that it becomes too tight owing to early swelling of the part, has been a frequent source of gangrene, especially of the fore-arm. So, too, has flexion at the elbow *after* the application of a bandage to the limb. The use of firm circular pressure round the upper part of a limb without giving support to the distal part might act as a cause. In these cases the surgeon would be to blame, first for the errors in treatment, and secondly for neglecting to watch for or failing to perceive the signs of impaired circulation in the digits.

(3) Spreading traumatic gangrene is fortunately rare, yet a compound fracture, not as a rule a severe one, has been one of its commonest starting points. Diffuse cellulitis, erysipelatous or "simple," may cause extensive sloughing. The surgeon may be to blame for allowing the wound to become septic.

(4) Closure of a main artery by complete laceration, rupture of its inner coats with thrombosis, or compression and thrombosis, complicated always by more or less laceration of soft parts and extravasation; division or wound of an artery with extensive extravasation; division of main artery and vein; very rarely, wound or compression of a main vein—form a group, including the most interesting causes of gangrene after fractures. (*See also* pages 132 and 147.)

Treatment.—Common sense suggests the measures to be taken to *prevent* gangrene from many of the above causes. The treatment is amputation, so soon as it is clear that gangrene must occur or has occurred. It must be performed high enough to render it probable that the flaps will live and be free from any infection which may have occurred. In gangrene from wound of main vessels, with which we have been unable to deal, amputation cannot be done lower than the wound. When the vessel has been secured, circulation may be restored to a greater or less extent in the threatened part, and this may be the case also in gangrene from thrombosis after injury; amputation must then be done above the gangrene in well-supplied and non-infected tissues.

Venous thrombosis and embolism in fracture.—Venous thrombosis must occur in every case in which veins are torn or bruised; it excites attention only when it spreads widely. The reason why some thrombi spread is unknown. It appears to be much commoner in simple than in compound cases, and is certainly far more frequent in the lower than in the upper limb, a fact which is probably connected with the slower circulation, tendency to varix, and degeneration of vein-walls in the former. As deep veins can only occasionally be felt when thrombosed, no diagnosis can be made till the thrombi in them are sufficiently extensive to excite marked œdema. This generally appears between the first and fourth weeks after the injury, and more or less suddenly; the swelling may be very considerable, involving a whole limb or more of the body; it is firm, pits little, and often last weeks or months—until the clot clears up or becomes canalised. (*See also* page 561.)

Embolism of heart and lungs is the great danger of thrombosis, and is fortunately rare. It is usually a thrombus from the lower limb which shifts, either without obvious cause or in consequence of some movement or manipulation; the clot is carried into the right heart, where it may stop, but it generally passes on into the pulmonary artery or its branches. The embolus may be quite small, or consist of a cylinder many inches long, coiled upon itself, and bearing the impressions of the valves in the vein in which it was formed. The smaller emboli produce the well-known morbid appearances of infarction; the larger cause sudden death.

As a rule, there are no premonitory signs other than, perhaps, œdema of the limb. The patient may be suddenly seized with fatal syncope. More commonly there is a short period of intense alarm; cries are uttered, "I'm dying," "I can't breathe," and the like; the face is pale and expresses terror, air is entering the lungs freely, the heart is palpitating violently; the patient struggles for a minute or two—then all is over. In the minor cases the symptoms are similar but less severe, and they pass off. From such patients we may learn that they felt something move from their leg and pass up into their chest. The attack may be followed by brick-red expectoration and signs of a patch of consolidation.

Injuries of nerves in fracture.—These appear to be more common in connection with fractures than injuries of vessels. They are far more common in the upper than in the lower limb, because in it the musculo-spiral (injured in more than one-third of all cases), brachial plexus, and ulnar nerves (Fig. 253) lie close to bones at spots where fractures are common, and more frequent in simple than in compound injuries, probably because the latter are unusual in the arm. In the lower limb the peroneal nerve is most frequently involved where it lies on the neck of the fibula; then comes the anterior tibial.

Injury of a nerve may be due to direct violence or to a displaced fragment; usually it occurs with the fracture (primary lesion), but occasionally compression is due to late displacement of fragments or splinters (secondary lesion), or to involvement in callus or scar tissue.

By far the commonest primary lesion is a contusion, which may, however, amount to a crush, destroying physiological continuity more or less completely. In rare instances a nerve may be more or less completely torn through, or pierced by a splinter driven into its substance, or jammed between the fragments, or drawn tight and kinked over the end of one which is displaced. (*See also* page 689.)

Symptoms.—The symptoms of these lesions must be:—impairment of function of the injured nerve, varying from slight interference with, to complete loss of, conducting power of sense and motion; and pain from the injury, which is rarely, if ever, of the nature of a clean cut, and which consequently is likely to cause somewhat prolonged aching. In cases in which a nerve is partly torn through, transfixed by a splinter, caught between the two fragments, stretched over the edge of a displaced fragment, or contained in a rough-walled canal, pain may be constant and agonising (in several cases of old date it gave rise to amputation), or it may occur in violent shocks with certain movements. But these irritant lesions have worse consequences: symptoms like those of tetanus, and resulting in death, have occurred even in simple fractures; an ascending neuritis reaching even to the cord and ending in tabes has been traced lately to lesions of peripheral nerves; and prolonged neuralgia, paralysis, wasting, contraction, glossy skin and other trophic lesions have not uncommonly resulted. Division of a nerve, actual or physiological, is followed by its degeneration, and, in case of a motor nerve, by rapid atrophy of the muscle supplied and development of the "reaction of degeneration." If physical continuity persists, the length of nerve fibres destroyed is not great, and the nerve is relieved from all pressure of displaced fragments, it is likely that in six to eight weeks sensation will return, the muscles will react better to galvanism, and then to stimulation of the nerve trunk *above* the lesion. If, in the course of four to six months, no regeneration occurs, it may be concluded that there is some obstacle to it, such as section and separate healing of the ends, or a considerable length of scar tissue.

Diagnosis.—The distribution of the nerve affected; the extent to which its function is impaired; the severity of the pain—constant when the cause acts constantly, intermittent when it is due to certain movements, active or passive; whether function improves and pain diminishes, or *vice versâ;* whether wasting of certain muscles and reaction of degeneration appear early and progress rapidly—these are the points upon which we endeavour to base a conclusion as to the physical state of the nerve.

If movements are not performed as part of the treatment of every fracture, it will often happen that loss of motion will not be noticed till after union of the bones; it will then be difficult to decide whether the nerve lesion occurred early or late, whether it was a primary contusion or a compression by callus. Electrical examination is of no assistance: it will not tell whether a nerve is divided or compressed—the axis cylinders being sound.

Treatment.—Acute pain should lead to careful examination and setting, under an anæsthetic if necessary. This failing to relieve, the nerve should be exposed, freed from pressure or resected and sutured if much damaged. With tetanus in view, much time should not be wasted over anodynes. If no recovery of function occurs in three to four months, the nerve should be exposed, resected, and sutured. While awaiting recovery, galvanism two or three times a week and massage should be used to prevent as far as possible wasting of the muscles; but if there is any sign of neuritis, neither of these should be practised, at least near the injured nerve, which then requires rest, moist warmth, and belladonna locally; later, counter-irritation along its track.

DEFECTIVE UNION IN FRACTURE.

Delayed union.—In about one in eighty cases the average periods (page 763) required for the union of various bones are markedly insufficient—union is "delayed" for weeks, months, or even years: on removal of the hindrance, union occurs.

J. Hutchinson, junr., recently quoted a case of ununited fracture of the radius, of four years' standing, in a syphilitic patient; without much hope of success, iodide of potassium was exhibited, and bony union occurred. Presumably, a gumma had been active during the period of non-union. This case is very exceptional; unless some cause of delayed union has evidently been acting all the time, we may feel sure that bony union will not occur after a year. An anatomical examination much earlier than this would often show that there was no chance of bony union. In some cases *callus forms slowly and in small quantity*—either on account of general depression or because the stimulus of the injury has been slight, as in fissure of the skull: mobility remains, the callus tumour is small or absent, the ends of the fragments, if superficial, remain sharp and clear to touch. In other cases the *callus tumour forms well, but does not ossify firmly* for weeks or months: on removal of splints more or less mobility or excessive yielding is noticed, and deformity ensues if the limb be used.

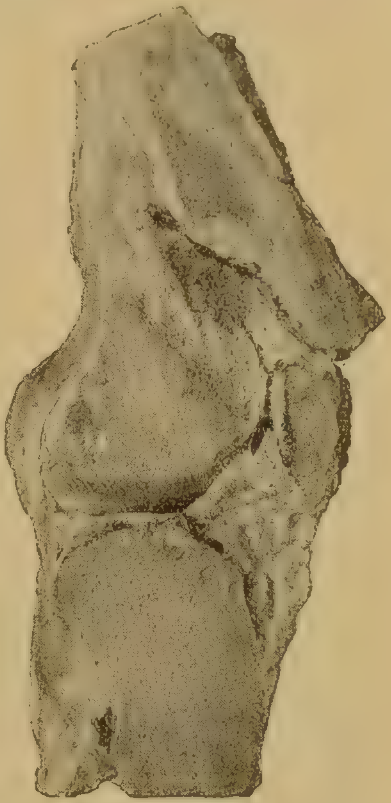


Fig. 228.—A longitudinal Section of an oblique supra-condyloid Fracture of the Femur. No union has occurred, the upper fragment having pierced the quadriceps tendon which is seen between the two fragments above the patella. (St. Bartholomew's Hospital Museum.)

Pseudarthrosis. — This is a general term used to indicate the permanent presence after a fracture of abnormal mobility in the length of a bone due to (1) fibrous union, (2) non-union, or (3) false joint. Pseudarthrosis occurs once in 200 to 250 cases; at all periods of life, even intra-uterine; more often in men than in women (5·5 to 1); it is most common from 20 to 30 years, but common from 20 to 50; it is uncommon in childhood and old age in proportion to the fractures occurring. Fractures of the humerus and femur yield an excessive number of delayed unions and pseudarthroses, those of the leg-bones about a normal, and those of the fore-arm bones less than a normal share.

1. **Fibrous union** is by far the commonest variety. Callus forms, but fibrous tissue with little bone develops from it. Ultimately a long and loose, or short and dense fibrous bond unites the fragments, the ends of which may be rounded off or rendered irregular by bony stalactites; displacement and mobility may be very great or very slight, the utility and wasting of the part varying inversely as the mobility.

2. **Non-union** is much less frequent. There is little or no bond between the fragments; they are widely separated by the intervention of some mass of tissue (Fig. 228), or loss of substance in one of two parallel bones has left a considerable gap, or the end of

one fragment touches only a sound surface on the other. Each fragment is healed and rounded off. Mobility may be extreme ("flail-joint"); and uselessness and atrophy of the bones are proportionate.

3. **False joint** is the rarest form. It probably develops from a close fibrous union of a roughly transverse fracture. The new joint is of the ball-and-socket type (Fig. 229), one fragment being hollowed out, the other rounded so as to fit; the surfaces are smooth and covered with dense fibrous tissue, fibro-cartilage, or even hyaline cartilage; a dense fibrous capsule, fairly distinct from the surrounding soft parts, holds the ends together, and its smooth inner surface lined with flattened endothelium secretes a synovial fluid. The resemblance to a normal joint is close, and is increased by liability to diseases of joints—as subacute synovitis and particularly rheumatoid arthritis, all the signs of which may be found.

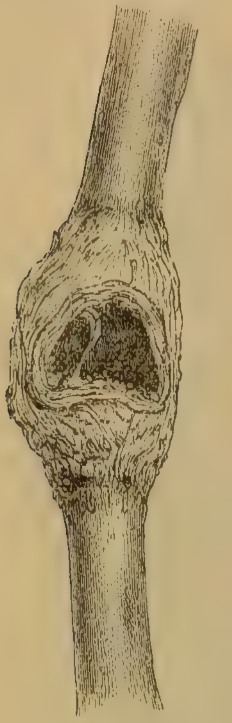


Fig. 229.—A false Joint of the Ball-and-socket Type. The two fragments are held together by a dense fibrous capsule, the interior of which is in parts fasciculated, and gives attachment to clusters of small pedunculated bodies. Otherwise the inner surface is smooth and shining. The end of the upper fragment is slightly concave, that of the lower convex. (University College Museum, No. 62.)

Causes of defective union.—This is a difficult subject. Frequently no cause is discoverable, the subjects seeming to be in perfect health. Again, obstacles which in some cases appear to be causes of non-union, in others have no obvious effect—even though they are well marked. The causes are general or local.

The general causes produce general depression, and it is thought impair the bone-forming function; they are frequently present without observable effect. *Starvation*, as many observations on besieged troops show, often delays union: so also does *hæmorrhage*. *Pregnancy* and *lactation* have a very doubtful effect. *Acute febrile illnesses* may delay union for months, or cause absorption of callus formed before their onset, as has been specially observed in the case of pyæmia. Callender made the interesting statement that he had never seen delayed union in a child from scarlatina, nor in any patient from erysipelas. Any *chronic wasting disease* might be suspected as a cause. *Syphilis* in its early stages has little or no effect; possibly when producing cachexia it may act more strongly, but gummatous infiltration of a bone is the most likely way for it to exert its influence. Sometimes antisyphilitic treatment is efficacious in bringing about union—again, it has no effect; but this is hardly an argument against the influence of syphilis; for treatment directed against clearly syphilitic lesions may be quickly successful in one patient but without effect in another. Both syphilis and fracture are so common, that they must often coincide without evil result. *Scurvy* in former days was looked upon as a cause of delayed union or of absorption of young callus. Severe *rickets* often delays union. *Chronic Bright's disease* and *diabetes* have also been regarded in this light. *Defective innervation* is an alleged cause. Ataxic fractures may unite feebly or not at all. Bognaud collected six cases of paraplegia from fracture at or below the twelfth dorsal vertebra, in which fractures of the leg bones failed to unite; when the spinal lesion was higher, he found that union occurred. But objection may be taken to most, if not to all, of his cases of non-union Many cases of good union in paralysed limbs may be adduced, and callus formation is found to proceed better in limbs of animals paralysed by section of their nerves than in normal limbs.

In a larger number of cases of pseudarthrosis, excellent health has been noted, rendering it likely that **local causes** were to blame. This becomes almost certain when in multiple fractures all but one have united; or when one of two parallel bones fails to unite. From a collection of 400 cases, Bruns finds the following to have been conditions unfavourable to union:—*Great obliquity*, with which intervention of some tissue is likely, occurred frequently; *comminution* and *double fracture* comparatively rarely. *Compound fractures* yielded proportionately twice as many pseudarthroses as simple: *primary resection* of fragments or removal of splinters, especially in the aged, predisposed to non-union, which was particularly frequent after removal of part of one of two parallel bones. *Extensive necrosis* of the fragments sometimes prevented union, as also, in the great majority of cases, did *abscess of bone*, *hydatids*, and *malignant new growths*. *Marked dislocation*, separating the broken surfaces, was a frequent cause. *Intervention of muscle*, *tendon*, *capsule*, *splinter*, even *nerve* between the fragments, is regarded by Bruns as frequent and as an absolute hindrance to union. Gurlt thought that tissues so caught would atrophy rapidly; but Bruns says that in many cases a bundle of more or less wasted tissue has been found after one to ten years. The large number of cases occurring among sailors on the high seas, suggests strongly that *imperfect treatment* allowing *excessive mobility* is a great cause, a view which is supported by the fact that by daily movements kept up for forty days or longer, permanent false joints have been established in animals. *Defective blood supply* due to immediate and absolute immobilisation and compression by plaster may be responsible now and again for non-union; and the local use of *cold* may similarly have exercised a prejudicial effect upon callus formation. Ligature of the main artery of the part for arterial hæmatoma seems to have delayed union a few weeks in some cases—not in the majority. Guéretin, Curling, and others have endeavoured to show that fractures causing *rupture of the nutrient artery* of the shaft were specially liable to pseudarthrosis; but Gurlt's criticism of the view after examination of many of the specimens adduced in favour of it, strongly supports those who regard such a result as *a priori* unlikely. Callender stated that slow union often occurred in cases of obstinate œdema from deep-seated venous thrombosis.

Diagnosis of defective union.—Both delayed union and pseudarthrosis are characterised by mobility continuing at the seat of fracture. Short lapse of time since the injury, only slight mobility, pain induced by movements, the presence of a well-marked cause of delayed union, and a history of crepitus having been obtained just before or during reduction, are in favour of delayed union rather than pseudarthrosis; but time must often be allowed to decide between them. The loss of this time is regrettable when pseudarthrosis ultimately becomes clear; for progressive anæmia and atrophy of the limb are unfavourable to any operative treatment. These conditions are most marked in the young, in whom impaired growth is added to atrophy, and the bones may remain of almost infantile size: rarely is an operation successful in such cases, and statistics show that in all cases the chance of success diminishes with the duration of the pseudarthrosis.

As a rule, an ununited fracture of a long bone impairs the usefulness of the part greatly—especially in the weight-bearing lower limb. The impairment is least with a small ununited fragment with a transverse fracture, short fibrous union, and steadying masses of bony callus thrown out, or with only one of two parallel bones affected. Cases occur now and again in which function is marvellously preserved: those of great hypertrophy of the fibula to compensate for loss of strength in the tibia are well known.

Treatment of defective union.—Any fault in the general health should be removed, if possible: even though no fault be found, it is right to place the patient before and during treatment under conditions most conducive to health. In **delayed union,** proper treatment of the fracture must be continued. The broken ends must be brought into contact by extension if they overlap. Absolute fixation is probably never indicated; but splints, if used, should be combined with regular massage, and such movements of muscles as can be practised by the patient without causing displacement; with percussion of the fragments, covered with felt, with a mallet—either lightly each day or more severely at a week's interval (Thomas); with free counter-irritation with linimentum iodi; with manipulation of the seat of fracture by the surgeon sufficiently forcible to excite decided tenderness or even to rupture any existing union. With the lower limb it can generally be arranged that the part shall be used whilst displacement is prevented by splints and excessive weight is taken off by crutches, frequent friction, slight irritation and affluxus being the result. Should these means not induce bony union after patient trial, the case must be regarded as one of pseudarthrosis.

In **close fibrous union,** even of some duration, bloodless means, such as the above, should first be tried, for they have not uncommonly induced ossification. But there is little hope of their success in **loose fibrous union, non-union,** and **false joint.**

Here the choice lies between resection of the pseudarthrosis and the application of some *apparatus* to stiffen and support the damaged part. Remarkably good results may be obtained with the aid of an instrument maker. Thus assisted I have seen a man with fibrous union of a fracture about the middle of the femur doing duty as a carter on a farm; and in the Transactions of the Literary Society of Bombay for 1820 (vol. ii. p. 167) is recorded the case of an Arab with an ununited fracture of the right humerus who, provided with a silver tube which was clasped round the upper arm, pursued the calling of a pirate, boarded ships, and used a sword with much success.

As, however, antiseptics have robbed the operation of *resection* of most of its dangers, it is certainly desirable to endeavour to obtain for the patient a sound limb. The operation is never easy, and may be most difficult. An incision is made of such length as freely to expose the fracture, the position and direction of which have been carefully determined by examination under the anæsthetic. The wound is so placed as to reach the fragments with least injury to soft parts, and it is a great advantage when a skin-flap can be raised—with its base at right angles to or parallel to the long axis of the limb in different positions—and laid down at the end of the operation as a complete cover to the deep wound. By carrying the distal portion of the limb in a suitable direction, one of the fragments is caused to project in or from the wound, any uniting fibrous tissue being divided or cut away, and any mass of intervening muscle, etc., being suitably dealt with.

It must now be decided how the ends shall be freshened—what form will enable them to be most accurately fitted together. It is desirable to bring into contact considerable surfaces of raw, bleeding bone—from which all trace of fibrous tissue has been removed—and to raise and preserve all periosteum from such portions of the ends as may be cut away. The ends may be cut at right angles to the shaft; one may be given a wedge-form and the other be grooved to match; both may be freshened and left oblique; but if there be any tendency to shortening, it is wise to cut them in "step-form," *i.e.* to cut into each fragment at right angles to the axis for a sufficient depth, where the broken surface joins the normal shaft, and to remove the broken surface by another cut starting on the end of the fragment and joining this. The end of each fragment must be cut square so as to fit into the "step" thus formed upon the other, and the fit must be accurate when all deformity is corrected.

Fresh, wide, well-fitting surfaces of bone having been prepared, the ends are drilled at suitable spots with a long bradawl of suitable size, or with a dental engine, which enables the work to be done more easily and with less disturbance. Stout silver wire is then passed through the holes, and Thomas' hollow needle, or a cannula large enough to take the end of the wire, is often useful in drawing it through from the deep surface. Two stitches are generally enough; they are drawn tight when both are passed, twisted, cut short, and hammered down. Some prefer pegs of ivory or hard bone driven through both fragments, silvered or nickeled screws, or an ivory rod fixed in the medullary canal of each fragment. The limb being now most carefully held, the periosteum is sutured round the junction, and the wound closed or drained for a short time. The further treatment is that of a compound fracture.

Occasionally circumstances will suggest a special treatment of the bones: thus, an atrophied distal fragment has been thrust into the medullary canal of the proximal fragment; in one case, where a gap of 8½ cm. existed in the tibia, Hahn divided the fibula and thrust the lower fragment into the medullary canal of the upper tibial fragment, obtaining in nine months solid union and a useful limb.

But a gap in one of two parallel bones has generally been obviated, either by cutting out a section of the other and shortening the limb, or by grafting bone into the gap. Thus, Nussbaum split off from the larger fragment, previously irritated by nails driven into it, a piece covered with periosteum and hinged towards the gap, long enough when twisted down to more than fill the interval. He succeeded in two ulnar defects. Macewen's case of building up 11 cm. of the shaft of a humerus, lost by total necrosis, with fragments of bone and periosteum cut out of rickety limbs is well-known. Volkmann cured a pseudarthrosis in a child's thigh by thrusting into the medullary canal of each fragment a splinter of bone which he had just chipped from the sheath of new bone in a case of sequestrotomy; a bit of dead bone or ivory might have done as well.

Even after this treatment the number of cures amounts only to 50 or 60 per cent., and there is reason to fear that the cures were slightly more frequent in septic than in aseptic cases; but the deaths and amputations in the septic cases have been by far the more numerous. Most of the deaths have occurred in thigh cases. It is probable that much better results would be shown by some surgeons than by others, for the skill and determination necessary to treat well a pseudarthrosis of the femur are of no mean order; it can be foretold of many cases as they leave the table, that they are not at all likely to unite.

CHANGES AFTER UNION AND VICIOUS UNION.

Softening and absorption of callus may occur under the influence of depressing diseases during the healing of fractures (page 792). Rarely, under similar influences, softening and more or less complete absorption have occurred weeks, months, or even a year after firm union has taken place. Scurvy appears to have been the commonest cause of this, then the acute exanthemata. Inflammatory processes in the limb, especially severe erysipelas, have also induced softening (probably inflammatory) of recent callus. These have almost always occurred in compound fractures with sequestra, bullets, or other foreign bodies retained. Very rarely an inflammatory process has arisen about a united simple fracture causing softening of the uniting material: in Thiersch's case, four small sequestra were found between the granulating ends of the tibia and were regarded as splinters which had died and caused irritation.

Repeated fracture.—Violence will, of course, refracture a bone; but when soundly united, the second fracture generally occurs at a spot other than that at which the first took place. But cases are not very rare in which, from slight strain, bending or refracture has resulted at the seat of a previous fracture. Often the patient has got about too soon, has been too rash, or has had another fall—especially likely when learning to use crutches; or he has occasionally made a strong effort to save himself from falling, and muscular action has been responsible. The accident is the more likely, the more recent the callus and the less perfect the contact between the fragments — displacement necessarily leading to weak union. The great majority of the cases have occurred in the lower limb, and generally soon after removal of the splints; but a few have been recorded as late as one to three years. In a few cases also a bone has yielded at the same spot several (6 or 7) times in the course of a couple of years.

Callus is not always equally strong after the period given as the average for union. An over-average time should be given in patients in feeble health, also in severe injuries—comminuted fractures, compound fractures, even though the course has been aseptic, fractures complicated by difficulties in vascular supply and those in which vicious union occurs. Careful examination and testing of the bone should be made before permitting use of the limb; caution should be enjoined in beginning its use, crutches should always have rubber shoes, and assistance should be given till their use is learnt. Refractures seem to unite well—those occurring early in less than the usual time; the frequently recurring unite firmly after a prolonged rest.

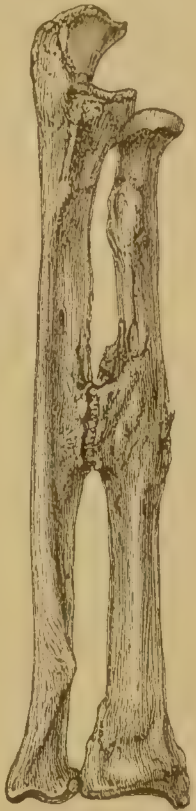


Fig. 230.—Radius repaired after Fracture about its Middle; callus has formed in large amount, has bridged across the interosseous space, and has formed a false joint with the shaft of the ulna. (University Coll. Museum, No. 210 A.)

Exuberant callus.—The free formation of callus about a comminuted fracture, or one uniting with much displacement, is not excessive; it is necessary to bind the fragments together (Fig. 218). But sometimes callus forms out of proportion to the needs of the case or to the obvious irritation, forming irregular, sharp or rounded masses, extending more or less widely into the surrounding tissues, and perhaps causing symptoms by pressing on nerves, etc. It does not, like the ultimately unnecessary portions of healthy callus, become absorbed. This *callus luxurians*, like spurious cheloid in superficial scars, may occur in any bone, but is most common about the joint-ends of long bones, and especially the upper end of the femur. The bony masses thrown out interfere with the movements of the joints, and may absolutely fix them by extending from bone to bone—*e.g.* the masses on the femur irritating the hip-bone till new bone is formed upon its surface with which they blend. No true ankylosis occurs. Again, exuberant callus occasionally joins together two parallel bones (Fig. 315), even when only one is fractured. This is of importance only in the fore-arm, where it may destroy pronation and supination; but some mobility may be preserved by the formation of a false joint between the callus and the second bone (Fig. 230).

Treatment.—Removal of the mass causing trouble, or excision of the joint in suitable cases.

Tumours of callus.—New growths occurring at seats of fracture, either recent or old, are very rare. To get a very moderate number together, statisticians have been forced to include cases of tumour appearing years after the injury. Two points of interest have prompted the inquiry—(1) a fracture induces the formation of a mass of embryonic tissue (callus) in place of the relatively quiescent cells of the bone and periosteum: is it not likely that this mass will sometimes grow uncontrollably and develop into a tumour? Such a question can arise only in connection with recent callus. (2) Irritation is often given as a cause of tumour: may not the irritation of a fracture lead to the formation of a new growth? From this point of view, old callus might possibly be regarded as scar tissue; but the chief interest lies again in cases of tumour developing in close time-relation with a fracture. But in no case does the close relationship of time and place between the tumour and the fracture *prove* anything; the cases are so rare that the occurrence of an ordinary fracture at or close to a point of bone in which a tumour in its earliest stage was present may equally well be assumed. In the later cases there must always be room for doubt as to the exact point of origin of a growth—from callus or adjacent bone—and as to the existence of any causal connection between the old injury and the tumour.

Varieties.—B. Pollard reported the case of a girl of five whose leg was amputated by Marcus Beck for supposed sarcoma of the tibia. At one year she had fallen, and was unable to use the leg for two weeks (fracture?); at two-and-a-half years the leg began to swell and curve. On section a large mass, having the structure of callus, occupied the central portion of the tibia, and a smaller similar tumour, quite distinct, lay on the fibula. Pollard regarded the case as probably one of hypertrophy of callus, but several points are doubtful. A few *enchondromata* developing six months to several years after a fracture, apparently at its seat, are on record; some reached an enormous size. Some which became generalised should be included under sarcomata. A good many cases of *sarcoma*, occurring in more or less close relation to a fracture, have been recorded by Paget, Barwell, Cripps, Folker, and others. Not strictly connected with callus is the *osteoma fracturæ* of Virchow. A piece of bone, generally a growing epiphysis (head of femur or of humerus, tuber ischii), has been broken off, and has grown into a considerable mass, sometimes requiring removal. After fracture of the patella, similar growth of a fragment has several times been noticed.

Vicious union.—Vicious union, or union with marked displacement, may be due either to non-reduction of the primary or to the occurrence of secondary displacement. It may be the result of ignorance or neglect on the part of the surgeon, and heavy damages for malpractice have often been awarded in these cases. But frequently the surgeon is in no way to blame. Excessive, early and long-lasting swelling or thick fleshy covering may prevent the discovery of the real nature of an injury. The fragment separated, say from the head of a radius, may be small and inaccessible, or it may be impossible for the fingers to seize, or for any apparatus to control a fragment, though large, of the pelvis, malar, or sternum. To leave unreduced deformity due to impaction may be the best treatment in certain cases. A bone may be so crushed that it is impossible to restore its form. A long bone may be so comminuted that, extended to its proper length its fragments would not touch. Irregularity of line of fracture may prevent accurate fitting together of fragments. Wounds and sores may render the control of a part extremely difficult; as also may disobedience, delirium tremens, or mania.

In many cases vicious union is of little consequence, in others it greatly impairs the value of a limb—*e.g.* union of a femur with great shortening or marked rotation out of its lower end into the position shown in Fig. 282, persistent displacement of the foot (Fig. 231), angular union or synostosis of the fore-arm bones.

Treatment.—The only radical treatment of vicious union is that by refracture and resetting. When seen early, and especially in the young, the hands often suffice to straighten a bent bone. Later, it becomes a question whether an attempt to produce a subcutaneous fracture with an osteoclast shall be made, or whether the bone shall be divided through an open wound with mallet and chisel or saw. The osteoclast (of which there are many forms) seems to have been used of late more frequently than formerly, the fear that it would cause sloughing of soft parts and break the bone at some improper point having only slight foundation. At least, in tolerably recent cases (six to twelve months) of angular and rotatory deformity it may be used. But for complicated deformities—displacements of the foot at the ankle (Figs. 231, 232), synostosis or angular union of the fore-arm bones, ill-united fragments of joint ends, and so forth—the knife, chisel, and mallet must be used. It is impossible to describe such operations, they must be planned for each case. They are always difficult, and depend upon asepsis for their success, if not for their justifiability. The bones should be fixed in their new

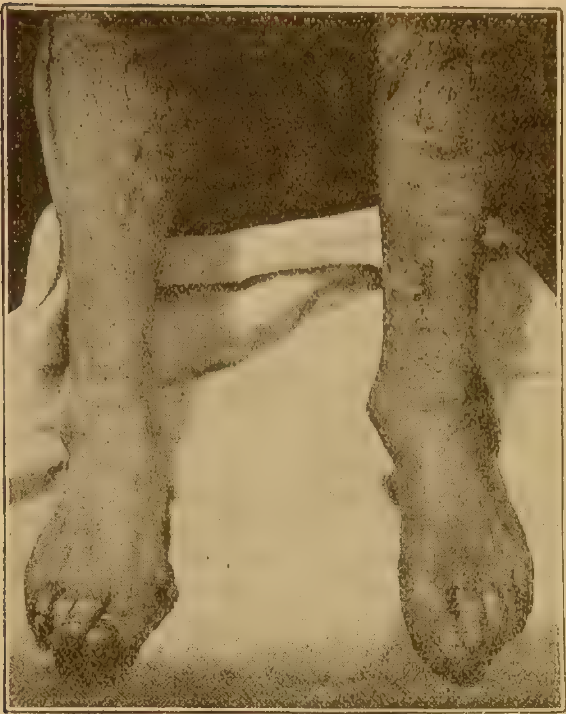


Fig. 231.—The Feet of a Man who had, sometime previously, sustained a Pott's Fracture with Displacement outwards of the Foot on the left side (Watson Cheyne.)

position by sutures, pegs, or screws, and suitable apparatus.

FRACTURES OF INDIVIDUAL BONES.

Fractures of the clavicle. — Fractures of the clavicle constitute fifteen to sixteen per cent. of all fractures, those of the radius only being more frequent. More than one-third of the cases occur in children under five years—many of the fractures being incomplete; among adults, the injury is much commoner among men than women.

Causes.—Usually indirect violence—falls upon the outstretched hand or on the shoulder; direct violence, especially being driven over, in a small number of cases, including most compound or complicated cases; muscular action in a few instances. Intra-uterine fractures from external violence, and others from manipulations during birth have occurred.

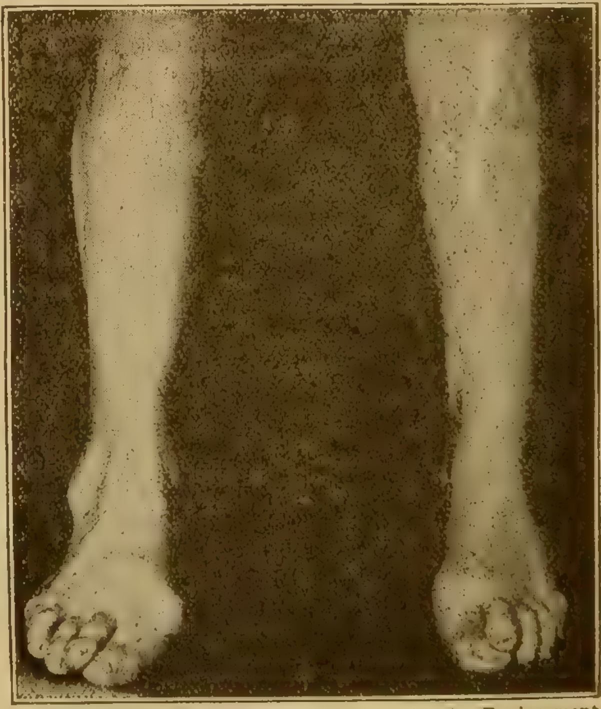


Fig. 232.—Shows the Result of an Operation for Replacement of the left Foot. (Watson Cheyne.

The clavicle is a spanner between the sternum and scapula; its outer freely movable end, from which the arm is hung, is supported by muscles, whilst the inner is firmly bound to the sternum; these points explain the deformity in fractures. Usually the bone breaks at its weakest point outside the middle at the meeting of the two curves, but it may give way in the outer or inner third.

Symptoms. *Middle third.*—When the bone is completely broken in this region, the line of fracture is generally oblique, from without, backwards, and inwards (Fig. 233). The patient generally inclines the head towards the injury, and supports the elbow with the hand of the opposite side. The injured shoulder is lower than the sound, is nearer the mid-line, and is rotated so that its outer surface looks more or less forwards. Often, especially when the fracture is at or internal to the middle, the skin is stretched over a bony prominence—the outer end of the inner fragment. Unless the amount of immediate swelling is unusual, some deformity is readily felt, and the usual signs of fracture are present. Measurement shows the injured clavicle to be shorter than its fellow—even by 1 to $1\frac{1}{2}$ inch; and, though it may not be felt, it will be clear in most cases that the outer fragment is lying with its inner end pointing inwards, backwards, and upwards below the prominent outer end of the inner fragment, perhaps pushing it upwards and forwards; otherwise its prominence must be due to tension of the skin over it from dropping of the arm, for though contractions of the sterno-mastoid will raise it temporarily, it is doubtful if the muscle will keep it raised.

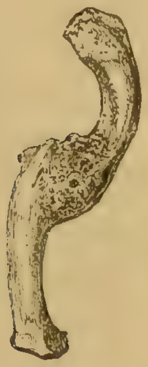


Fig. 233.

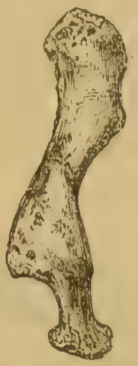


Fig. 234.

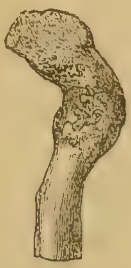


Fig. 236.

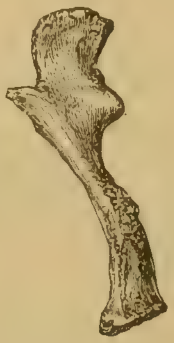


Fig. 235.

Fig. 233.—A right Clavicle repaired after Fracture with some Comminution in the Middle Third; all the fragments are set in a large oval mass of callus. The deformity due to falling back of the outer fragment is unusual.

Fig. 234.—A right Clavicle repaired after Fracture outside the rhomboid Ligament: the outer fragment remains displaced forwards, inwards, and somewhat downwards.

Fig. 235.—A left Clavicle repaired after Fracture just internal to the Ligaments; the outer fragment is displaced downwards, forwards, and inwards; rotation was probably corrected by treatment.

Fig. 236.—A right Clavicle repaired after Fracture between the Ligaments; the outer fragment is still bent sharply forwards. (University College Museum, Nos. 180, 181, 182, 183.)

If the fracture be transverse and the fragments locked, or incomplete, the bone presents an angle backwards, and perhaps upwards, with a corresponding hollow on the anterior surface, which can be readily increased, but removal of the angular deformity requires some force. In greenstick fractures crepitus may be slight or absent, and so may be the deformity. Such fractures are limited to posterior fissures (Fig. 237), after the infliction of which the bone springs back to or towards its normal form; but localised "fracture-pain" and tenderness are present with more or less impairment of movement, and in a few days a callus tumour appears. To detect such cases digital examination of the whole length of the bone must be very careful.

In the rare double, multiple, and comminuted fractures of the clavicle, deformity is great, and the intermediate fragments are much displaced.

Outer third.—Fractures here are often transverse; they are divided according as they lie between (Fig. 236) the coraco-clavicular ligaments (inter-ligamentous) or external to them (extra-ligamentous). In all, the coraco-clavicular ligaments continue to sling the coracoid process and arm from the inner fragment, so that the outer fragment is not so much depressed by the weight of the arm as it may be in the previous group. The deformity depends largely upon the intensity of the original violence. In the fairly inter-ligamentous cases, from slight or moderate violence, the fragments remain closely connected by the ligaments, trapezius, and deltoid, and often become interlocked, so that little deformity may result. Usually the shoulder rolls forwards and drops a little, so that the outer fragment slopes outwards, downwards, and abnormally forwards from the line of fracture. In extra-ligamentous cases the fragments are more loosely connected; the scapula swings forwards, turning on the coraco-clavicular ligaments, until the short outer fragment may lie at right angles to the inner; the scapula also falls inwards, lowering the thin outer fragment till it may become disengaged from the inner. Then a striking resemblance to a dislocation of the clavicle on to the acromion is produced, the inner fragment standing out prominently half an inch or more above the outer fragment and acromion. Error is prevented by noting the form and roughness of the prominence, by the discovery of the outer fragment, and by the crepitus accompanying reduction.

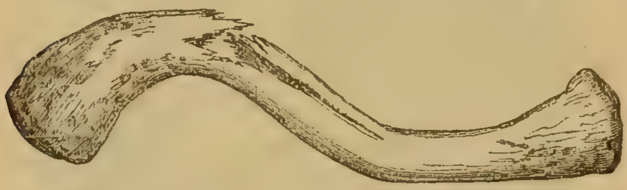


Fig. 237.—Greenstick Fracture of the Clavicle from a Child aged 5 years. The fracture is situate at the junction of the outer and middle thirds; the posterior lamellæ are broken transversely, the anterior merely bent. A long fissure runs inwards from the fracture, a short one outwards. (St. George's Hospital, Series I. 76.) (Pick.)

Inner third.—Fractures in this region are quite unusual, but several of those from muscular violence have been situate here. The line of fracture is very variable; perhaps the commonest is that shown in Fig. 234, with the outer fragment displaced in front of and below the inner, which may be pushed up by it and pulled up by the sterno-mastoid ; the pectoral muscle tending to draw the outer fragment towards the humerus.

Separation of the sternal epiphysis.—Few clear cases are recorded of separation of this thin plate of cartilage, which ossifies at 17 to 18 and joins at 25. When the injury occurs the inner end of the clavicle appears too prominent, as if dislocated forwards. The main points in the diagnosis are : the age of the patient, the position of the lesion, the sharpness and irregularity of the inner end of the shaft, and the presence of the thin epiphysial plate still bounding the pre-sternal fossa. In two out of four cases muscular action was the cause assigned.

Complications of fractures of the clavicle.—Fractures of the clavicle are very rarely compound or complicated, even from direct violence other than gunshot; in which case, it is the bullet rather than the fracture that does the mischief.

C. Heath recorded a case of "aneurysm" of the *subclavian artery* appearing within a month after fracture of the clavicle; the post-mortem seemed to point to a spiculum of callus on the clavicle as the cause of the injury to the artery. Dupuytren, in a lecture, said he had seen two or three cases of "aneurysm" following these fractures; but it is impossible clinically to eliminate simultaneous fracture of the first rib.

The *great veins* have in a few cases been injured, and a hæmatoma of great size has rapidly developed. In one instance the extravasation extended from the cheek to the fingers, with slight pulsation and systolic bruit in the supraclavicular region; the limb became paralysed, and the radial pulse was lost, but reappeared on the second day, when increasing tension of the skin caused Manoury to incise above the clavicle, compress the vein, and turn out the clots; the patient died of entry of air into the subclavian vein, which was torn almost across, the plexus being merely infiltrated. In Sir Robert Peel's case the swelling was similar, but less extensive. Peel died of concomitant injuries, but recovery has resulted in cases treated with evaporating lotion or gentle elastic pressure calculated to limit the extravasation.

If forced to operate, the first incisions should give the freest access—the gap between the bones being utilised; and circulation in the limb below the axilla should be arrested.

Erichsen amputated at the shoulder for gangrene, apparently due to compression of the subclavian vein by a fragment of clavicle; and Annandale cut down and removed a depressed fragment which he thought was acting similarly. In the St. George's Hospital Museum is a specimen showing the internal jugular vein wounded by a splinter; the patient had both clavicles and sternum broken by a falling bough, and died immediately.

There are several cases of injury to the *brachial plexus* recorded : in some J. Hutchinson, senr., has noted hyperæmia and sometimes sweating of the same side of the head and neck, with a rise of 2° to 3° F.; injection of the conjunctiva and lachrymation, flattening of the cornea from diminished intraocular tension, contracted pupil and ptosis—phenomena obviously associated with injury of the *sympathetic nerve* in the neck. Slight exertion sometimes brought out the above symptoms, or the contracted pupil and narrowed palpebral fissure might alone be present.

Emphysema from wound of the lung has sometimes occurred with fractured clavicle, and become very extensive.

Treatment of fractures of the clavicle.—The bandages and splints for fractured clavicle are very numerous. The most generally useful is *Sayre's strapping.* Two strips of plaster, as wide as the patient's hand and long enough to go once and a half round the body, are applied as shown in Figs. 238 and 239. The first strap is pinned round the upper arm as high as possible, non-adhesive surface towards the skin, which is guarded by a strip of boric lint. The surgeon draws both shoulders fully back, and an assistant carries the heated or turpentined strap round the chest. This strap fixes the shoulder and elbow well back in the position given by the hand of the surgeon. The second strap starts below the sound collar bone, crosses the junction of neck and shoulder (the skin being guarded by boric lint or by a bit of moulded guttapercha) and the back obliquely to the point of the elbow, is then carried upwards along the extensor surface of the fore-arm, rather towards its ulnar side, to the neck, which it crosses, and ends over the scapula. This strap draws the elbow forwards (thus throwing the shoulder and outer fragment still farther back—the loop acting as fulcrum) and pushes the shoulder up till the outer fragment is on a level with the inner. With an adult the hand may often be left free, supported by a loop of strapping round the wrist, the long end of the loop being carried over the inner fragment (which it tends to depress) to the scapula. No hole should be cut for the olecranon; no strap should pass outside the arm on the injured side; the second strap should cross the neck exactly where it tends to remain naturally. The axilla and fold of the elbow should be well dusted with boric powder, or greased with boric ointment; a piece of boric lint should be placed between the palm and the chest.

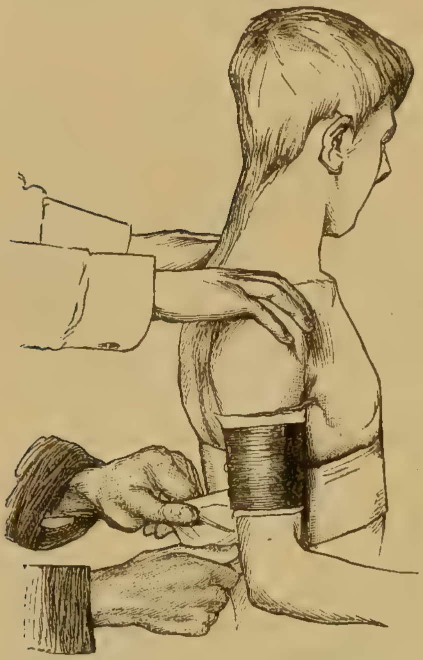


Fig. 238.—Sayre's Strapping. Application of first strap while the shoulders are well drawn back.

The above directions should not be carried out in a routine manner, but so as to correct whatever deformity is present. With good strapping there is little tendency to slip or stretch; the bandage is fairly enduring, and even a child has difficulty in wriggling out of it. The deformity of a greenstick fracture often disappears under its use. It fails, however, in complete fractures to draw the outer fragment sufficiently from the mid-line, and shortening of the bone remains. Nothing but an *axillary pad* will obviate this. It is wedge-shaped, 6 in. long, 5 in. wide, and 3 in. thick at the base, which is rounded and turned towards the axilla, where it is fixed by a padded strap over the opposite shoulder; the cushion is best stuffed with horsehair; or, less firmly, with bran. The elbow is brought to the side by a turn of strapping or bandage, and raised by a sling taking the whole length of the fore-arm. Even a hair cushion may cause unbearable pressure on nerves; or œdema or even gangrene from pressure on vessels. If a pad be used, therefore, it must be well watched.

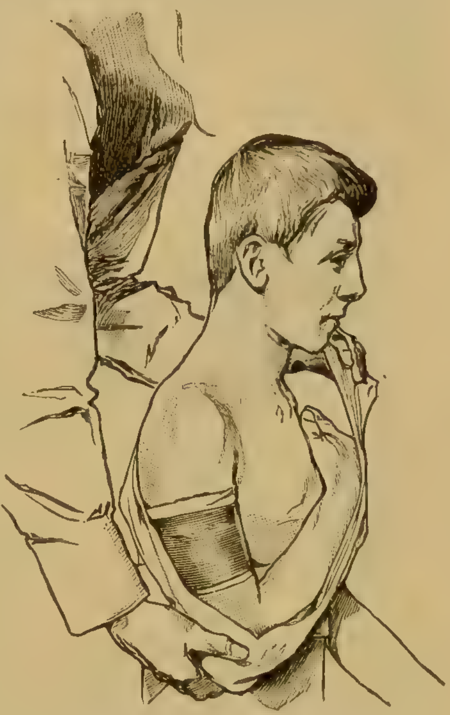


Fig. 239.—Sayre's Strapping. Application of second strap so as to raise and carry forwards the elbow, and fix the hand.

In slight cases without deformity, a *sling* supporting the elbow is sufficient. Where it is important to reduce deformity to a minimum, in fracture of both clavicles, in badly comminuted fractures, and in some complicated cases, the patients should be *kept in bed*, lying flat on the back with only a neck pillow. The arm can be well fixed by a loop of strapping below the axilla, passing round the back to the front, and by another above the elbow going in the opposite direction; in double fractures these straps should pass from arm to arm. Championnière employs massage: he thinks, with advantage.

Non-union is very rare, and, as a rule, impairs function but little. The coraco-clavicular ligaments have ossified after injury, and masses of callus have connected the clavicle with the first rib—impairing movement more or less.

Fractures of the scapula.—These contribute about 0·8 per cent. to the total list of fractures. They are commonly due to direct violence, and are far more frequent in men than in women, in adults than in children. Fractures may affect—(1) the blade and spine; (2) the acromion; (3) the coracoid; (4) the surgical neck; and (5) the glenoid fossa.

(1) The **blade** may be smashed by fissures running through it in all directions (Fig. 240); but limited injuries are more usual, especially a fissure running horizontally below the spine (Fig. 244). A similar fissure may cross the supra-spinous fossa, and sometimes a vertical one (Fig. 241) near the vertebral edge has been met with; farther out the dense spine is an obstacle to such fissures. A few cases, but no specimens, in which the spine was thought to have been broken from the blade have been recorded.

These fractures are detected and localised—by evidence of injury to the part; by crepitus on moving the arm and scapula, actively or passively, whilst a hand is kept flat over the scapula; and by discovering mobility between points which can be seized, like the inferior or superior angle or the spine. As a rule the fragments are little displaced, but in fractures below the spine the lower fragment is usually drawn forwards and upwards by the teres and latissimus dorsi muscles (Fig. 242). Union with little callus occurs. A sling for the arm is often sufficient treatment; a piece of strapping with three tails, carried one over the shoulder to the front, the other two round the chest, is sometimes used to press and fix the scapula against the thorax. Massage should be practised, and the arm set free as soon as doing so causes neither displacement nor pain.

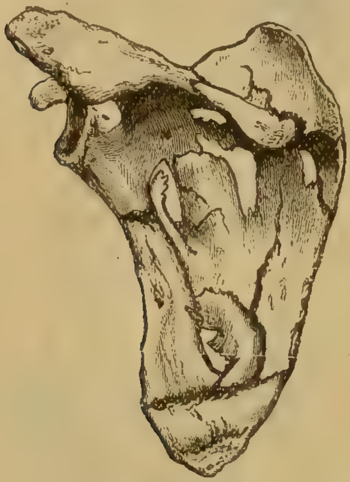


Fig. 240.—A Smash of the Scapula detaching and displacing a Portion of the Spine. (St. George's Museum, Series I. 83.) (Pick.)

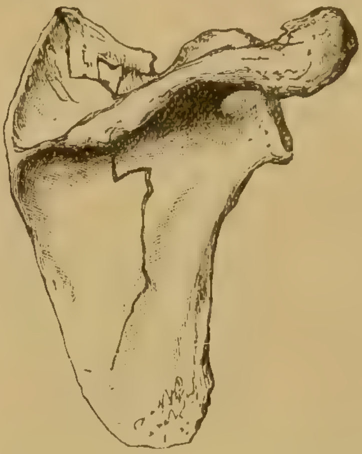


Fig. 241.—A vertical Fissure, with comminution in the supra-spinous Fossa. (Ibid. 84.) (Pick.)

(2) The **acromion** for surgical purposes extends to the external border of the spine; the acromial epiphysis is very variable, but is always smaller than the segment of bone just indicated. It ossifies from two or three centres at sixteen, and joins at twenty-five. Generally broken by direct violence (blows and falls upon the shoulder), it may yield before the humerus driven against it; muscular action has proved a very rare cause. Small fragments may be broken from the tip and outer edge with little deformity; less often the cleft lies

more or less mesial to the acromio-clavicular joint (Fig. 243), and it would lie here should the epiphysis separate.

The *signs* of the injury are—flattening of the shoulder; difficulty in abduction; irregularity on the subcutaneous surface; removal of the deformity on pushing up the humerus, mobility of the fragment, and perhaps crepitus.

The usual *treatment* is a figure-of-8 bandage under the elbow, crossing on the shoulder, and under the axilla on the opposite side, applied so as to raise the humerus. Better than this is Sayre's strapping (Figs. 238, 239), no backward traction being made, and the "first" strap being applied after the "second," and over all. Union is usually fibrous, but the result is good. It would be easy to wire the fragments, but, unless the arm were kept abducted for a month, the result would probably be no better. Several specimens without histories have been shown of separation on both sides of a part of this process, with no irregularity or sign of callus, and a plate of "what looked like cartilage" intervening. These specimens may have been due to injury, but were probably examples of non-union of the epiphysis. No certain cases of separation of the epiphysis are known.

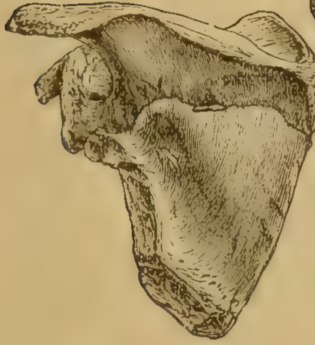


Fig. 242.

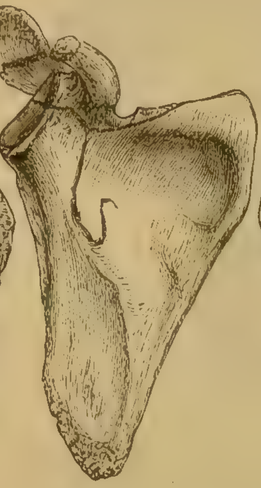


Fig. 243.

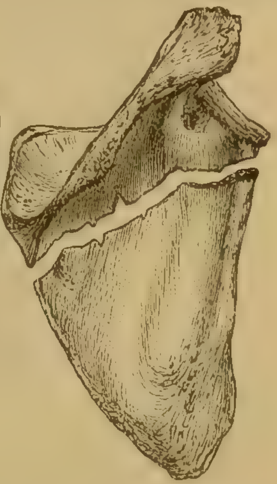


Fig. 244.

Fig. 242.—A Scapula repaired after such a Fracture as is shown in Fig. 244. The lower fragment has been drawn forwards by the teres muscles, and lies rather behind the upper. Little callus has formed, and there is a small gap in the line of union at one or two spots.

Fig. 243.—A Scapula showing: (1) A fracture of the acromion near the spine; (2) a fissure separating the whole coracoid process with the upper end of the glenoid fossa and a bit of the upper edge of the blade; (3) a fissure running from the above into the blade.

Fig. 244.—A Scapula recently fractured transversely below the Spine.

(3) The **coracoid process** is so well protected that it is very rarely broken. The tip has been torn off by muscular action—*e.g.* in wringing—but otherwise direct violence has been the cause, both of this fracture (Fig. 245) and of that through the base of the process into the glenoid fossa (Fig. 243). Separation of the process by direct violence has occurred before its junction with the blade at puberty. The diagnosis is difficult. The most important points are

the detection of mobility or crepitus on pressing on the tip of the coracoid, and the discovery in this neighbourhood of a displaced fragment. Usually the dense fibrous tissue round about prevents the muscles attached to the tip from dragging the fragment far. Action of these muscles should be specially painful. Serious complications, in the way of injuries to neighbouring bones and soft parts, must be expected. A sling till pain subsides is the best treatment. Union is fibrous in fractures of the tip (Fig. 245).

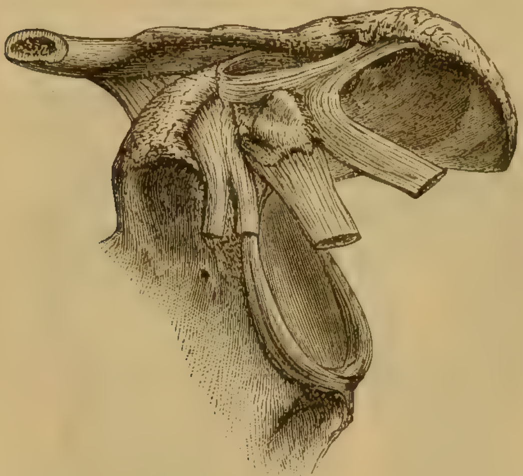


Fig. 245.—Fracture of the Tip of the left Coracoid Process; union by a fibrous band ½ in. long. Part of the coraco-acromial ligament, the coraco-brachialis and biceps, and the pectoralis minor, are attached to the fragment. No history. (University College Museum, No. 186 A.)

(4) The **surgical neck** is the seat of a very rare fracture from direct violence. The fragment consists of the glenoid fossa and coracoid process with the pectoralis minor, coraco-brachialis, both heads of the biceps, and more or less of the long head of the triceps attached to it. It will drop down and in—under the influence of the weight of the limb, and of the muscles of the axillary folds—as far as the coraco-clavicular ligaments and soft parts around the fracture will allow. The *signs* are said to be—flattening of the shoulder, tension of the deltoid, prominence of the acromion, some lengthening of the arm, change in position of the tip of the coracoid process, mobility of the process, and crepitus when it and the head of the humerus are grasped and moved, whilst the scapula is fixed by the other hand; presence of a mass high in the axilla, in which the fragment may sometimes be distinguished from the head of the humerus; the elbow lies against the side. Upward pressure on the elbow remedies the deformity easily, but it recurs immediately the support is removed, points which, with crepitus, distinguish the accident from a dislocation. Perfect movement of the head with the shaft eliminates fracture of the neck of the humerus. If, as in one of Sir A. Cooper's cases, a fracture of the clavicle outside the ligaments is also present, the lengthening of the arm (measured from the acromial angle), due to falling of the inner fragment of the collar bone under the weight of the limb suspended from it, is considerable; the acromial fragment in Cooper's case over-rode the internal. It is not quite clear why lengthening should occur from fracture of the neck of the scapula; possibly the statement is made on the authority of Cooper's admirably described, but complicated case.

There does not appear to be any specimen of fracture of the *anatomical neck*—immediately mesial to the glenoid fossa.

Treatment.—Sayre's strapping applied as for fractured clavicle would probably meet all requirements—raising the fragment and drawing it out and back. Cooper recommended a figure-of-8 bandage round the shoulders, crossing behind, together with an axillary pad, a bandage fixing the elbow to the side, and a sling supporting the whole fore-arm. He says that a good result should be obtained in seven weeks. Fibrous union has occurred, once with a useful, once with a useless limb.

(5) Fractures of **the glenoid fossa** are also rare. They include such injuries other than fracture at the base of the coracoid, as the chipping off of smaller or larger portions of the glenoid surface and head of the scapula. The majority occur in connection with dislocations, and are unrecognised unless unprovoked recurrent displacement suggests such a complication; for the fragment is small, deeply situate, and unlikely to yield crepitus. But the glenoid fossa may be "starred," or considerable pieces of the head of the scapula—*e.g.* the lower half, as in Spence's case—may be broken off by direct violence without dislocation of the head. Here the fragment might be felt, and deep crepitus obtained by manipulations in the axilla, and by pushing up the dropped head. The coracoid process is firm. Sayre's strapping will form the treatment; or a sling may suffice.

Fractures of the humerus.—These constitute 7·5 per cent. of all fractures. They diminish in frequency with each decennium. In the first, fractures of the lower extremity predominate over fractures of the shaft, and fractures of the upper end are few; from 10 to 20 years of age the same order holds, but the differences are less marked, injuries of the upper end being much more common. Injuries of the lower end now rapidly fall off, and disappear in the highest periods; but those of the upper end increase relatively to those of the shaft, until, in advanced age, they become the commonest fracture of the bone.

Fractures of the upper end of the humerus.—The typical fractures of the upper end may be classified according as they affect: (1) the head; (2) the tuberosities; (3) the anatomical neck; (4) the epiphysial line; (5) the surgical neck.

(1) **The head.**—Fissures of, and the chipping off of bits from the head are very rare, and probably unrecognisable during life. Massage and a sling form the treatment.

(2) **The tuberosities** are rarely fractured—the lesser very rarely, and always in connection with dislocation. *Greater tuberosity.*—Fracture is generally associated with inward dislocation; it accompanies impacted fractures of the anatomical neck. Direct violence, and, very rarely, muscular violence have detached the process without other injury. The tuberosity is driven or dragged up and back beneath the acromion (Fig. 246), and there is nothing to depress it.

Signs.—The shoulder is swollen and tender, especially on the outer side; it is said that in complete detachment of the process the head of the bone falls and is drawn inwards. Through a thin covering a groove may be felt between the process and its former site, and crepitus will be detected on pressing upon the fragment, while abducting and rotating the shaft; voluntary abduction (deltoid) is possible, but rotation out (infra-spinatus and teres minor) is impossible.

Treatment.—The best result will be given by keeping the arm abducted on a Middledorpf's triangle or a large cushion; or by pinning the process in position. Union, if it occurs, is bony.

(3) **The anatomical neck.**—A fracture separating the head from the tuberosities is rare, and appears to be always due to direct violence, *e.g.* heavy falls upon the shoulder, generally in people beyond mid-life. The separated head may remain loose within the capsule, displaced in any direction with regard to the shaft: it may even be rotated so that its cartilage-covered surface rests against the broken surface of the shaft. The head may be impacted into the upper end of the shaft, splitting it and separating from it one or both tuberosities—though all are generally held together by capsule and tendons. Lastly, the head may be dislocated downwards, or downwards and inwards, through a rent in the capsule into the axilla; here it may be found in front of the axillary edge of the scapula, as low as the third rib, or near the coracoid process, free or, in old cases, fixed by bone to neighbouring bone; perhaps it may be eroded even to disappearance (Fig. 247).

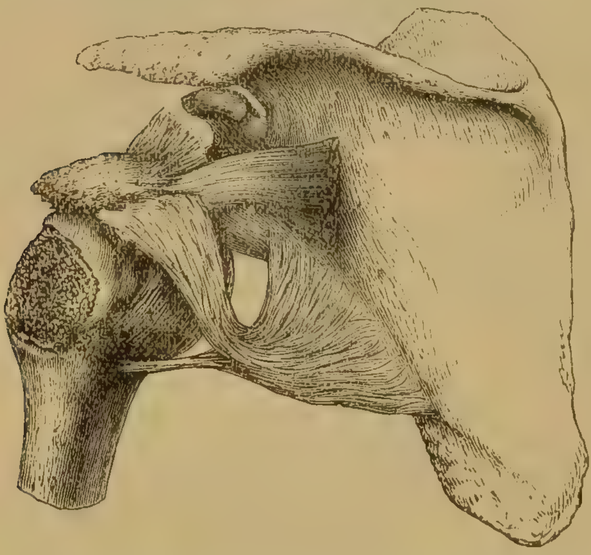


Fig. 246.—Fracture of the great Tuberosity of the Humerus from direct violence; the supra- and infra-spinatus and teres minor are attached to the fragment. The capsule of the shoulder is sound below. (University College Museum, No. 192.)

Signs.—If swelling does not obscure it, there will probably be some flattening, posteriorly and externally, with proportionate prominence of the acromion and tension of the deltoid (these signs are best marked when the head is dislocated); the humerus hangs vertical, and the elbow is close to the side. It is said that there may be slight lengthening from failure of injured muscles to hold up the bone; otherwise, displacement upwards ($\frac{1}{2}$ in. or less) and inwards may be expected from the action of the longitudinal muscles and of the rotators and adductors, aided by the original violence. Movement in all directions is free, but very painful, and is often accompanied by crepitus if the surfaces are in contact and free, or if the tuberosities are split off. The fingers cannot be pressed in beneath the acromion to any considerable extent, nor is there any prominence in the axilla or beneath the pectorals unless the head has escaped from the capsule; then it is felt in its abnormal situation, is movable under the fingers, but not with the shaft, emptiness of the glenoid fossa becomes evident, and shortening may increase to one inch. The brachial plexus may be severely contused in this accident. With firm impaction the deformity at the upper end of the bone and its superficial consequences are to be relied upon; there may be slight crepitus from small splinters.

J. Hutchinson, noted that in cases of slight or no impaction, shortening sometimes increased during the first weeks, owing to muscular action, the head being forced down and in as the shaft rose; such cases, two or three months after the accident, have been taken, he says, for unreduced dislocations, and treated accordingly.

Treatment.—The application of a shoulder cap, with such extension as may be necessary to overcome shortening, meets the requirements of most cases when swelling is subsiding: till then the limb may lie on a pillow with weight-extension applied. A dislocated head may be reduced by digital pressure, with the possibility that it will assume a faulty position, and the probability that it will not unite; or it may be left, very likely to interfere with movement; or it may be removed after a few days, which seems the best course.

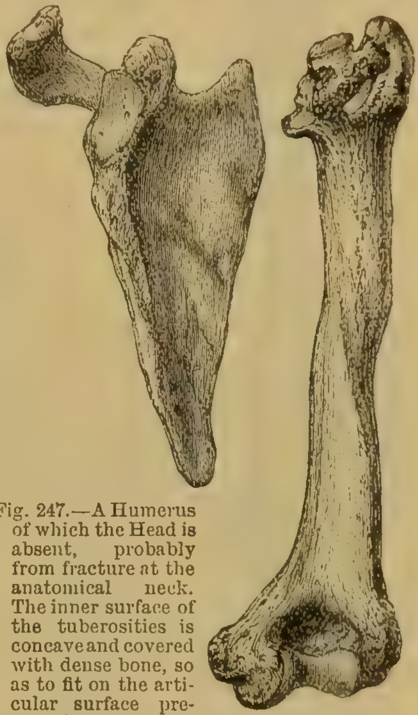


Fig. 247.—A Humerus of which the Head is absent, probably from fracture at the anatomical neck. The inner surface of the tuberosities is concave and covered with dense bone, so as to fit on the articular surface presented by the head of the scapula. This is long and concave from above down, narrow and convex from before back. The bones are strong. (University College Museum, No. 193A and 193B.)

4. **Separation of the upper epiphysis.**—Ossifying from two or three centres which are separated by violence only with extreme rarity, this epiphysis becomes one bone after the sixth year, forms a sort of cap to the pyramidal top of the shaft, and usually unites at twenty to twenty-one. To it the capsule is almost entirely attached, and it receives the insertions of the subscapularis, supra- and infra-spinatus, and teres minor. These connections with the glenoid fossa are almost always stronger than the epiphysial disc, and consequently before 20 separation of this epiphysis replaces dislocation—not 1 per cent. of dislocations of the shoulder occurring

under twenty. Separation of the epiphysis does not necessarily open the shoulder joint.

Causes : Traction on the arm during labour, sudden pulls in and out to save children from falling, falls on the elbow or hand, and direct violence to the shoulder.

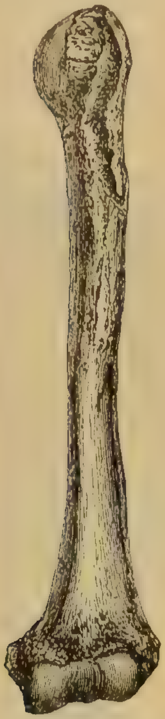


Fig. 248.—A Humerus repaired after a Fracture of its surgical Neck, downwards and backwards from just below the lesser tuberosity to 3 ins. below posteriorly. The angle between the head and shaft is much diminished, as also is the measurement from the centre of the head to the great tuberosity. The shaft is drawn slightly in. The whole bone is atrophied.

Signs.—There may be no displacement—especially in the young—the periosteum being untorn; unless slight mobility or crepitus can be detected, there will be no positive sign of the injury. In a large proportion of the cases the shaft is displaced more or less inwards, and upwards if the inward displacement be complete. Then the roundness of the shoulder is preserved, but the deltoid is tense, and the elbow points out or back; the upper end of the shaft may form a visible swelling below the coracoid process—where the fingers will feel it, not round and smooth like the head, nor so sharp and irregular as in a fracture. The fingers cannot be pressed beneath the acromion, the head being felt in the socket, but they do sink in through the deltoid 1½ in. or so below the acromion opposite the line of fracture. The head does not move with the shaft, and crepitus is obtained only when the broken surfaces are brought into contact. Sometimes the broken surface of the epiphysis looks forwards and outwards, being probably driven into this position, or at least fixed in it by the shaft locked against its inner margin. Sometimes the shaft is displaced upwards in front of or outside the epiphysis—no doubt by the initial violence.

Grave complications seem to have been frequent. Several cases have been compound; the great vessels have been torn or compressed, and gangrene has resulted in at least two cases, the brachial plexus has been injured, and even permanent paralysis has resulted; in a few cases marked arrest of development has been noted, but the arm has been strong. Suppuration has been rare, but bony ankylosis has occurred in three simple cases (J. Hutchinson, junr.). In two early cases a false joint formed.

Treatment.—As swelling is usually rapid, early examination and setting under anæsthesia are necessary if there be any doubt or difficulty; for it is most desirable to effect an accurate diagnosis and replacement with all possible gentleness. Traction on the limb downwards and outwards aided by pressure on the epiphysis is generally successful; and the cup and ball form of the fragments opposes re-displacement. The shaft will thus be brought to an abducted epiphysis, and if they can be interlocked the arm may be carefully brought to the side; if not, the patient must remain in bed and abduction be maintained by weight extension. Ordinarily, a shoulder cap, with or without an axiliary pad and a sling, should be applied. Difficulty in reduction is due to interposition of soft parts, usually the edge of the periosteal collar round the shaft. In compound cases the obstacle must be removed by operation, and in simple cases the question of operation must be carefully considered. Great displacement of an important epiphysis with years of growth before it or signs of any complication, would render operation desirable; even after several weeks, resection of a displaced shaft and reduction have been performed with good results. If necessary, the epiphysis may be fixed to the shaft for a time by a steel pin (Helferich).

(5) The **surgical neck**, between the tuberosities and their muscles—the resultant of whose action is believed to be abduction—and the bicipital groove, to which the great adductors and internal rotators, are attached, is the most frequent seat of fractures of the upper end of the humerus (Fig. 248). It occurs at all ages, both from direct and indirect violence. Swelling is often great. The displacements and *signs* are the same as in separation of the epiphysis, except that crepitus is bony, and the fragments sharp, irregular, and likely to excite more pain when they are moved.

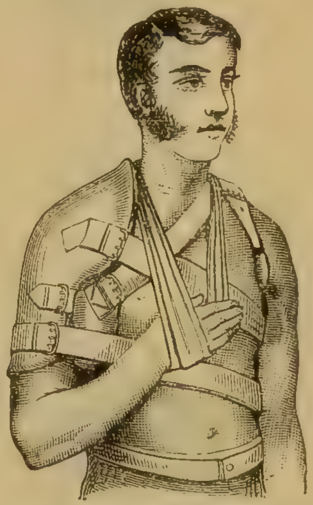


Fig. 249.—Treatment of Fracture of the Surgical Neck of the Humerus by the shoulder cap, axillary pad, and sling for the hand.

Occasionally the lower fragment is impacted into the upper: this is the more likely the higher the fracture and the more cancellous the upper fragment. Generally the outer side of the shaft penetrates the upper fragment, the usual deformity is present in slight degree, and some crepitus may be obtained on firmly grasping the head and rotating the shaft.

Compound and complicated fractures at the surgical neck are very unusual. The commonest complication is dislocation of the head. These cases look like simple dislocations: the shoulder is flattened, the arm shortened, the elbow stands out from the side, and there may be fulness beneath the pectoral; the fingers sink in beneath the acromion, and encounter the rounded head in the axilla; but they show also that it does not move with the shaft, and that crepitus occurs between them.

Treatment.—During the period of early swelling in this and other injuries to the shoulder the patient should be kept in bed with the limb comfortably placed on a firm pillow, ice packed round the joint, and weight-extension maintained from the arm. When oozing has ceased massage should be begun, and fomentation may quicken absorption. Splints may be applied when the swelling is subsiding. When there is firm impaction a sling may be sufficient; but it would probably always be safer to apply a shoulder cap lightly. When there is deformity the shoulder cap (Fig. 249) or Erichsen's splint (Fig. 250) must be used if the patient is to get up; and if, with either, much extension has to be made to prevent shortening, the fingers and the limb below the splint must be bandaged as firmly as it is necessary to bandage the splint to the arm. The hand only is then supported in a sling, so that the weight of the limb to which further weight may be attached, may aid the extension. But, unless hypostasis is feared, the patient is better in bed under weight-extension so long as there is any strong tendency to shortening; for both the above splints are poor ones.

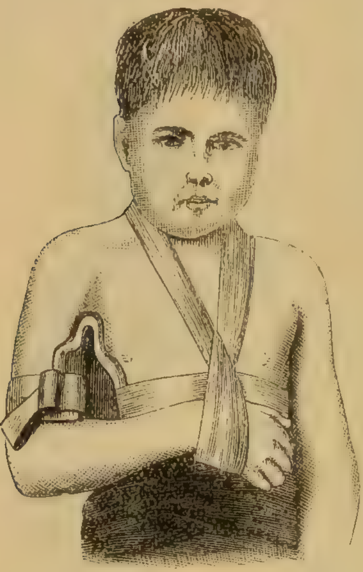


Fig. 250.—Treatment of Fracture of the Surgical Neck of the Humerus by Erichsen's Splint, a piece of leather or poroplastic of the form shown. The rounded bend at the upper end acts as an axillary pad, and some extension may be made. A bandage should fix the elbow to the side.

When the head is dislocated, an attempt should at once be made to reduce it under an anæsthetic. Failing this, the choice lies between (*a*) waiting eight to twelve weeks till union has occurred, and then attempting reduction with the chance of a second failure and a bad result or of refracture; (*b*) leaving the head alone and pushing the shaft towards the glenoid fossa; and (*c*) cutting down on the head and replacing it, or, if this seem unlikely to give a good result, removing it and placing the shaft in the glenoid fossa. In favourable circumstances the latter plan would now be chosen. Since the above was written, Clutton has shown an excellent result in a boy in whom, through an anterior incision, he fixed the head and surgical neck on to the shaft by means of an ivory peg driven vertically through the former into the latter; he then reduced the head, and the boy recovered with a practically perfect joint.

Diagnosis of injuries of the shoulder.—For purposes of diagnosis it is well to bring together several of the foregoing injuries to the shoulder which resemble one another in some respects, and to draw attention to the main points of difference which we should endeavour to establish. Accurate diagnosis may be impossible when the broken-off fragment is small, the coverings thick, or extravasation extensive.

The chief injuries to be considered are—the inward dislocations of the humerus; fractures of the glenoid fossa and surgical neck of the scapula; and fractures of the anatomical and surgical necks of the humerus (including separation of the upper epiphysis) without and with dislocation.

The first point is to ascertain the position of the head; if it be in the socket, the shoulder is rounded, the fingers cannot be pressed beneath the acromion, and they feel the smooth rounded head when they grasp the shoulder from before back. But the head is dislocated if the shoulder be flat, the deltoid tense, and the fingers meet no resistance other than that of muscles in pressing towards the glenoid fossa; if the deformity cannot be removed without some difficulty, but, when removed, does not easily recur. If, while the head has escaped, the end of the shaft has risen up beneath the acromion, the signs last given will be less marked, and the shape and irregularity of the shaft will be helpful.

If the head be out of the socket, and there is no fracture, the shaft may be regarded as a pointer to it; it swings upon the deltoid insertion and the elbow points the more back and out the farther the head goes up and in. So the eye often enables us to lay a hand on the head. If it cannot be felt at once, gentle rotation of the shaft is practised, and the movement thus communicated to the head makes its situation evident. But if, in a case which looks like a dislocation, with some shortening and some projection of the elbow, we get crepitus in performing rotation; if we feel that the rotating end is sharp, irregular, and causes pain; and if, elsewhere in the axilla, we feel the mass of the head and tuberosities, not moving with the shaft, but grating against it occasionally, we have a fracture of the surgical neck and a dislocation of the head. If the arm hang by the side, yet the signs of emptiness of the glenoid fossa are evident, though in a less striking degree, if a small hemispherical mass be felt free in the axilla, while rotation of the shaft is excessive in degree, and the finger detects absence of the head from its place, we have to do with a fracture of the anatomical neck with dislocation of the head.

If the head be in its socket, we grasp it and rotate the shaft; if they move together and without crepitus, either there is no fracture between them, or it is firmly impacted, in which latter case the deformity present, and any irregularity of the bone discovered, must be considered. If there be no abnormal mobility between the head and shaft, and yet crepitus occurs, this must indicate either fracture of a process of the humerus or of a bit of the head, or a fracture of the scapula. Touch will give some idea as to the seat of crepitus. If superficial, the region of the great tuberosity must be examined. With deep crepitus the scapula is probably its seat. In both the fractures in our list, simply raising the elbow removes the displacement with crepitus, and it recurs at once on removal of the support. In fracture of the surgical neck, the coracoid moves with the fragment; in fractures of the glenoid fossa it does not.

Lastly, if there be abnormal mobility between the head and the shaft, there is a fracture of either the anatomical or surgical neck, or a separation of the epiphysis. Under twenty the injury is probably of the latter kind. The signs of it and of fracture of the surgical neck bear some resemblance to those of inward dislocation, but the head is in the socket, the end of the shaft in the axilla is not like the head, there is not much difficulty in pressing the elbow to the side and removing the deformity, but it returns at once on removing the restraining force, and crepitus is marked when the conditions are suitable.

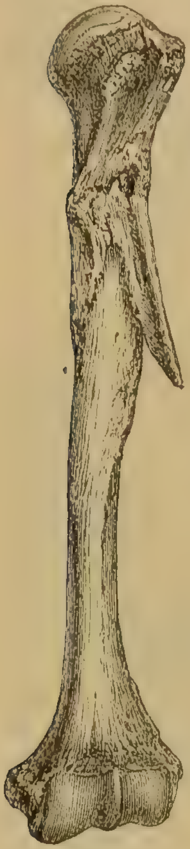


Fig. 251.

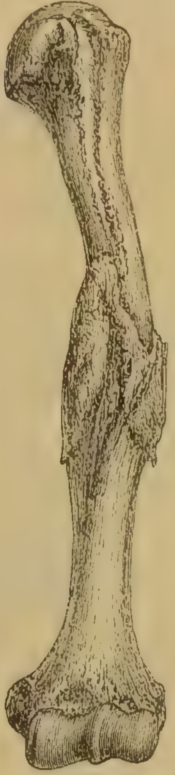


Fig. 252.

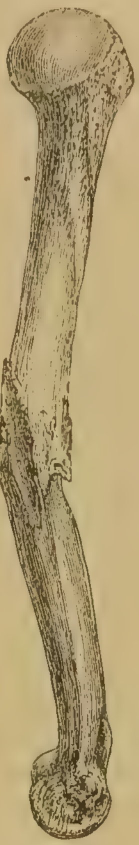


Fig. 253.

Fig. 251.—A Humerus repaired after a comminuted Fracture at the lower End of the bicipital Groove. The upper fragment has been drawn inwards. A splinter 2½ ins. long projects into the insertion of the deltoid, having apparently been displaced by that muscle. (University College Museum, No. 197.)

Fig. 252.—A Humerus repaired after Fracture about the Middle. The upper fragment has been abducted by the deltoid. The lower fragment lies behind and inside the upper. A column of dense bone, 3 ins. long, and nearly as thick as the shaft fills up the angle inwards between the fragments. (University College Museum, No. 200.)

Fig. 253.—A Humerus repaired after transverse Fracture about the Middle. The fragments form an angle salient forwards, and the lower fragment is rotated slightly in upon the upper. A deep groove is shown which was apparently occupied by the ulnar nerve. (University College Museum, No. 199.)

Fractures of the shaft of the humerus occur at all ages, and from all forms of violence; but of 85 cases of fracture of long bones from muscular action, 57 were of the humerus. More or less transverse fractures are common in children; later, the line is usually oblique, with tendency to shortening. All the signs of fracture are, as a rule, easily obtained. When the fracture is between the pectoralis major and the deltoid, the upper fragment is drawn in and the lower out (Fig. 251); when it is below the deltoid, the upper fragment is drawn out (Fig. 252) and the lower is undisturbed, except that, in all fractures of the shaft and upper end, the longitudinally running muscles endeavour to draw the lower fragment up. The arching forwards shown in Fig. 253 is quite unusual so high up.

The special complication of fractures of the shaft is primary injury to nerves or their later involvement in callus, the musculo-spiral being that which suffers in most cases.

Treatment.—Rest in bed on Stromeyer's or an ordinary cushion, and ice, if the injury have been severe. When swelling is subsiding, or at once in the slighter cases, apply three or four short padded splints of Gooch's material round the arm, and secure them by a webbing band buckled around them above and below the fracture. If they must be firmly applied, the parts below must be bandaged. The hand is slung. If extension be required, an outside or inside angular splint (with a ring pad over the epicondyle) should be substituted for one of the above, fixed to the fore-arm by bandage, and extension should then be made, as shown in Fig. 254, until shortening is removed; the buckles should then be tightened. Fractures so high that no satisfactory hold above the fracture can be obtained by the upper strap are best treated like fractures of the surgical neck.

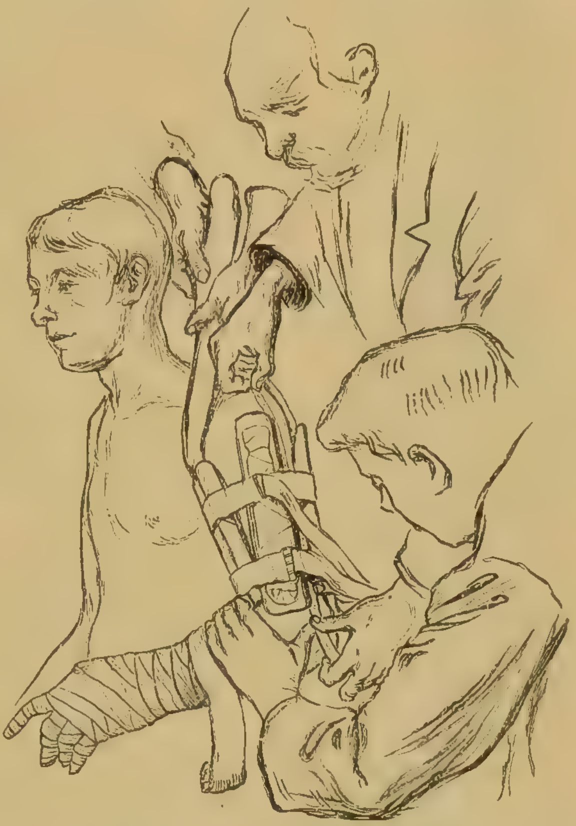


Fig. 254.—Treatment of Fracture of the Shaft of the Humerus by internal angular and short side Splints. The hand and fore-arm are bandaged; the splints are buckled loosely round the arm; extension is being made from the elbow, whilst a towel held by an assistant forms a point of counter-extension; the surgeon tests the result with a tape.

Pseudarthrosis is commoner in the humerus than in any other long bone, especially in the middle third (Fig. 229).

Fractures of the lower end of the humerus.—These injuries are very frequent in the first and second decennia; if Bruns's statement, based on 4,000 hospital cases, may be trusted, they are comparatively rare after forty. There are several types described. (1) The supra-condyloid; (2) separation of the whole lower epiphysis; (3) the T-fracture and comminuted fractures; (4) fracture of the capitellum or of the trochlea; (5) fracture of the internal or external epicondyle or separation of the corresponding epiphysis.

1. **Supra-condyloid fracture,** in which a transverse or oblique fracture crosses the bone above the epicondyles: the lower fragment, with the fore-arm bones in their normal relation to it, is usually drawn backwards by the triceps, moving on the lower end of the upper fragment as upon a hinge, or displaced wholly backwards behind the upper.

Signs.—Seen from the side, with the fore-arm flexed or hanging, the elbow projects markedly backwards, but its point is not very acute; there is bony swelling in front, immediately above the flexor fold, and an obtuse angle opening backwards and upwards at the same level behind. The arm, from acromion to epicondyle, is a little shortened, but the fore-arm, from epicondyle to styloid process, is normal. The relations between the olecranon and the two epicondyles are normal, and neither the greater sigmoid notch of the ulna, nor the pit on the head of the radius, can be felt. The upper fragment has not the shape of the lower end of the humerus. Deformity is easily reduced with crepitus, and easily recurs.

In exceptional cases, and especially when the transverse fracture is low—perhaps even crossing the cartilage-covered surface—the lower fragment is displaced forwards. Side to side obliquity of the line of fracture will tend to cause the lower fragments to glide in or out. The joint may be opened in any ordinary case; various nerves and vessels are liable to injury from it. When compound the upper fragment generally protrudes in front.

2. **Separation of the lower epiphysis.**—This epiphysis is so shallow that it forms little of the olecranon fossa, but it mounts on each side to the epicondyle; the lower end of the shaft becomes with growth increasingly convex, especially from side to side. Centres appear in the capitellum at 2 to 3 years, in the trochlea at 11 to 12, in the external epicondyle at 13, and in the internal epicondyle at 5. The first three soon blend and unite with the shaft at 16 to 17, whilst the internal epicondyle unites at 18. After 10 the epiphysis adds only about 1 cm. to the length of the bone. Up to 5 or 6 a pure separation of the whole cartilaginous end is probable; later, a mixed separation and fracture are more likely. The fracture may be incomplete, the epiphysis being "bent" inwards or outwards. In one case (age 12) examined anatomically, the epicondyles did not accompany the capitellum and trochlea. In over fifty cases (between 1½ to 14 years) J. Hutchinson, junr., found no instance of suppuration in a simple case. Callus in front of the joint tends to be exuberant and to limit flexion, especially if reduction be imperfect.

Signs.—As in supra-condyloid fractures in complete cases. There may be no displacement, only an antero-posterior "give" just above the elbow (J. Hutchinson, junr.).

3. **The T-fracture** (Fig. 255) consists of a more or less transverse fissure, crossing the bone at a varying height above the articular surface, and of another, vertical, which descends from the former and generally through the trochlea. It is not very common, and usually occurs in adults, being almost always the result of direct

violence—falls or blows upon the elbow; still greater force may cause much comminution. A fracture of the olecranon is a not infrequent concomitant of these serious injuries, which are always accompanied by great swelling and are often compound or complicated by injuries to nerves or vessels. As to displacement—almost anything may happen; but usually the lower fragments are drawn backwards and upwards, whilst the upper fragment sinks towards the joint between them, forcing them apart.

Signs.—If there be not too much swelling, the lower end of the humerus seems much widened; everything is loose, crepitus perceived at each movement; the relations between the olecranon and epicondyles are more or less abnormal, yet the sigmoid notch and radial head cannot be felt; when extension is made, the condyles can be pressed together, and moved upon each other.

4. **Fractures of the trochlea or capitellum,** usually with the corresponding epicondyle, are common accidents in this region, especially in children. They arise from falls upon the hand, and violent lateral wrenches of the joint.

Signs.—In these cases, when seen early, swelling, pain, and tenderness may be limited to the side injured. Dislocation of the fragment will disturb the relation between the epicondyles and the olecranon; the ulna accompanies the trochlea, and when the two slip backwards, the radius also may be found below and behind the capitellum. The tendency to displacement of the radius in fracture of the capitellum is less marked. Pressure on the epicondyles shows mobility and crepitus, and these may be detected, together with angular movement, in the joint when the elbow is extended and the fore-arm abducted or adducted. Carried to excess, these movements would easily induce dislocation of the fore-arm bones, especially in the outward and backward direction when the trochlea and epicondyle are loose. Dislocation of the fore-arm bones and injury to the ulnar nerve are the chief complications.

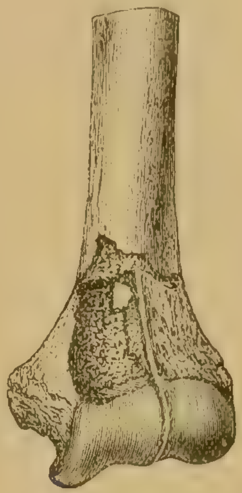


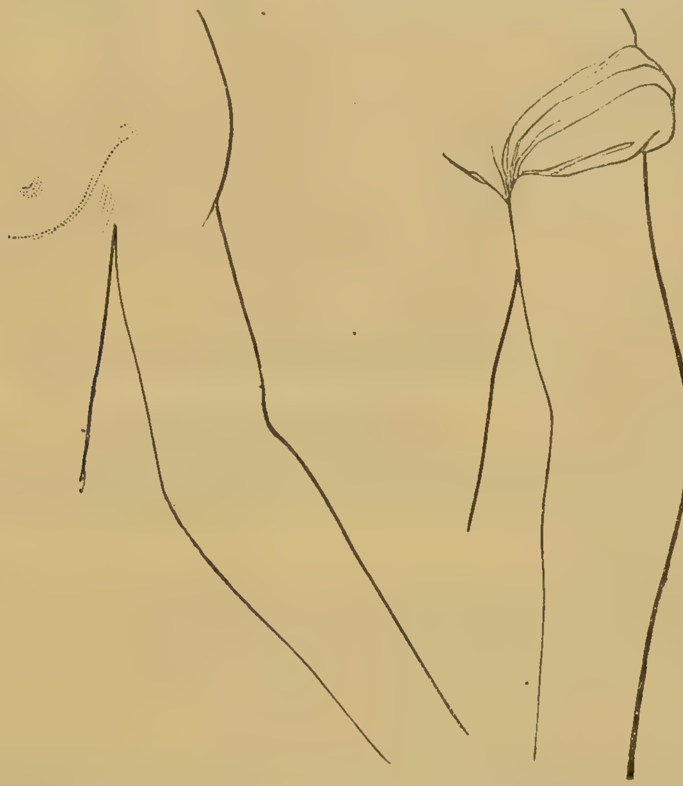
Fig. 255.—A comminuted T-Fracture of the lower End of the Humerus; from a man who fell from a ladder. The fracture was compound and suppurated. Amputation after one month. There is scarcely a trace of new bone about the fragments. (University College Museum, No, 202.)

5. **Fracture of the internal or external epicondyle.**—The latter is very rare, and is due to direct violence or muscular action. The small fragment is felt at the seat of pain, held in position by untorn tendon, or displaced downwards by extensor muscles. It unites by bone if not displaced, otherwise by ligament. In either case little or no inconvenience results.

The internal epicondyle may be broken off by direct violence, or by muscular and ligamentous traction in falls bending the fore-arm outwards; it is not uncommonly pulled off in dislocations of the elbow, and its condition should be ascertained after reducing all such injuries. Detachment of the internal epicondyle always opens the elbow joint, and, the internal lateral ligament being attached to it, permits abnormal abduction of the fore-arm when the elbow is extended so that dislocation occurs under slight violence. The bit of bone is displaced downwards by the superficial flexors, and can be felt; no crepitus, unless it can be replaced; a sharp termination of the internal supra-condyloid line can be felt. Union is almost always fibrous, of greater or less length. The ultimate result is generally satisfactory, but to obtain this passive movement has, as a rule, to be kept up for weeks. J. Hutchinson, junr., believes that this is due to pressure on the ulnar nerve with each attempt at flexion after its guardian process has been removed. In more severe cases of neuralgia, paresis or paralysis in the ulnar area, with great rigidity of the joint, the process has several times been found pressing on the nerve, and has been removed with a perfect result.

Diagnosis of injuries to the elbow.—Early examination to anticipate swelling is most desirable, and an anæsthetic is often necessary. With the patient's hands clasped in the mid-line, the injured joint should be carefully surveyed from all sides. If the elbow be projecting unduly backwards, the resemblance to a dislocation backwards of the ulna, or of both bones, is close. *In the dislocation*, the olecranon by itself forms the point of the elbow, and the projection is therefore sharper, both from above down, and from side to side, than when the joint end of the humerus moves back with the fore-arm bones; the triceps is tense and curved, the curve being somewhat lower and sharper than in the fracture; the length from the acromion to epicondyle is normal, from epicondyle to styloid process short. Taking an elbow in each hand, holding them symmetrically, and placing the forefinger on the olecranon, and the thumb and middle finger on the epicondyles (the elbow should always be examined in this way), it is found that the distances between the olecranon and both epicondyles are much increased in the dislocation, but are normal in the fracture; the projection in front is smooth, and presents the irregularities of the lower end of the humerus; the fingers can be pressed into the sigmoid notch of the ulna, and the pit in the head of the radius; movement is limited in the dislocation—extension is incomplete, and flexion cannot be carried to 90°—it is abnormally free in the fracture; reduction is more or less difficult, but when accomplished, the deformity does not easily recur; there is no crepitus. Systematic attention to these points when they can be recognised will enable us to distinguish between dislocation backwards of both bones and the supra-condyloid fracture, T-fracture, and separation of the epiphysis, with the ordinary displacement. If the displacement is extraordinary, the resemblance to a dislocation is lost; and none exists in the other cases above described.

Treatment of fractures of the lower end of the humerus.—In the more severe simple injuries there is often so much swelling when the patient is seen that effective examination is impossible; the limb should then be laid on a pillow and ice or pressure used to check swelling. As soon as possible a careful examination is made and any displacement reduced. Where no diagnosis can be made, the successful treatment of the fracture depends upon the adoption of that position which is most likely to bring the fragments into their normal relation. In all these injuries there is a difference of opinion as to whether the limb should be put



Figs. 256 and 257.—Cubitum Valgum and Varum after Injuries to the Elbow Joint. In the former the carrying angle is increased; in the latter it is reversed. (From a paper by Mr. Nunn in the Clinical Society's Transactions, 1892.)

up straight or more or less flexed. In favour of the straight position it is said that it is impossible otherwise so to set the fracture as to maintain the natural angle ("carrying angle") of about 170° outwards between the humerus and ulna—cubitum valgum and varum (Figs. 256, 257), corresponding to genu valgum and varum, being the result. This being regarded as a test-point of perfect reduction, it is held that stiffness is more likely to result from the flexed position. Those who use the flexed position—and they include the great majority—deny this, and quote good results. They speak of stiffness resulting frequently from the straight position—a much more disastrous result than a stiff elbow at 90°—and of the discomfort of the constantly hanging limb to the patient. It may be added that cubitum valgum and varum seem to interfere little with the use of the limb, though they might be a bar to entering the public service. There is probably truth on both sides. The displacement in each case must be studied, and the position found in which it will be most perfectly reduced. In doubtful cases flexion to 90° is probably the best position. When a fracture has been complicated by a backward dislocation of the fore-arm bones, a position of sufficient flexion to prevent risk of recurrent dislocation *must* be employed. With a low transverse fracture, full flexion, advocated by some, may produce forward displacement.

Flexion is best maintained by a posterior gutter splint of plaster of Paris or tin bracketed at the elbow (Fig. 258), a short anterior being applied to the arm if there is much projection forwards ; the two are bandaged together and weight-extension in the line of the humerus may be added during the early days if tendency to shortening is marked. The fore-arm should be in the mid-position, so as to render tense neither the superficial flexors nor the extensors. The maintenance of flexion by an internal or external angular wooden splint is likely to cause a lateral displacement. Massage and movements should be practised as soon as possible without causing pain or displacement.

In fractures of the epicondyles, and probably of the trochlea or capitellum, it would be possible if the fragment could be got into good position to fix it by means of an aseptic acupressure pin driven through it into the sound bone beyond ; the pin to be removed in two to three weeks.

In compound cases the treatment is usual. A stiff though strong elbow is less useful than a movable but weaker joint ; consequently, when the injury seems to threaten ankylosis, the question of primary excision must be considered, and should certainly be undertaken in much comminuted cases : it consists in picking out fragments and smoothing corners. In separations of the epiphysis the end of an irreducible shaft has been removed with good result.

Fractures of the bones of the fore-arm.—Either ulna or radius may be broken alone, or both bones may be broken together.

Fracture of the olecranon (1 to 2 per cent.) is the most important injury of the ulna, making up about one-third of the fractures of the ulna alone. Fracture of the olecranon is generally due to a fall or blow upon the elbow ; as, however, it is unusual to find marks of violence on the skin of the part, it is probable that muscular action plays an important part and breaks the olecranon across the trochlea when the hand comes upon the ground. Fractures from pure muscular action are rare, and the fragment torn off is small. The tip of the process may be detached without opening the joint ; but almost always the fragment encroaches on the cartilage-covered surface, sometimes detaching the whole process above the coronoid.

The fragment is drawn up more or less according as its fibrous coverings are more or less torn through, and this is effected chiefly by muscular action. The joint is swollen early, and a hæmatoma forms over the cleft, which can usually be felt through it on the subcutaneous aspect; active extension of the arm, even in the horizontal plane, is usually impossible; the fragment can be moved, and crepitus is easy. With complete detachment of the olecranon, dislocation forwards of the ulna occurs easily.

Union is almost always fibrous, of greater or less length; bony union, without operation, probably occurs only in cases from direct violence with little or no separation. When fibrous union is long, extension is proportionately feeble, and may be impossible against gravity. Rarely the upper fragment has become attached to the humerus without absolutely preventing extension, or the joint has become fixed. Rheumatoid changes of the elbow seem to be excited with special frequency by injury.

Comminuted and compound injuries are uncommon.

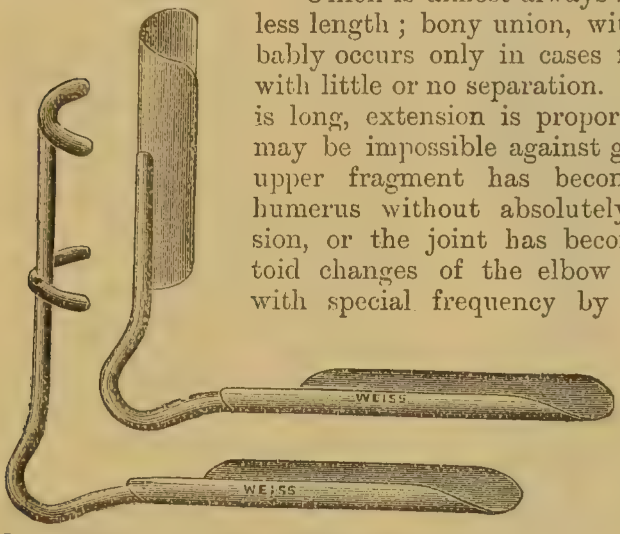


Fig. 258.—Posterior angular Gutter Splints of metal bracketed at Elbow. Most useful in compound cases.

Treatment. — The joint should be aspirated if it contain much fluid. Some endeavour to bring down the upper fragment by means of long strips of strapping passing over it from arm to fore-arm, or by a U-shaped piece placed above the fragment, while the limbs are carried over the front of the fore-arm. If no separation be caused by flexion, the limb may be fixed by plaster in this position. As a rule, the best position is slight flexion; full extension may lead to dislocation forwards of the ulna or to pushing of the olecranon out of the fossa into which it should sink. This position is best maintained by plaster-of-Paris bandages with or without an anterior splint from axilla to wrist, thickly padded opposite the elbow. If the fragments do not lie well together, the upper may be fixed against the lower by cutting a large window over the back of the joint, fixing a pair of Malgaigne's hooks, with antiseptic precautions, through the triceps on to the upper surface of the olecranon, and the other pair into the lower edge of the window.

Another plan is Mayo Robson's, of passing one stout needle through the triceps as close as possible to the bone, and another through the fibrous tissue over the lower fragment (Robson now uses this plan), or through a hole drilled in the latter; the projecting ends are then firmly wired together. Both fragments may be drilled, and a wire suture passed through them from above downwards; or the lower may be drilled from side to side, the wire being passed deeply through the fibres of the triceps; or a screw may be put in downwards from the insertion of the triceps. For all these operations a considerable flap should be raised, inwards or outwards. I have tried Robson's operation once, drilling the lower fragment, short fibrous union resulting.

When the fragments are wired or screwed together no splint is necessary, only a compressive dry dressing. Massage should be begun as soon as the wound has healed or sooner, and the patient encouraged to perform gentle movements.

A long fibrous union may be excised, the fragments refreshed and wired or screwed with excellent result. Ankylosis in bad position, not yielding to forced movements, will probably require excision.

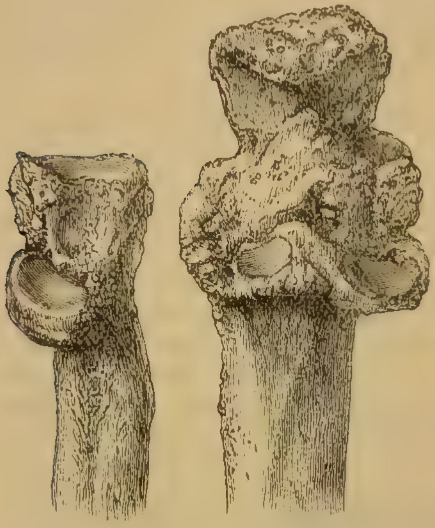


Fig. 259.—Union by Bone after Fracture and downward Displacement of the anterior half of the Head of the Radius and of the Coronoid Process. The humerus, but little altered, lay in a socket formed by the surfaces from which the above portions had been torn and the fragments themselves. No history. (University College Museum.)

Separation of the upper epiphysis of the ulna.—It ossifies at ten and joins at seventeen; it scarcely forms a third of the olecranon. Separation of it is very rare.

The coronoid process is very rarely fractured, unless unrecognisable bits are chipped off in backward dislocations, which Malgaigne thought frequent. The process, as Liston said, may be either pushed or pulled off—by falls on the hand or by muscular action. He believed the latter cause to have produced the accident in a boy who hung for long on a wall, afraid to jump down.

Signs.—Detachment of a considerable piece is said to cause swelling in front of the elbow, where the fragment may be felt; easy dislocation backwards of the ulna or of both bones, and easy reduction. Crepitus if the fragments can be approximated. Union would probably be fibrous; and Sir A. Cooper recorded a case believed to have been a fracture of this process in which the olecranon slipped backwards and forwards somewhat in extension and flexion. That bony union is possible of a small fragment without displacement is shown by Fig. 259, in which the tip of the coronoid process and front of the radial head have been pushed off; partial dislocation backwards of the bones occurred, and the humerus modelled a new surface for itself.

Treatment.—Flexion to 50° or 60° would meet the requirements.

The shaft of the ulna is not commonly (2·5 per cent.) broken alone: it occurs from direct violence, at the point struck; or, in children, from falls on the ulnar side of the hand with the elbow flexed—a greenstick fracture often resulting. The superficial position of the bone renders the detection of fractures comparatively easy, even though displacement be slight. It is worth noting that a marked angle may develop in the ulna, towards the subcutaneous edge, the radius being sound.

Separation of the lower ulnar epiphysis, ossifying at four to five years and joining at twenty, is very rare (Fig. 205).

Fractures of the radius.—The radius is liable to fractures of all parts, but fracture of the lower end and separation of the lower epiphysis are the most frequent injuries; then come fractures of the shaft, those of the head and neck being rare.

Fractures of the radius alone are slightly more frequent than fractures of both bones; they are common throughout life, separation of the lower epiphysis being specially common up to twenty, and Colles's fracture after mid-life.

The **head** may have small or large fragments chipped from it (Fig. 259); remaining loose or uniting, they may greatly impair the movements of the joint. In such a case Cheyne removed about half the head, which lay loose, greatly crippling the joint; he obtained, after six months of treatment, a perfect joint.

Separation of the upper radial epiphysis, a thin plate ossifying at five to six and joining at eighteen, is very rare.

When **the neck** is broken (rare) there is no muscle to displace it, and the balance of power on the shaft is undisturbed; so there is no typical displacement.

In **the shaft** greenstick fractures are common in children (Fig. 204). Fractures of the shaft of the radius are more frequent than those of the ulna, because in falls force travels chiefly along the radius. When a fracture occurs between the tubercle and the insertion of the pronator teres, it is practically certain (Figs. 215, 230, 260), unless the fragments are inseparably locked, that some contraction of the biceps, and perhaps of the supinator brevis, will completely supinate the upper fragment; neither voluntary nor passive pronation of this fragment is then possible, for there is no pronator attached to it and the fragment cannot be grasped by the surgeon. Whether the pronator could pronate the hand after fracture of the radius may be doubtful; but if the hand be pronated, there it remains, for the supinator longus is incapable of supinating it. The

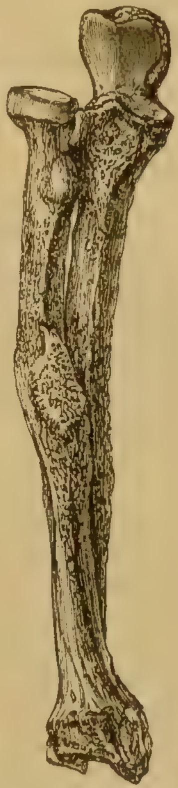


Fig. 260.—Fracture of the Radius just above the Insertion of the Pronator Teres. The upper fragment is supinated, the lower more than semi-prone. The specimen shows the result of treating this injury by anterior and posterior splints. (University College Museum, No. 210.)

only way to bring the fragments into a corresponding position is for the surgeon to supinate the hand—which he easily does. The usual *signs* of fracture may be clear; but the upper half of the radius is so thickly covered with soft parts, and it is so difficult, when swelling is present, to be certain whether the head of the radius moves or not with the lower fragment, that fractures without displacement must sometimes be inferred to exist rather than diagnosed.

In fractures below the insertion of the pronator teres, active supination of the hand is again lost, but the upper fragment remains in the mid-position. It is said that the oblique and transverse pronators tend to approximate the fore-arm bones when broken in their neighbourhood.

Fracture of the shafts of both ulna and radius is less frequent than fracture of the radius alone. Generally both bones are broken about the same level, whether the injury result from direct or indirect violence. The commonest seat is at the middle or at the junction of the upper and middle thirds; the commonest deformity is an angular bend towards the subcutaneous edge of the ulna (Fig. 261); the injury is more easily recognisable than fracture of one shaft only, by greater deformity, mobility, and crepitus. No complication occurs with special frequency, and all are rare; but gangrene from improper treatment has occurred here oftener than elsewhere. Pseudarthrosis is infrequent. Luxuriant callus occasionally forms, its worst result being obliteration of the interval between the bones and prevention of pronation and supination. When the ulnar and radial fragments are displaced towards each other, their union together is facilitated; and when this displacement can be shown to be due to faulty treatment, an action for malpraxis would probably succeed.

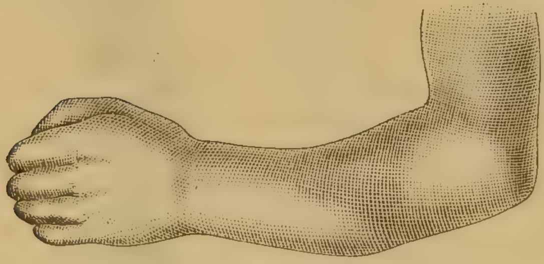


Fig. 261.—A Fracture of both Bones of the Fore-arm. (Cast, No. 219, University College Museum.)

Treatment of fractures of the shafts of the ulna and radius.—Formerly all the above injuries were treated in the mid-position between pronation and supination, between anterior and posterior wooden splints (Figs. 262, 263). These may be used for fractures of the ulna alone, or for fractures of the radius or of both bones below the middle. The splints are cut from a plank ¼ to ⅓ in. thick, wider than the widest part of the fore-arm, and long enough to reach from below the external epicondyle to the knuckles, and from the internal epicondyle to the wrist. The splints having been padded, deformity is reduced, the elbow is bent to 90°, the hand turned until the thumb points upwards, the splints are placed in position and fixed by webbing straps, above and below the fracture, drawn fairly tight. Owing to the width of the splints these do not touch the borders of the limb, and do not press the bones together. The hand is fixed to the longer posterior splint by a figure-of-8 bandage or strapping leaving the thumb and fingers free. The whole length of the fore-arm should be supported horizontally in a sling.

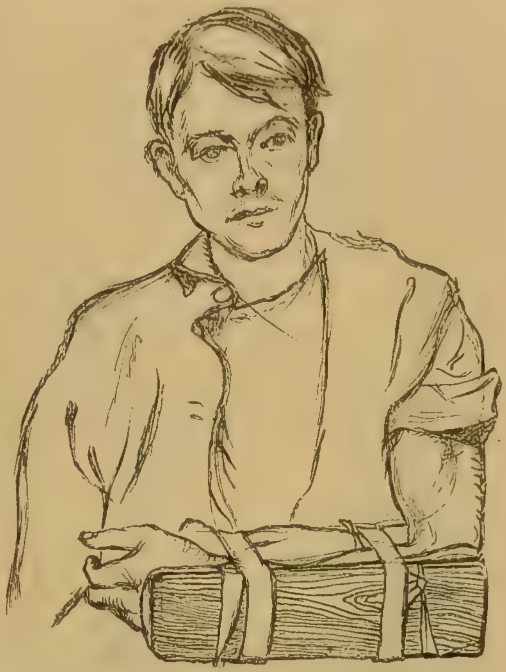


Fig. 262.—Anterior and Posterior Splints for the Fore-arm, padded and applied with Strips of Strapping.

If the fracture be at or above the middle (of the ulna) the hand may be set free, and the extra length of the posterior splint cut off so soon as pain has ceased. The patient should frequently perform all movements of the joints which are free. Massage should be practised regularly, splints being undone when the surgeon thinks it safe; and at each *séance* all joints should be exercised. Attention should be specially directed to the prevention of angular deformity — particularly the dropping of both bones towards the ulnar side above alluded to. This may be very difficult to overcome. In the mid-position the hand should be allowed to hang, the splints should not grip the wrist between them, and special support should be given to the ulna down to the fracture. But in most of these cases a posterior moulded gutter splint should be used, the hand being placed comfortably supine—the thumb pointing up and out.

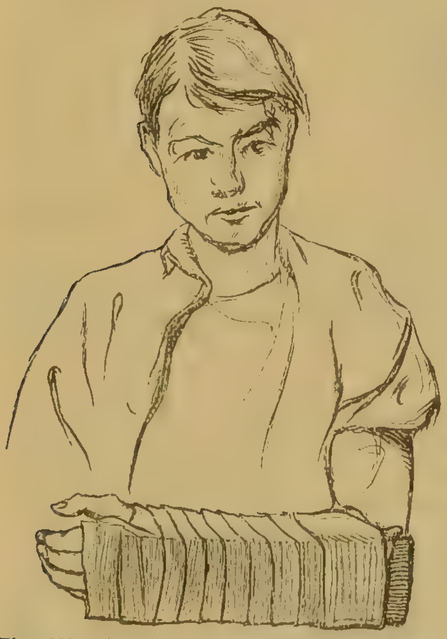


Fig. 263.—Anterior and posterior Splints for Fore-arm. The hand, but not the fingers, is secured to the posterior splint by a bandage which has been carried up to the flexed elbow.

In all fractures in which the radius is broken above the middle, the supine position, and a posterior angular, flat or gutter splint should be used; it may be of plaster, wood, or metal, and should pass far enough up the arm to prevent its displacement. When swelling has ceased an ordinary plaster bandage may be safely used to maintain this position, care being taken not to draw the bones together: but it prevents massage.

Fracture of the lower end of the radius (Colles's fracture).—In this injury a fracture starts on the anterior surface of the radius $\frac{1}{4}$ to 1 in. above the joint surface, and runs transversely backwards, or obliquely up and back. The lower fragment is either displaced directly backwards, or it turns up and back as upon a hinge; usually it is displaced outwards (to the radial side) also, and, rotating in this direction, the styloid process is raised. The posterior wall of the upper fragment not uncommonly penetrates the lower fragment, often splitting it into several pieces; or, again, becoming impacted with extraordinary firmness. The carpus moves outwards with the lower fragment, and sometimes carries the ulnar styloid process with it. The lower end of the ulna may be broken. Further violence may rupture the radio-ulnar

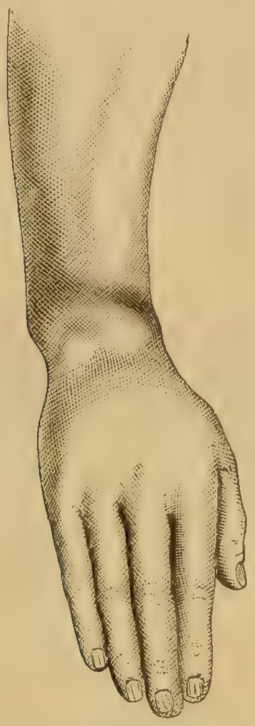


Fig. 264.

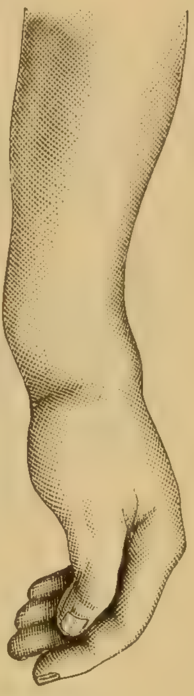


Fig. 265.

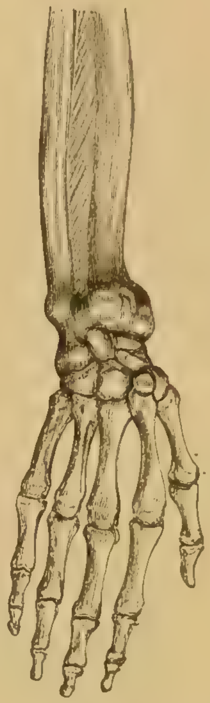


Fig. 266.

Figs. 264 and 265.—Back and side Views from a Cast of a Case of Colles' Fracture, showing the points noted in the text. University College Museum No. 215.)

Fig. 266.—The Bones from a Case of Colles' Fracture, explaining the deformity; the displacement backwards and outwards of the lower fragment with the hand, the rotation of the lower fragment on its transverse axis so that its carpal surface looks down and back, and the prominence of the ulnar styloid are the points most clearly shown. (University College Museum, No. 211.)

ligaments, and drive the ulna through the skin (Figs. 264, 265, 266, and 267).

This fracture is very common, especially in elderly women; but it occurs at all ages and in both sexes. Almost always the cause is a fall on the hand—in the old merely to the ground, in younger patients from a height. The shock is received upon the palm near the base of the thumb, which point becomes fixed; the radius, directed downwards, forwards, and more or less inwards, is driven by the momentum of the body on to this point, as a rule; firm resistance is offered; the lower end becomes fixed; the force acting along the oblique shaft may be resolved into vertical and horizontal components; the effect of the vertical component is to crack through the lower end of the bone and to drive the end of the shaft downwards (towards the ground); the effect of the horizontal component is to drive the shaft forwards into the lower fragment, and, if this has not been carried completely backwards, the radial and posterior walls of the shaft will crush and penetrate the cancellous tissue of the lower fragment. All this can be readily seen if, sitting squarely, we place the palm on the table in front of us, on one side of the mid-line, and imagine first the effect of a vertical blow on the lower end of the radius, and then that of a violent push of the shaft directly forwards. Slight differences in the direction of the radius—whether it is more or less nearly vertical, whether it inclines more or less inwards—will account for all the differences we have noted in the anatomy of the fracture. As a rule, extension of the wrist in these falls is insufficient to stretch the anterior radio-carpal ligament greatly; but experiments on the cadaver and a few cases show that the fracture may be produced by over-extension of the wrist, though sometimes the ligament yields. The dislocation of the lower fragment upon the upper may be maintained either by impaction or by spasm of the radial carpal and thumb extensors. In a very few recorded instances falls on the back of the hand and flexion of the wrist have caused a similar fracture with displacement forwards of the lower fragment.

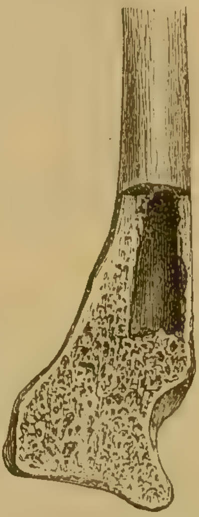


Fig. 267.—Antero-Posterior Section through a Radius from a Case of Colles' Fracture. The displacement back and rotation (moderate) of the lower fragment are shown; the angle forwards above the wrist can be estimated and contrasted with that in Fig. 268. (University College Museum, No. 213.)

Signs.—There may be no deformity; but when the displacement is maintained the appearance is very characteristic. Seen from the radial side, the hand and fingers are more or less flexed, but the outlines about the wrist are irregular; at the flexor fold there is a deepish depression, and above it is a prominence—the lower end of the upper fragment; on the dorsal aspect, opposite the latter, or rather higher, is a depression due to displacement back of the lower

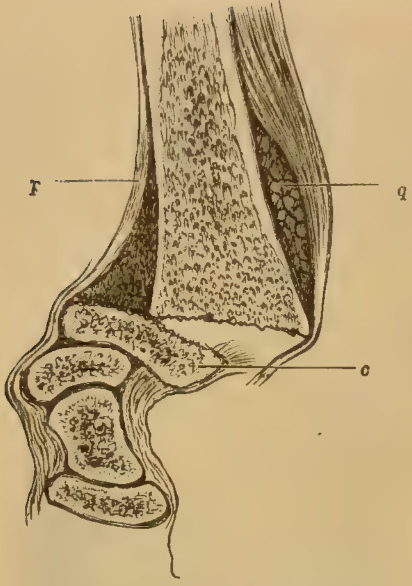


Fig. 268.—A preparation showing Separation of the lower Epiphysis of the Radius, and explaining the Deformity. (Royal College of Surgeons.) Figs. 268, 269 are from Mr. Hutchinson s drawing.

q, Pronator quadratus; r, widely separated periosteum; c, displaced epiphysis.

fragment, whilst lower down, opposite to and below the wrist joint, is a marked prominence formed by the lower end of this fragment and the carpal bones flexed upon it. The side view was aptly likened by Velpeau to that of a silver fork. Seen from the back or front a greater or less concavity is noted on the radial side above the wrist, and a corresponding prominence of the head of the ulna; besides being carried outwards the hand is generally a little abducted, and the thumb and other tendons are tense and prominent. Reduction of the deformity may be easy with crepitus, or very difficult from impaction. Evidence of comminution of the lower fragment, and especially of splitting off of the posterior edge of the radial surface, may be obtained.

Separation of the lower epiphysis of the radius is perhaps the commonest accident of this kind. The epiphysis begins to ossify at two years and joins at twenty to twenty-two. The accident may occur at any age up to twenty, and the majority of the patients are over ten. The causes, pathology, and signs are those of Colles' fracture, except that the lower end of the shaft projects with great sharpness (Fig. 268), forming an angle forwards of 90° to 100°, whereas the corresponding angle in Colles' fracture is 140° or more (Hutchinson). The accident has not infrequently been compound. Hutchinson found ten such among fifty-four; and though they were nearly all recent cases, the results obtained were very bad. In four the shaft was resected to reduce, with a good result in all; in six reduction was effected, more or less imperfectly; two died of pyæmia and tetanus, one recovered with a crippled wrist after long suppuration, three were amputated for gangrene. Other complications

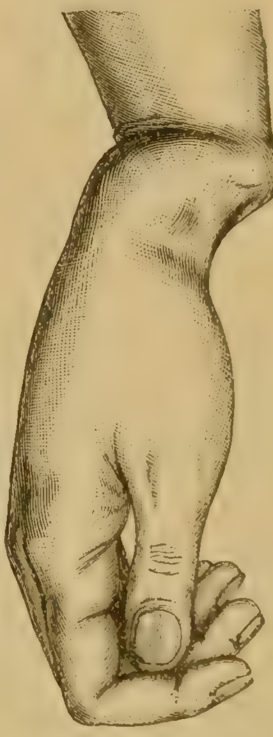


Fig. 269.—The Deformity in a recent Case of Separation of the lower Epiphysis of the Radius. The angle in front should be specially noticed.

noted are: tearing off of the lower ulnar epiphysis, or, more often, fracture of the shaft 1 inch up; and obstruction of main arteries by pressure. Suppuration of simple cases is not to be feared, though the periosteum is widely separated (Fig. 269). Arrest of growth is quite exceptional, and should be treated by resection of enough of the lower end of the ulna.

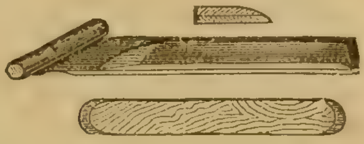


Fig. 270.—Carr's Splints for Colles' Fracture.

Diagnosis. — Distinction between the fracture and separation of the epiphysis rests upon (1) age—though Colles' fracture may occur in young people; (2) the situation of the cleft; (3) the acuteness of the angle forward; and (4) the character of the crepitus. Both the above accidents are distinguished from dislocation back of the wrist by the normal relation which the radial styloid process bears to the carpus and by the absence of any resemblance in form between the prominences on the front and back of the wrist to the concave smooth lower edge of the radius and the rounded convexity of the carpus.

Treatment of fractures of the lower end of the radius.—The deformity should be reduced by traction on the hand, and especially by direct pressure on the fragments: anæsthesia may be necessary. Permanent deformity markedly limits flexion movements. There may be no deformity; when deformity has been reduced it may not recur, or it may return as soon as the part is free. Only in the latter case is a splint absolutely necessary, and even here it can probably be abandoned in seven to ten days. The best splint is Carr's (Fig. 270). After reduction has been effected the limb is laid on the palmar splint covered with a layer of boric lint, and the patient is made to grasp the oblique bar—when this is done any deformity disappears; if necessary, the fingers are closed round it, and for twenty-four hours, or till spasm has subsided, fixed by bandage over wool; the little dorsal splint is applied, and the roller run up

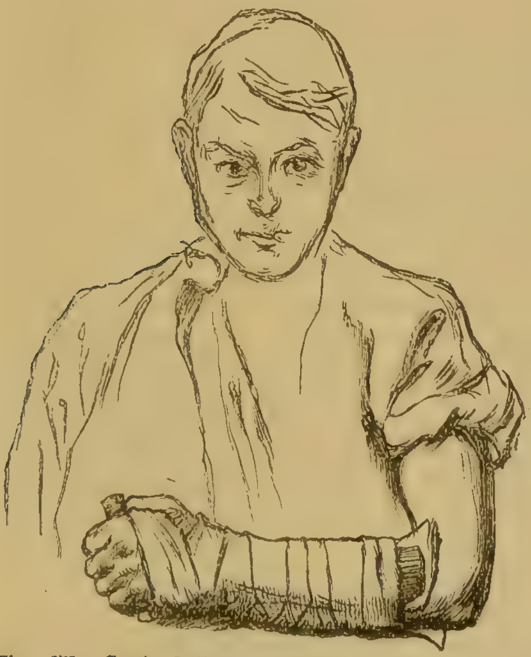


Fig. 271.—Carr's Splints applied. The line of the knuckles shows adduction of the hand when the bar is grasped.

the arm (Fig. 271). As soon as spasm has subsided the fingers and thumb are left free and movements of extension, abduction and adduction and of flexion (grasping the bar) should be frequently practised.

If Carr's splint be not at hand, anterior and posterior splints (Fig. 262) may be used. The hand is left hanging to the ulnar side, lightly restrained by a bandage across the palm, or, if there be much spasm, a strip of strapping may be used to adduct it and fix it firmly against the splint. The posterior splint is thickly padded up to the level of the wrist, the anterior down to the level of the fracture; the splints are buckled firmly together. Again, freedom to the hand and fingers should be allowed as soon as possible. Massage should be practised before putting the fracture up, and should be repeated as soon as spasm has subsided. With less tendency to deformity, cases may be treated with a sling, over the edge of which the hand hangs adducted by its weight, or, to prevent accidents, a light wooden back-splint may be worn. Massage is practised daily and all movements, voluntary and passive, are begun as early as possible, the surgeon giving proper support. By this treatment the long-lasting stiffness and painfulness of fingers and wrist, which used to form the chief seriousness of this accident, may generally be avoided. In separation of the epiphysis extravasation may be very great; splints must not be too tightly applied at first, circular compression must not be practised, and the fingers should be carefully watched.

The carpal bones.—These are generally fractured by severe crushes, often doing much damage to soft parts, which constitutes the most serious part of the injury. There is little or no displacement, as a rule, but a fragment may be forced quite out of position. So far as the fracture is concerned, short immobilisation and elastic compression to limit extravasation are necessary at first.

The metacarpal bones and phalanges.—These bones are generally broken by direct violence; the phalanges are liable to have pieces torn off their bases by twists to the side, and they then, when not supported, drop over from the injured side. The flexor muscles often cause projection backwards of the metacarpal and larger phalanges when broken near the centre. The usual signs of fracture are easily obtained. The *treatment* of simple cases is best carried out by a gauntlet moulded in the case of a metacarpal, and by a palmar trough of guttapercha, carried into the palm, if necessary, for the phalanges. In compound injuries and crushes, treatment should be most conservative: nothing should be amputated because we think it will not live—render the parts aseptic and wait. Crushes of the last phalanx rarely require amputation at the last joint: if a flap can be obtained for this, the part can probably be saved. The dressing is usually a splint also.

Fractures of the pelvis.—These fractures constitute only 0·3 per cent. of the whole list of fractures. They are best classed according as they do or do not break the pelvic ring. The former are by far the more serious, and are almost always due to forcible compression, as when a heavy weight falls upon a man or his pelvis is driven over or crushed between buffers. The results are very various. A fracture of the horizontal pubic ramus and of the pubic arch below it is common on one or both sides (Fig. 272), and this is often accompanied by a fracture through the lateral mass of the sacrum, or through the ilium on the same side (double vertical fracture) more or less parallel and close to the sacro-iliac synchondrosis. This articulation may be torn open; so, too, may the symphysis—by direct violence, forcible abduction of the thighs, and even by the wedge action of the fœtal head. The pubic symphysis may be driven back; the sacrum is very rarely displaced forwards; one hip-bone may fall back and out, or be drawn upwards; the head of the femur is very rarely driven against the acetabulum with such violence as to split the hip-bone into pieces; it may project into the pelvic cavity.

The less serious class is made up of fractures of the ilium above the ilio-pubic line, of the crest, the anterior spine, the margin of the acetabulum, of the tuber ischii; of transverse fractures of the sacrum and coccyx, and of luxation of the coccyx—all due almost invariably to direct violence. The epiphysis of the crest is said to have been torn off by the abdominal muscles. These fractures are rarely *compound* through the skin, the force that causes them acting over a wide area.

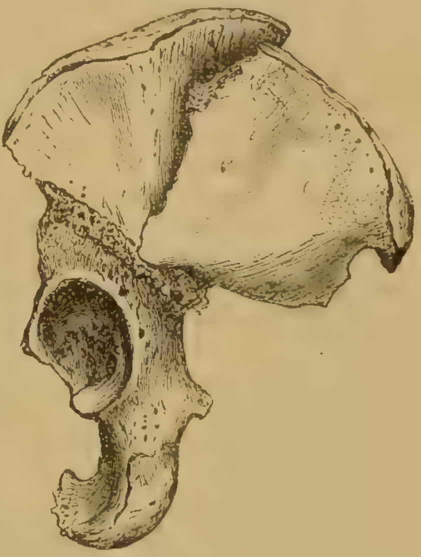


Fig. 272.—A left Hip Bone. The ilium above the acetabulum has been split off and broken into two pieces by a T-fracture; the fragments have united with displacement. There has been a vertical fracture also of the horizontal ramus of the pubis and of the pubic arch. (St. George's Museum, Series I., No. 122.) (Pick.)

Injuries to internal organs and parts by the fragments constitute the chief dangers of these injuries. Rupture of the bladder from the violence causing the fracture, rather than wound by a fragment, occurs occasionally. But *the* special danger is partial or complete laceration of the subpubic (membranous, sometimes bulbous) urethra by displacement of the fragments of the pubic arch in the relatively common anterior vertical fracture. Fixed as this portion of the urethra is, it may be simply kinked; but this is rare. Laceration of the rectum has not infrequently accompanied fractures and luxations of the sacrum and coccyx. Injuries of the great vessels and nerves are very rare; the gluteal artery has generally been the vessel to suffer. An injury of such severity is frequently accompanied by other internal lesions.

Signs.—The diagnosis of fractures not breaking the pelvic ring is made by attention to the nature of the accident, the seats of injury, of pain and tenderness, and by careful examination from outside, from the rectum and vagina. Passive movements of the lower limb may help. As a rule, deformity, mobility, and crepitus, one or all, can be obtained. A dislocation of the femur easily reducible and easily recurring, characterises fractures of the acetabular edge.

In fractures breaking the pelvic ring there is often great shock; if the patient has attempted to bear weight on the limb of the injured side, he has found it impossible: there is a sense of loss of support, and the limb is helpless and rotates out fully, because the muscles have lost their *point d'appui*. *Deformity* is not often present, other than swelling from extravasation. More or less of the hip-bone, the sacrum, or the pubes, may be obviously displaced, and the limb in the first case may appear shortened or otherwise deformed. In the very rare cases in which the head of the femur is driven into the pelvis, the trochanter is depressed, the limb is slightly shortened, and a swelling is felt per rectum. *Crepitus* is often obtained during unavoidable lifting of the patient; it should be sought for only in the gentlest way, seeing the danger with which displacement of fragments threatens the urethra. *Abnormal mobility* will be tested at the same time by direct pressure on the pubes, by directly pressing the ilia towards one another or away from each other by the thumbs on the anterior spines. If in this examination *pain* be referred constantly to a certain region not pressed upon, fracture may be inferred. *Localised extravasations*, especially in the perineum when this part has not been struck, are of the highest value. If left in doubt, treat as if a fracture were present, and watch for the disappearance of symptoms.

The *urethra* must always be examined with a scrupulously cleaned catheter, the orifice of the urethra also being disinfected before the instrument is introduced. There may be blood dripping from the meatus, or bloody urine may have been passed, or there may be retention of some standing; in cases seen late, perhaps even signs of cellulitis from micturition into the cellular tissue round the urethra. The catheter may fail to enter the bladder, its point turning constantly into the perineum, or it may enter after more or less difficulty—the urethra is completely or partially torn across. Or it may enter easily and draw off urine, more or less blood-stained (sometimes very slightly) in small quantity; on moving the catheter about, a further, perhaps considerable, flow may occur, the end of the instrument having passed through the hole into the peritoneum which contains the urine—a rare piece of good luck; on running in a known quantity of warm saline solution no bladder tumour forms and only part of the fluid returns; or a suprapubic tumour forms, but does not disappear when the bladder is emptied—a rupture on the non-peritoneal surface exists. Before using the latter method of examination the surgeon should acquaint himself accurately with the physical signs present in the hypogastrium and per rectum. The early diagnosis of ruptured bladder is often very difficult; it must always be remembered that blood may come down from an injured kidney, and that very little urine is secreted in the state of shock in which these patients often are.

Injury of the *rectum* is easily discovered by examination.

The symptoms of vascular and nervous complications present nothing special.

In the course of treatment *suppuration* has occasionally occurred in the pelvic tissues even in simple cases; but it is far commoner in cases rendered compound by communication with the urethra and by incisions for various purposes. The abscesses forming about the fractures burrow in all directions, often lead to necrosis of fragments, and on bursting form so many urinary fistulæ. A recent patient of mine passed all his urine through an opening situate behind his great trochanter.

Treatment.—Transport must be most careful; on a stretcher, if possible, in all cases in which the ring is broken.

Nothing can be done in most fractures not breaking the ring. A displaced sacral or coccygeal fragment may be replaced and perhaps kept in position by a tampon round a tube. The patients can generally get about in six to eight weeks.

In vertical fractures a wide well-padded band should be buckled closely round the hips after any luxation has been corrected; the lower limbs should be kept at rest between sand-bags. The slipper bed-pan should be used with the least possible disturbance, and care of the back requires much skill. A framework on the bed attached to pulleys for raising the patient may be useful, but these appliances often allow a good deal of movement of the pelvis. A simple case may stand in from two to three months.

Rupture of the urethra may be met by drainage, if possible with a rubber catheter; or by perineal incision, with search for the posterior end and drainage of the bladder through it. Immediate suture of the torn urethra is unlikely to succeed, and the proceeding is lengthy and adds much to the shock. But later, if all goes well, an attempt to resect and suture the divided ends may be made. Extravasation requires free incisions. In ruptured bladder we have the choice between drainage by catheter, or through a perineal cystotomy (Mason) or urethrotomy wound, and suture of the rent. This is most easy to perform on the non-peritoneal surface high up; it is most difficult in Douglas's pouch, largely on account of rigidity of the recti. Nevertheless, suture and cleansing of the peritoneum is the most reliable remedy. A rectal wound must be cleaned and treated to prevent cellulitis and extravasation of fæces; the fracture being compound in the worst way, free incisions will improve matters should they be indicated.

Fractures of the femur.—These form 6 per cent. of all fractures. They occur from all forms of violence, and at all ages; yet it is chiefly the shaft which is broken in the young, the base of the neck, shaft, and lower extremity in the adult, and the narrow portion of the neck in the aged. The fractures are grouped into those of the upper end, of the shaft, and of the lower end.

Fractures of the upper end of the femur include: (1) separation of the upper epiphysis; (2) fractures of the neck; (3) fracture of the great trochanter, or separation of its epiphysis.

1. **Separation of the upper epiphysis.**—Up to eighteen months the femur is capped with cartilage representing the head and great trochanter; this is not known to have been torn off. Then the neck ossifies from the shaft, and separates a centre which appeared in the head in the first year from another in the great trochanter appearing in the fourth. These epiphyses join at eighteen to nineteen, and up to this age either may be detached. There is only one known case (M. 15, run over) in which separation of the epiphysis of the head was proved by post-mortem examination to have occurred (Fig. 273). Hutchinson collected over twenty cases, and Battle gives four more, in which ½ to 1½ in. shortening, eversion, elevation of the trochanter and more or less crepitus were found in patients under twenty; but, as fracture of the neck of the femur occurs, though rarely, in children, clinical evidence cannot be accepted. Bony union seems to have occurred in all but one, in which pseudarthrosis resulted.

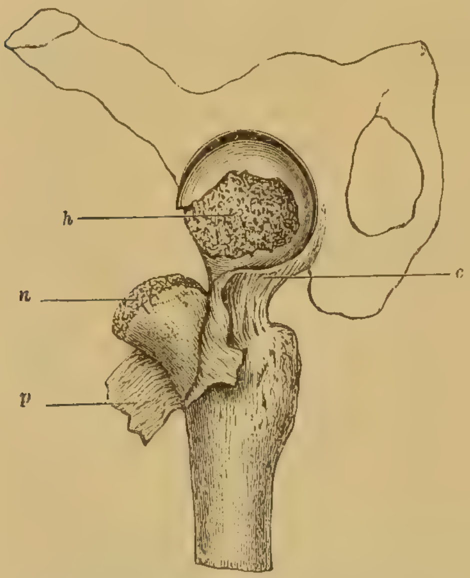


Fig. 273.—M. Bousseau s case of Separation of the Upper Epiphysis of the Femur; from a boy of 15, who was run over by a cart.
h, Head; *n*, neck; *p*, periosteum; *c*, capsule.

2. **Fractures of the neck** are rare in the first and second decennia, then increase rapidly until, from 50 to 90, they form an increasing majority of all fractures of the thigh. Up to 50 they are said to be six times more common in men than in women; after 50 they are 2½ times more common in women. The fractures in earlier life and in male adults are generally situate at the base of the neck, whilst those in later life and in women occur generally near the head.

The *anatomy* of the neck has such important bearings on the injuries of the part, that a few words of reminder as to its structure will not be out of place. The neck projects inwards from the shaft at an average angle of 125° in the adult—the extremes being 110° and 140°. The shorter the stature the lower the angle: hence it is less open in women than in men. There is no reason to suppose that the angle diminishes in advanced age on account of senile degeneration (page 726). At the base the vertical diameter is twice the antero-posterior; passing inwards the former decreases rapidly, the latter increases more slowly, and the two diameters become equal a little below the head—this is the "narrow part" of the neck. In a coronal section of the head and neck we note that the under surface of the neck is formed by a layer of compact bone of considerable thickness continuous below with the inner wall of the shaft, ending above in numerous cancelli passing to the head. The concavity downwards and inwards of this arch increases, and its power of resisting vertical pressure diminishes the lower the angle of the neck. From its upper convex aspect most of the "pressure lamellæ" rise which radiate to the great trochanter, to the upper surface of the neck, and especially to the head, and which are crossed and bound together by the "tension lamellæ" from the outer wall of the shaft and upper boundary of the section. More than half the width of the head is seen to project inwards beyond the extremity of this strut. An oblique section through the same parts at right angles to the coronal plane shows that anteriorly the compact layer thickens to the line of attachment of the capsule (capsular portion of spiral line). Posteriorly the compact layer is everywhere thin, and that covering the roll-like intertrochanteric line seems to overlap rather than to be continuous with the layer on the back of the femur. The more nearly the section approaches the small trochanter, the more clearly do we see a layer of compact bone (calcar femorale) deep to the trochanteric ridge; and we find that it is a section of a vertical plate springing from the inner wall opposite the inferior tubercle of the neck, rising up in the line of pressure and giving origin to many pressure-lamellæ. It is said that the calcar is specially prone to waste in old age. Lastly, it must be noted that, with the toes turned out as usual in lying and standing, the neck slopes markedly backwards and the great trochanter lies in a plane decidedly posterior to that of the head.

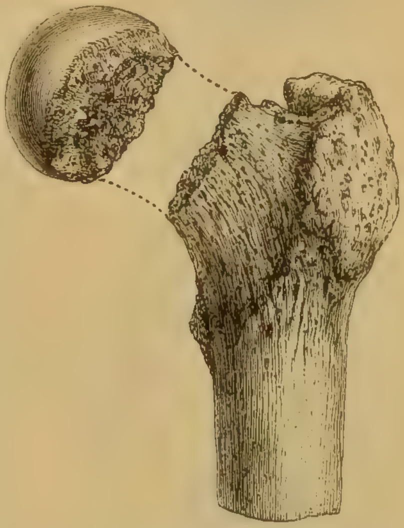


Fig. 274.—A Femur showing moderate Absorption of the Neck, after intra-capsular Fracture. The position of the head in the drawing was determined by careful measurements of the sound bone. In this case there was little or no union of the fragments, and the body-weight was transmitted to the femur by the capsule and muscles. (Charing Cross Museum, No. 375.)

From the above it seems likely that force acting in the line of the body-weight will snap the neck at the "narrow part," and that there will be some tendency for the compact layer under the neck to penetrate the substance of the head. Force acting from the front upon the trochanter may also break the narrow part of the neck, by driving the trochanter back; but the more the force acts directly from the side, the more will it tend to drive the great trochanter on to the base of the neck. The result of this is that the hinder part of the great trochanter and the intertrochanteric line move inwards over the back of the neck, tension is thrown on the front of the neck and trochanter, and the compact tissue cracks open here usually in the immediate neighbourhood of the capsular line. This crack opens like a hinge, as the posterior wall of the neck is driven more and more deeply into the substance of the shaft and great trochanter, becoming impacted more or less firmly in them, or splitting them into more or fewer fragments.

Everything tends to produce eversion: direct force as it usually acts from the side; spasms of the external rotators, the opponents being relatively feeble; and the weight of the limb, which always tends to roll out but is prevented by the ilio-trochanteric band, the action of which is annulled by loss of continuity of the neck.

Varieties.—Sir Astley Cooper divided fractures of the neck into "intracapsular" (Figs. 274, 275) and "extracapsular" (Fig. 276), for which terms Bigelow proposed to substitute "of the narrow part of the neck" and "of the base of the neck." For whilst fractures near the head are wholly intracapsular, a fracture of the neck wholly outside the capsule, which is in front attached to the limit of the neck, is impossible. But Bigelow's suggestion has not been adopted by many.

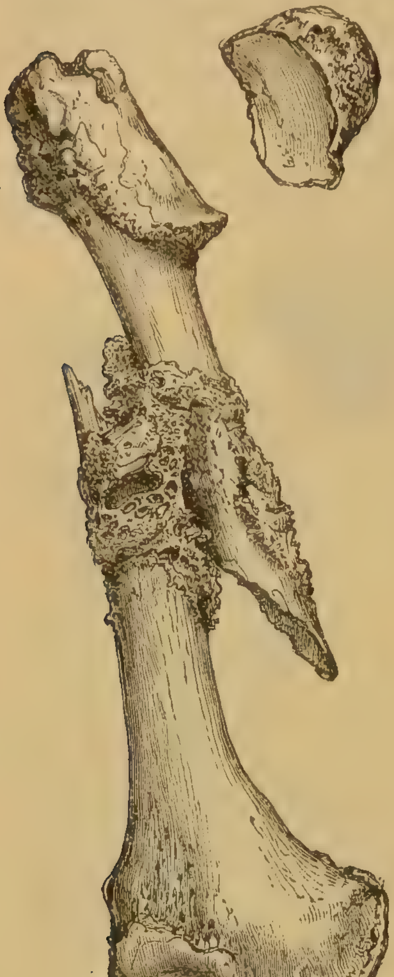


Fig. 275.—From a man aged 99. He had fractured the neck of his thigh-bone some years before his death, and had been able to get about until he sustained a fracture of the shaft. The formation of sores at all points of pressure prevented the use of extension. The neck has practically disappeared; the two surfaces are smooth and polished. The fragments of the shaft overlap for 6 ins., and are firmly united by bony callus. The lower fragment is moderately everted. (St. George's Hospital, Series I., No. 167A.) (Pick.)

Fractures are often mixed—partly intra-, partly extra-capsular; and the exact seat of fracture is often a matter of doubt. Yet it is one of much importance; for intra-articular fractures rarely unite by bone, while those of the base of the neck and trochanter generally do so.

Causes. — Fractures of the neck are caused either by direct violence to the hip, especially heavy falls, or by indirect violence, sometimes very slight, such as the jerk experienced from a stumble, from missing a step or tripping over something. In a few cases violent over-extension of the hip throwing heavy strain on the ilio-femoral ligament has resulted in a fracture, as if the ligament had torn the neck from the shaft.

Signs.—In unimpacted fractures of the neck, shock, pain, and swelling about the hip may be slight or marked, according as the cause of the injury has been slight and indirect or severe and direct violence. In the former case a little patch of bruising often appears after three or four days in Scarpa's space. The limb lies helpless and completely everted, though accidental inversion may be met with; unless swollen by hæmorrhage, the trochanteric region is flattened: a hollow may exist instead of the trochanteric prominence, and even where hæmorrhage obliterates this sign, depression of the trochanter will be made out if the bone can be felt and the two hips compared. The fascia lata on the injured side is lax owing to shortening, and can be pressed in above the trochanter. The trochanter is farther back than normal, corresponding to the eversion (measured by Bryant's triangle, page 751). The limb is shortened from $\frac{1}{2}$ in. to 2 or even 3 ins. and the trochanter is proportionately raised (Bryant's triangle). Shortening is

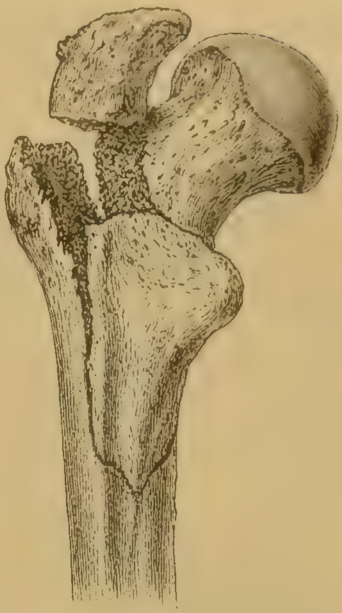


Fig. 276.—A left Femur seen from behind, showing a recent extra-capsular Fracture. This injury follows the lines shown with surprising constancy; to complete them, the anterior limb of the V below the small trochanter must be carried up along the capsular line to the cleft shown between the neck and the upper end of the shaft. This specimen was from a man of 67 who fell in alighting from a 'bus; there were only half an inch shortening and slight eversion. Sudden death occurred two days later with dyspnœa, but no embolus was found. (Charing Cross Museum, No. 379.)

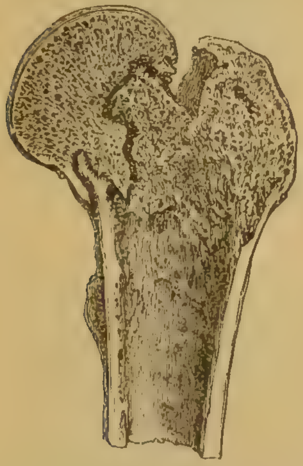


Fig. 277.—The posterior half of a left Femur, showing union after an impacted intra-capsular Fracture, the lines of which are clear. The neck has been driven almost straight into the head, producing depression of the trochanter, slight shortening, and perhaps slight eversion. (Charing Cross Museum, No. 378.)

slight in proportion as the displacing force is slight and leaves more or less intact such structures as the periosteum, and the retinacula or reflections of the capsule upon the neck; it is kept within moderate limits if the Y-ligament retains its hold on the outer fragment; locking of the fragments will also keep it down. The trochanter, though displaced, may feel normal or widened, or it may be represented by loose fragments. In the outer part of Scarpa's space is a tender bony swelling, the angle between the fragments. Passive movements of the limb are painful: inversion can generally be produced with crepitus in fractures of the base of the neck, and in such crepitus is generally marked. In fractures near the head inversion can scarcely be produced, and crepitus is obscure owing to insufficient play between the fragments. The arc of rotation of the trochanter lessens as the fracture approaches the base of the neck, when the trochanter merely turns on the axis of the shaft; it describes no arc. The minor degrees of this change are difficult to recognise.

The following table gives the points which enable a surgeon to form an opinion as to whether a fracture of the neck is situate near the head or near the base:—

	Near the Head.	Near the Base.
Sex, age, and health	Female: past mid-life: feeble.	Male: young or adult: strong and healthy.
Violence	Slight: indirect through the head.	Great: direct to the trochanter.
Bruising and swelling	Little or none: perhaps a late patch in Scarpa's space.	Great about hip.
Early shortening	½ to 1 inch.	1 to 2 or 3 inches.
Trochanter ...	Of normal shape.	Widened or comminuted.
Arc of rotation ...	Good.	Small or absent.
Crepitus	Obscure.	Easy, with extension.

But all these points taken together establish only a greater or less probability. Shortening, in particular, may lead astray, for it may be prevented from developing characteristically by some accidental circumstance (Fig. 276). In the course of a few days inflammatory softening, movements, manipulations may set the fragments free, and slight shortening quickly becomes considerable. Obviously, development of the higher degrees of shortening at once is most likely to occur in fractures which lie just beyond the capsule.

Impaction may occur in these fractures, and generally from direct violence. Near the head, the neck is driven into the head (rare), the posterior wall penetrating more deeply than the anterior (eversion) (Fig. 282), and almost always the inferior more than the superior (Fig. 277 is an exception) owing to rotation downwards of the head (shortening). Rarely these conditions are reversed (inversion).

In fractures near the base the neck is driven into the shaft and trochanter (page 838), the anterior wall cracks but does not penetrate; the posterior sinks deeply in beneath the trochanteric line (eversion). At the same time the head moves downwards (shortening, the amount varying much) in a circle round the base of the neck, and the lower wall of the neck passes deeply into the cancellous tissue of the top of the shaft (Figs. 280, 281). The special *signs* of these impacted fractures are more or less shortening, generally

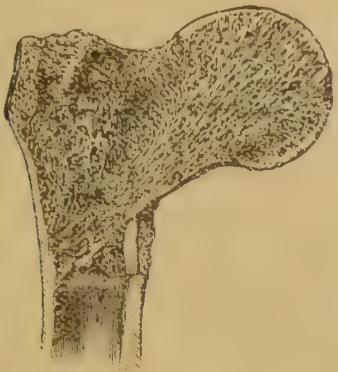
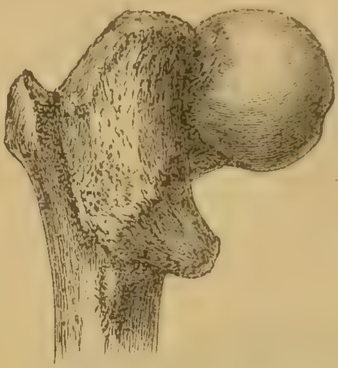


Fig. 278.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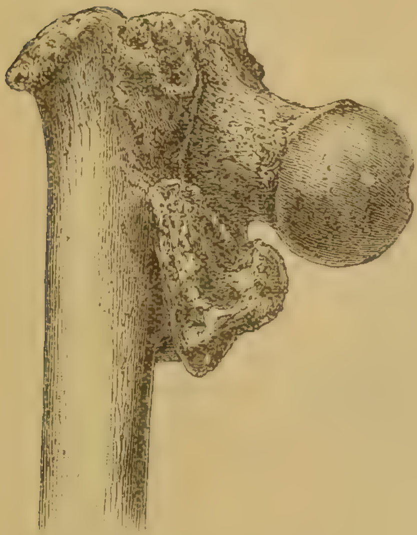
Fig. 280.

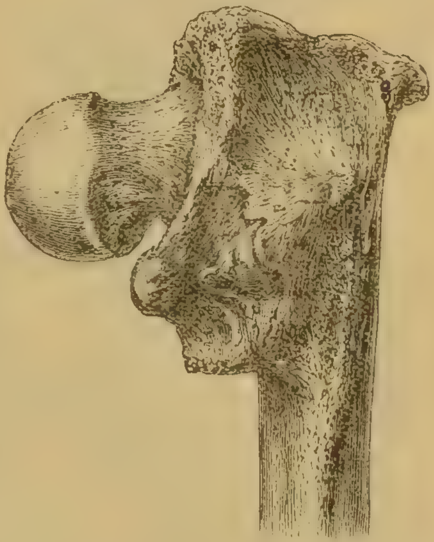
Fig. 281.

Figs. 278 and 279.—Union after an impacted Extracapsular Fracture, due to a Fall on the Hip: from a woman of 80. Fig. 278 shows that the base of the neck was driven deeply into the shaft and trochanter. Fig. 279 shows that much of the great trochanter, the intertrochanteric line and the small trochanter were split off, and have become united after displacement. In this case there was no eversion, and but slight shortening or flattening. (University College Museum, No. 247.)

Figs. 280 and 281.—Front and back Views of a right Femur repaired after an impacted extracapsular Fracture. The callus is in large amount and forms a collar round the lower attachment of the capsule. The head and neck form an acute angle with the shaft, and the shortening was probably 2 ins.; there was no eversion, and probably no flattening. (University College Museum, No. 248.)

less than in the average unimpacted case; eversion; full inversion impossible; retention of more or less power over the limb, even ability to walk, especially in intracapsular cases; but, often, the limb is perfectly helpless; the arc of rotation is good in both cases—far better in the impacted fracture of the base of the neck than in the unimpacted; crepitus is slight or absent. To distinguish between the impacted fractures of the base and of the narrow part of the neck the best points are the greater shortening, the greater depression, and the widening of the trochanter in the former. In impacted fractures of this region manipulations should be conducted with special care; for in the intracapsular fracture impaction offers the best chance of bony union.

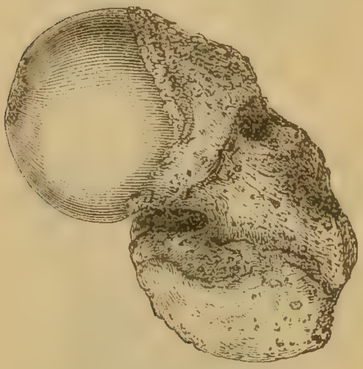

Fig. 282.—Bony Union after an intracapsular Fracture, the line of which is clear. The position of the trochanter, with its outer surface looking backwards, shows that eversion has been extreme; shortening was probably not more than half an inch. In this case the posterior part of the neck may have been impacted in the head. (Charing Cross Museum.)

Diagnosis.—A *bruise of the hip* may render the limb quite helpless, and cause it to be everted; it may, naturally, be a little shorter than its fellow: swelling may prevent examination of the trochanter; inversion may be resisted. Under these circumstances the patient should be kept in bed with the legs between sandbags; the symptoms will soon pass off if the injury has been a bruise only. A severe bruise of a joint, which is the seat of advanced *rheumatoid changes*, leading to shortening and grating, offers many difficulties. In *fracture of the acetabulum*, with entry of the femoral head into the pelvis, the shortening is slight, depression of a normal trochanter very great, and a swelling is felt per rectum; probably an extensive fracture of the hip-bone can be detected. *Dislocation of the femur on to the pubes* is spoken of as resembling fractures with eversion; the everted dorsal is somewhat more like; the ordinary dorsal dislocation is like fractures with inversion, inasmuch as inversion is present in it also. But in all other points—the characteristic position of each dislocation, the presence of the head where this position indicates, the limited mobility, the absence of crepitus, the mode and phenomena of reduction—the fractures are absolutely different from the dislocations.

Prognosis.—Danger to life in these injuries is considerable; especially in those due to slight violence in feeble old people, in whom, when confined to bed, hypostatic pneumonia, bed sores, delirium soon come on, and death quickly ensues. The more severe injuries kill by shock. As to function, fractures at the base of the neck practically always unite by bone: large masses of callus are found binding the neck and trochanteric fragments together as in a collar, sections showing little real blending between the neck and the bone around it (Figs. 278—281). As we pass towards the head the tendency to bony union gets less and internal to the middle it rarely occurs. The reasons assigned are, that in the absence of impaction, which is less common than at the base, it is difficult to fix the fragments together; that the head receives no arterial supply—the branches of the sciatic artery entering the neck behind being torn through, and the vessel in the ligamentum teres ending in a loop at the bone; that the subjects are often feeble and both fragments degenerate. If the fragments be held together by impaction or otherwise, bony union (Fig. 282) or short fibrous may occur; as a rule there is no union between the fragments, or merely a few long bands. Then the outer fragment mounts until the soft parts, especially the Y-ligament and the obturator internus, prevent it from going farther, and sling the body from the top of the femur; the neck wastes and disappears (Fig. 274), and the head in the acetabulum generally rests against the femur in the neighbourhood of the small trochanter. Rarely a mass of new bone is thrown out from the ilium as a kind of socket against which the trochanter can play (Fig. 283). Lameness results from these injuries, especially from the ununited intracapsular; but with a high boot and a stick even these patients may get about fairly. Sometimes no weight can be borne on the limb.

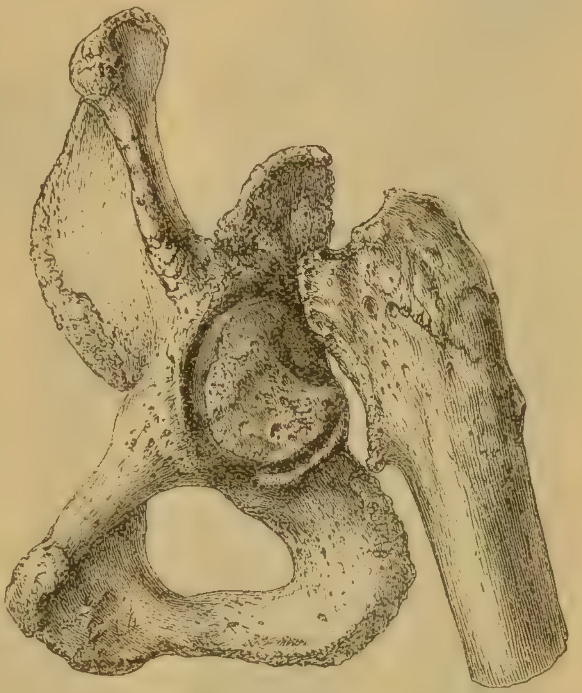

Fig. 283.—An old intracapsular Fracture. Most of the neck has been absorbed and the fragments seem shaped to fit. Above the acetabulum is a large projection of new bone, against which the lower fragment must have rested. (University College Museum, No. 235.)

3. **Fracture of the great trochanter: separation of its epiphysis.**—Both are uncommon. The latter occurs up to eighteen. Both injuries are due, as a rule, to direct violence, though a few cases seem to show that muscular action may tear off the process.

Signs.—Local swelling, pain, and tenderness: the fragment is felt movable and yielding; softer or harder crepitus if the torn surfaces can be approximated; or there may be little displacement, but the fissure between it and the shaft may be felt. There is no shortening: no interference with passive movements of the hip or with the weight-bearing power of the limb. Union will probably be bony, and, unless the fragment is pinned in position, should be facilitated by abduction of the limb. The results could hardly be worse than they have been in eleven cases of separation of the epiphysis collected by Hutchinson: six suppurated and five of them died.

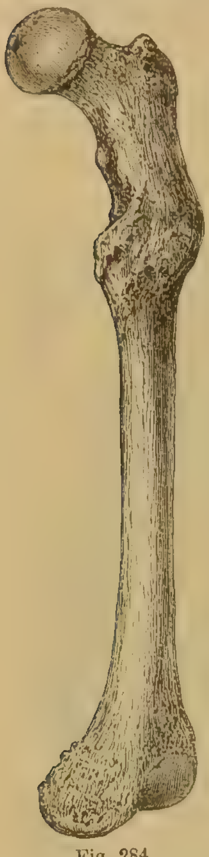
Fig. 284.

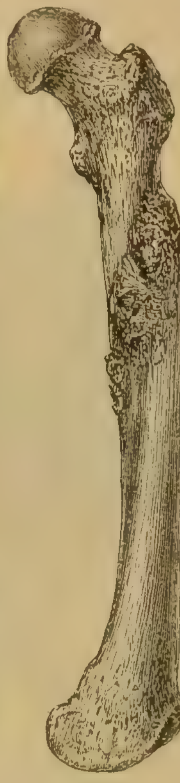
Fig. 285.

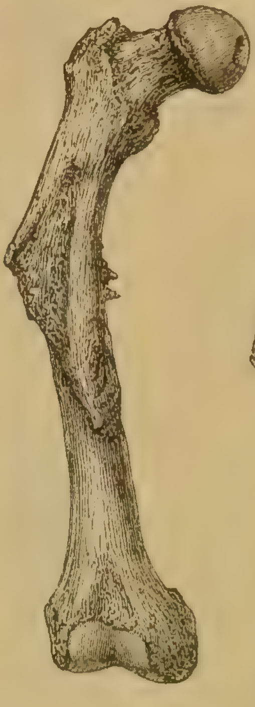
Fig. 286.

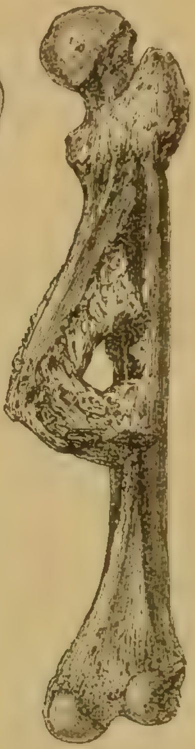
Fig. 287

Fig. 284.—A left Femur united after Fracture at the Junction of the upper and middle Thirds. It shows the usual deformity—slight flexion and abduction of the upper fragment, behind and internal to which is the lower fragment everted. (Charing Cross Museum, No. 385.)

Fig. 285.—Another Femur fractured about the same Spot, but downwards and backwards. The lower fragment lies in front of the upper and has prevented flexion. Abduction of the upper fragment is slight, but eversion of the lower is marked. (Charing Cross Museum, No. 386.)

Fig. 286.—A comminuted Fracture of the right Femur above the Middle. The upper fragment is moderately flexed and much abducted; there is no eversion of the lower. The deformity suggests that a Liston's long splint may have been responsible for it. (London School of Medicine for Women.)

Fig. 287.—A very oblique Fracture of the left Femur firmly united by strong Bridges of Callus in spite of marked separation of the Fragments. The upper is adducted, the lower is drawn up 2 ins. (University College Museum, No. 266.)

Fractures of the shaft of the femur.—These are common injuries throughout life; they are most numerous below ten, and diminish in frequency as age advances. They arise from direct indirect, and muscular violence. The line is sometimes transverse, but usually oblique.

Signs.—All the signs of fracture are generally present, but the size of the thigh may render their detection difficult. Except in transverse fractures, with little or no displacement, there is always shortening from the action of the hamstrings and quadriceps; when there is no shortening, angular deformity forwards and outwards, due to the hamstrings, is often present or is developed by any endeavour to raise the limb voluntarily. Eversion of the limb below the fracture is present in all but greenstick fractures, fractures without displacement, and a few cases in which violence or accident in loose cases has inverted the foot.

In the upper third the upper fragment generally points forwards and rather outwards owing to the almost unopposed contraction of the ilio-psoas and glutei (Figs. 284, 285); probably the lower fragment, which has dropped back and is then drawn up, often pushes the upper fragment forwards, and keeps it in this position (Figs. 286, 287), or in some other direction (Fig. 288). On the other hand, if the line of fracture be unusual, and the lower fragment lies in front of the upper, the above deformity is prevented (Fig. 285).

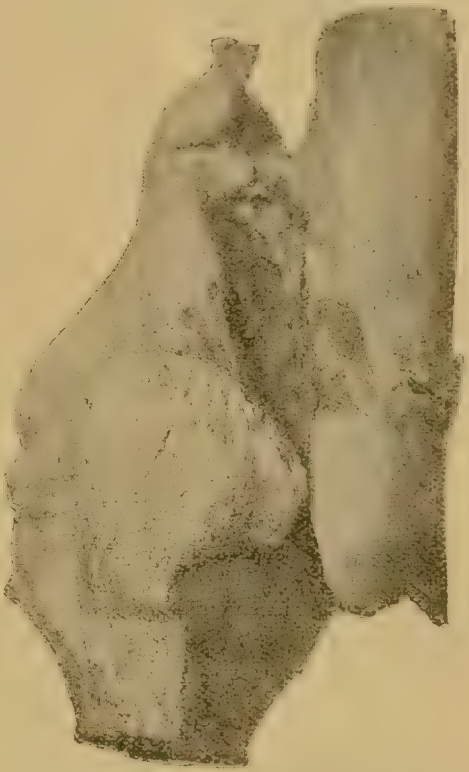

Fig. 288.—A compound supra-condyloid Fracture —oblique, downwards and inwards. The shaft projected 2 ins. through the skin, internal to the patella, and its end has undergone necrosis. (St. Bartholomew's Hospital Museum, No. 756.) (H. Marsh.)

Fractures of the lower end of the femur.—Here we have supra-condyloid fractures, transverse or oblique, from behind downwards and forwards (Fig. 288); T- and Y-shaped fractures (Figs. 211, 289), often comminuted; separation of the lower epiphysis; fractures of the condyles.

In all the above, unless interlocking prevent it, shortening will occur, and unopposed flexion of the lower fragment upon the tibia by the gastrocnemius. The latter displacement should always be looked for: if it be missed, either there will be no union, or the patient will recover with a straight leg but a flexed knee (Fig. 290). There is a tendency also for the lower fragment to drop backwards away from the upper. The rough end of the flexed

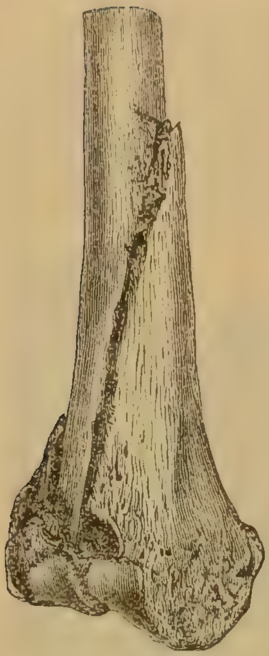

Fig. 289.—An irregular Y-Fracture of the lower End of the Femur, one arm of the Y being prolonged upwards on the inner side to the top of the lower third. The pointed shaft was impacted in the outer condyle, of which it depressed the anterior end. The inner condyle was pressed a little upwards before union occurred. (University College Museum, No. 283.)

fragment is towards the vessels and nerves of the popliteal space, and this spot is one where vascular complications are specially frequent. The T- and Y-fractures are due to direct violence or to penetration of the lower end of the shaft into the lower end, splitting it up (Fig. 211); firm impaction of the pointed shaft into the lower end may persist (Fig. 289).

Separation of the lower epiphysis (Fig. 291) has generally been due to forcible twisting and angular movements, as when the limb is caught between the spokes of a wheel; direct violence will also push it off. Generally the epiphysis is carried forwards (Fig. 290), and the lower end of the shaft forces its way through the periosteum into the popliteal space, when the vessels and nerves will be drawn tensely over it; other displacements, causing great deformity, occur. The results have been bad (Hutchinson). Of twenty-eight simple cases sixteen were well reduced and recovered; in twelve reduction was imperfect—six sloughed or suppurated, four requiring amputation, one excision of the knee, and one resection of the end of the shaft. Among the compound cases amputation and deaths from pyæmia were still more numerous: five cases in which the protruding diaphysis was cut off did well. Doubtless there are many pure and mixed cases in which there is little or no dislocation, which do well and are never recorded.

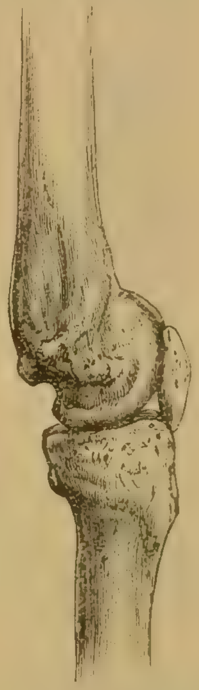

Fig. 290.—Separation of the lower Epiphysis of the Femur, Flexion of the lower Fragment, Displacement backwards of the upper Fragment, followed by bony union. From a girl of 17. Three years earlier her limb had been caught between the spokes of a wheel; after this injury the knee remained "painful and swollen, and she had a halt in walking." After three years extensive suppuration occurred and amputation was done by Liston.

Fractures of the condyles are rare, and generally occur from blows or falls on the bent knee. Such a fragment as is represented in Fig. 292 would probably be felt to move in certain positions of the joint, with crepitus and the development of genu valgum. Sometimes the condyles are comminuted; again, only a small bit is chipped off and forms a loose body.

This would very probably be missed until the blood was absorbed from the joint and use of it had begun.

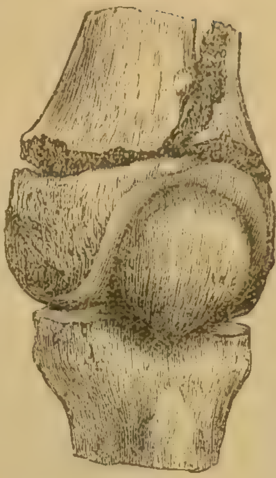

Fig. 291.—A mixed Separation and Fracture of the lower Epiphysis of the Femur starting within. The fissure runs more than half-way across the bone in the plane of the epiphysial disc, then mounts rapidly and obliquely to the outer surface. Acute suppuration resulted, and the parts shown were excised. (Charing Cross Museum, No. 391.)

Treatment of fractures of the femur.—In feeble old people the effects of an accident to the lower limb, especially about the hip, and the consequent confinement to bed, must always be borne in mind. In a fracture of the narrow part of the *neck*, in which bony union cannot be looked for, there will not be much hesitation, except on the score of pain, in getting the patient up early should this seem desirable; but it may be necessary to do so in other cases—to sacrifice the ideal treatment of the fracture to saving the life of the patient. Whilst in bed a good diet should be allowed, and frequent small doses of some stimulant are probably useful. Any change of position that can be permitted should be arranged for. The greatest care must be taken in these cases to avoid moisture, and either mechanical or chemical irritation of the back. Although a rubber layer beneath the drawsheet is undesirable, in many cases, it cannot be avoided if an air or water pillow be used, and one is often desirable in these cases; then a sufficient layer of porous material—*e.g.* tow—should cover it to absorb all moisture, and allow of some ventilation and evaporation. A slipper bed-pan should be used. Washing and rubbing of the back must be regularly performed, the patient being raised by a sufficient number of assistants to do it easily, or being rolled on to the sound side by one nurse taking the pelvis and shoulder whilst another sees that the limb turns comfortably in a circle, having the opposite trochanter as centre. A dusting powder containing boric acid ʒij to ℥j is helpful. Fracture boards should always be laid across the bed-frame beneath a thin mattress, to reduce to a minimum the sinking of the heavy pelvis carrying the upper fragment with it. All

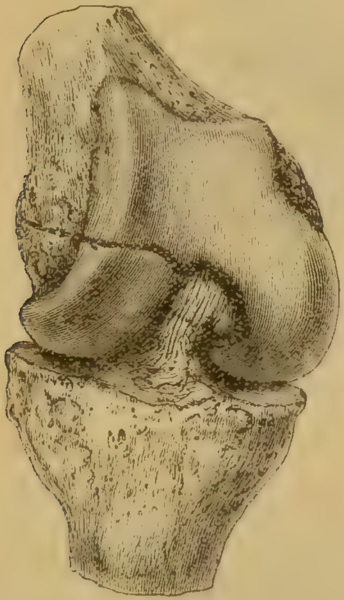

Fig. 292.—A right Femur, of which rather more than the whole tibial Surface of the external Condyle has been separated by fracture. (University College Museum, No. 283a.)

mechanical arrangements for raising patients from the bed for nursing purposes permit this sinking to a marked and painful degree.

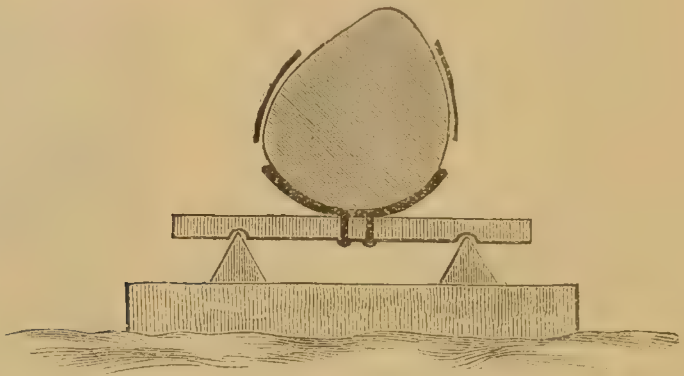

Fig. 293.—Volkmann's sliding Foot-rest.

In most *fractures of the thigh* we have to deal with longitudinal and rotatory displacement (shortening and eversion), and often with angular; there is often a tendency for the lower fragment to drop back and separate from the upper. Shortening is specially met by extension, the remaining deformity by the "setting" of the fracture, and by the application round it of splints, etc., to press the fragments into place, and prevent their displacement. Extension to be efficient should be constant—either elastic or by weight; the latter is generally the better. In all cases, friction should be reduced to a minimum, to diminish the drag upon the limb. As in some fractures it is desirable to raise the lower fragment to the

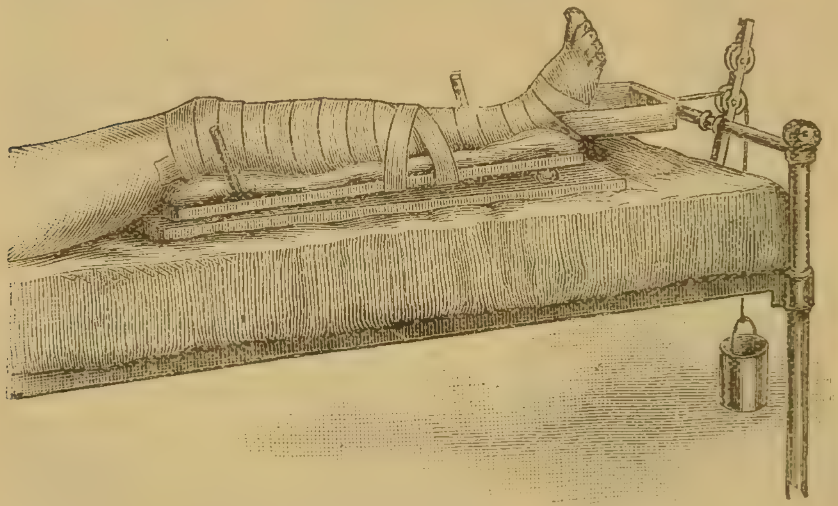

Fig. 294.—A running Cradle for Extension.

level of the upper, extension may have to be made in all lines between the vertical and horizontal. As to rotatory deformity, it must be borne in mind that, when at rest, the thigh is permitted by the ilio-trochanteric band to roll out, until the feet point up and out, and form with the vertical an angle of 45° to 50°. The degree of rotation of the sound limb should be noted, and the injured one should be "set" at the corresponding angle. The structure of many splints is such that they are calculated to fix the foot in the vertical position; but no splint will prevent rotation out of the upper fragment unless the hip be flexed.

To carry out the above indications we have many methods and splints.

(1) *Simple horizontal extension.*—A long wide strip of stout strapping is carried on each side of the leg and thigh up to the fracture, or even a little above it if the injury is low, so as to form a stirrup 4 to 5 in. long below the sole. Narrow ($\frac{3}{4}$ in.) strapping coiled *obliquely* round the limb from below up helps to fix this, as also does a roller firmly applied over all. The stirrup is held open by a "spanner" of wood, a little wider than the bi-malleolar measurement, with a hole through the centre; through this a stout cord is passed and knotted towards the sole, the other end for the weight being passed over the pulley. To diminish friction, the distal portion of the limb may be laid in a Hodgen's wooden cradle, or some corresponding trough, and the transverse bars beneath this

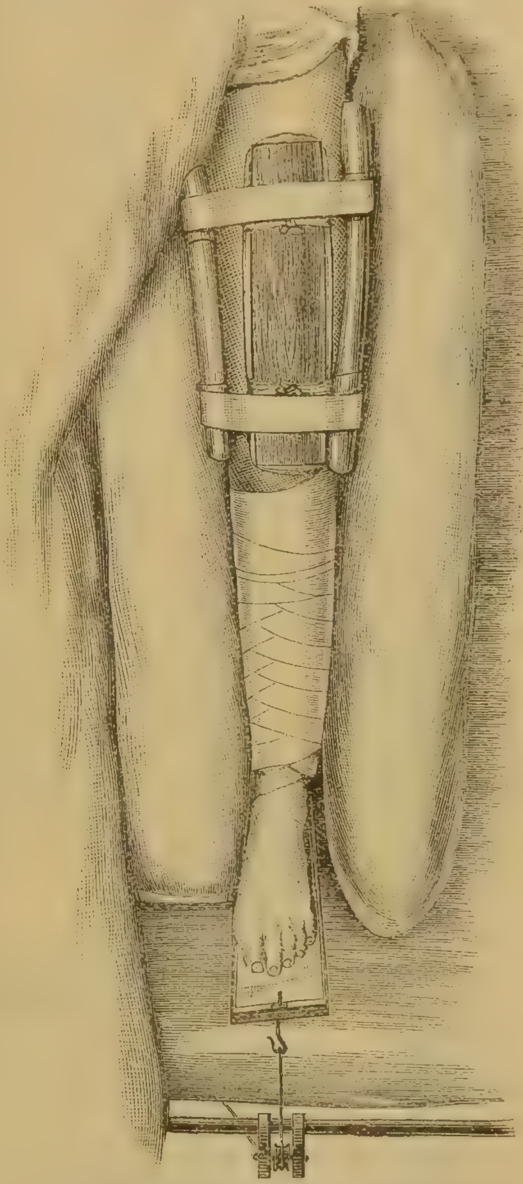

Fig. 295.—Treatment of a fractured Thigh by short side Splints and Weight Extension between Sandbags.

are laid upon a couple of wooden "knife-edges" raised above the level of the clothes by two stout cross bars, to the upper surface of which the knife edges are screwed (Fig. 293); or the limb may be laid on a padded board (cradle) running by means of four castors on another board (tray) with raised edges (Fig. 294). The lower castors should raise the foot-end a little, and the limb is kept on the cradle by a few wooden pegs in lateral holes, to which the foot is fixed in proper position. A comparatively light weight becomes effective when one or other of these appliances is used. Counter-extension is made by raising the foot of the bed. When there is no shortening, extension may be used as a comfortable means of fixing the limb: in addition, a sheet may be thrown over the limb, and in it, upon each side, a long heavy sand-bag is rolled up till it lies close to the limb placed as may be desired; pins through the sheet now fix the bags so that they keep the sheet tense. A limb may thus be very securely held; but friction is so great that there is no real "extension" acting.

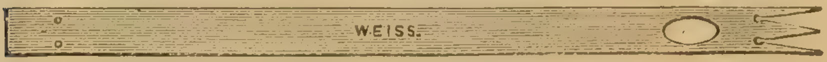

Fig. 296.—Liston's long Splint.

Extension plus a broad well-padded band buckled firmly round the trochanters is a good way of treating fractures of the neck.

Extension plus short side splints (Fig. 295) is an excellent method of treating many fractures of the middle portion of the shaft. Extension and counter-extension are made to reduce the shortening, and maintained while four short kettleholder splints padded with a layer of boric lint are laid round the limb, and fixed by two or three webbing straps and buckles drawn firmly round them. The upper and lower fragments should now rotate together, but a little extra support is given to the outer side of the foot by the stirrup.

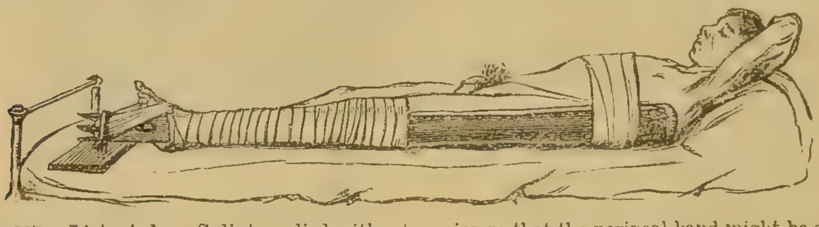

Fig. 297.—Liston's long Splint applied without a spica so that the perineal band might be seen

(2) *Liston's long splint* maintains horizontal extension, and fixes the fragments and all the joints concerned. The splint is $\frac{1}{3}$ to $\frac{1}{2}$ in. deal, 3 to $4\frac{1}{2}$ in. wide, and long enough to reach from below the axilla to 5 or 6 in. beyond the foot; it has two notches at its lower, two holes through its upper end (Fig. 296). The limb below the fracture is firmly attached to the splint by four figure-of-8 turns round the splint and dorsum of the foot, and through the terminal notches,

after which the roller is carried up to the fracture. Extension is now made upon leg and splint, and, when a satisfactory result has been obtained, the counter-extending perineal band is tightened. The upper end of the splint is fixed to the chest by a 12-in. bandage fixed to it, and carried backwards to the other side and round the trunk two or three times. Lastly, a hip spica is generally applied. This splint now connects the two fragments solidly, and they are held apart by the perineal band acting on the top of the splint, and the turns through the notches acting on the bottom (Fig. 297).

It is usual to split the end of the bandage, tie the ends through the holes at the top of the splint, and carry the bandage down between the splint and the pad. The latter is left long enough to pass round beneath the sole and cover the dorsum of the foot, a position given to it by the first turn of the bandage; the foot is thus well protected from pressure. The perineal band may be made of a silk handkerchief sewn round a firm sausage-like roll of tow, 9 in. long, placed in its centre. The roll may be made of thick rubber tubing, tied at each end to a tape; this is both more cleanly and comfortable. Before beginning to bandage, the band is placed with its centre opposite the tuber, and its ends are drawn through the holes in the upper end of the splint, and tied loosely. With the long splint some combine a large plaster splint, moulded to the front of the upper part of the thigh and groin; it is

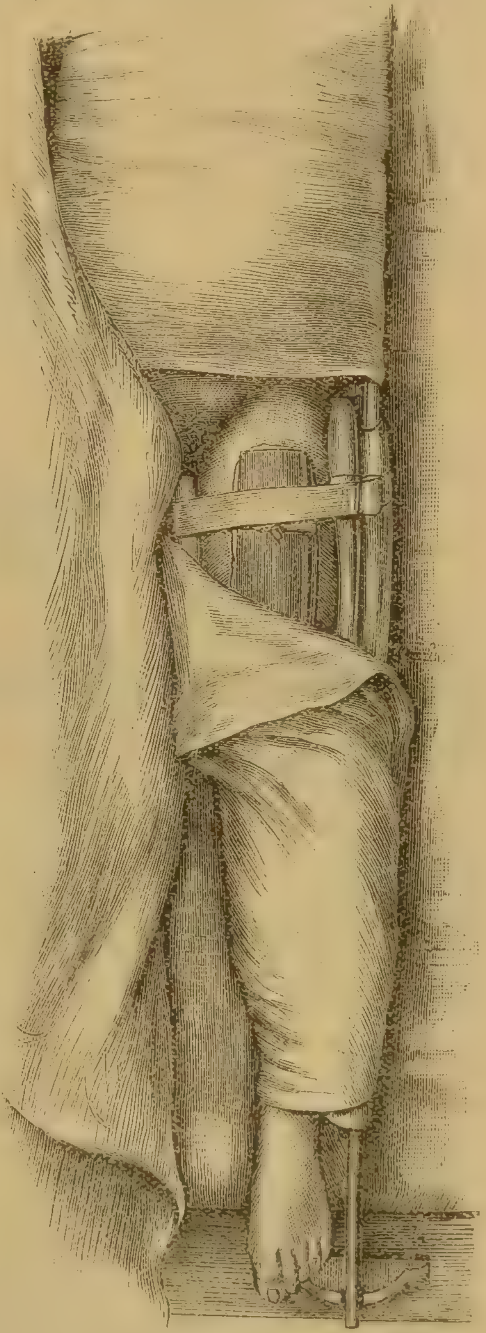

Fig. 298.—Treatment of a fractured Thigh by short side Splints, with the long splint and Scotch sheet.

thought to repress the upper fragment. There are many objections to the long splint. It is uncomfortable, and tends to ride forwards and inwards; this is met to some extent by dropping the lower end into a vertical slot, and by using a broad strap instead of a body bandage. The dorsum of the foot and, still more, the perineal region may get sore; the latter always gets very foul. The tendency of the bandage is to draw both fragments out to the splint, thus altering the shape of the femur (Fig. 286), and the perineal band and spica pull the upper fragment forwards and outwards. If the foot is firmly bandaged, and the splint kept vertical, rotatory deformity should result.

(3) *The long splint* and *Scotch sheet* are an improvement on the above. A plain board of the above dimensions is used simply to fix the joints. Four short splints are buckled round the thigh, and the limb is slung to the splint by the "Scotch sheet," as shown in Fig. 298. A broad bandage binds the splint to the body. Weight extension from the limb can be added. The splint is kept in position by being dropped into a vertical slot. The only advantage of this apparatus over extension and short splints is that the movements of an unruly patient are, to some extent, controlled; and discomfort is in proportion to the control exercised.

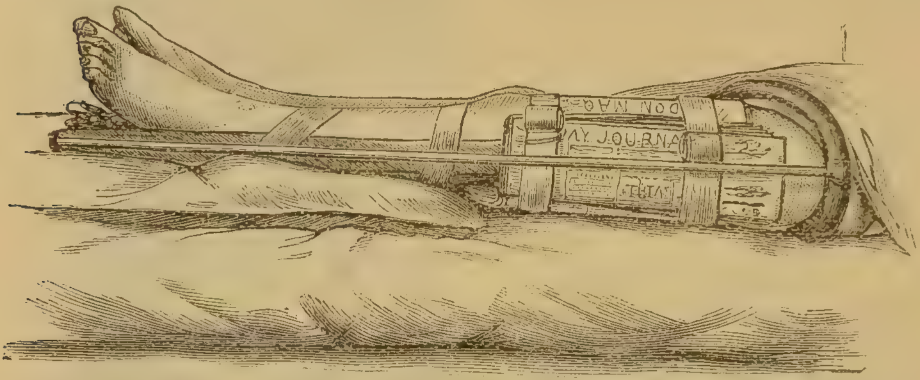

Fig. 299.—Treatment of a fractured Thigh by short side Splints of newspaper and extension between the ring and foot bar of a Thomas's knee-splint. A firm bandage should be carried round both splint and limb from above the heel to the ring.

(4) *Thomas's knee-splint.*—Extension straps are adjusted. Short side splints, which are very comfortable when made of newspapers, are buckled round the fracture, shortening and rotatory deformity being first corrected. Then the splint is put on, pushed up firmly against the tuber, and the shoulder strap is buckled. The extension straps are now drawn tight and wound round the foot-piece or a special bar; a firm bandage round splint and limb finishes. When everything is satisfactory a silicate bandage may be applied, rendering the apparatus "permanent." In this the limb is most securely fixed, and the whole can be moved about freely (Fig. 299). The part may be left lying or slung in a Salter's cradle, and this should be

done if there have been any tendency to tilting forwards of the upper fragment. Elastic extension is easily arranged.

When angular deformity is present, horizontal extension and repressive splints have a certain element of force about them. It is a common practice to raise the lower fragment into the line of the upper, and to maintain extension in this line.

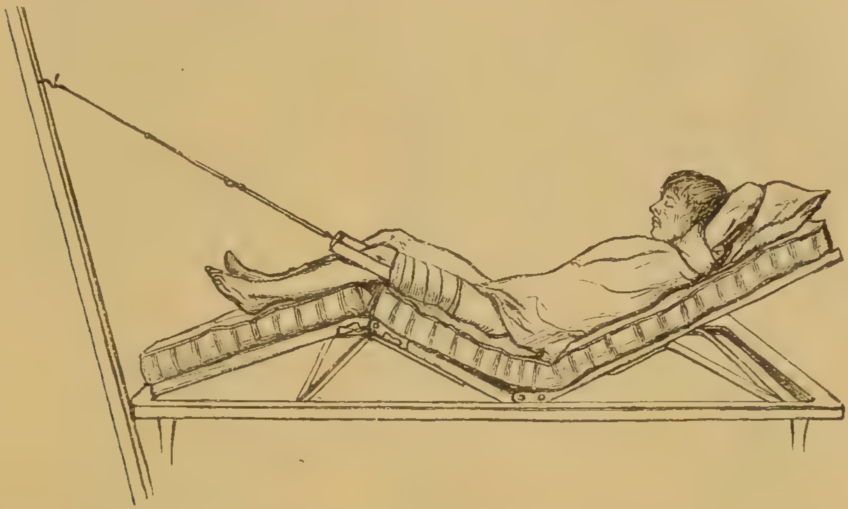

Fig. 300.—Treatment of a fractured Thigh by the double inclined Plane and Extension in the line of the Thigh.

(5) The *double inclined plane* for one or both limbs was the earliest form of apparatus having this object. The angle between the two planes can be varied as required. A frame such as that shown in Fig. 300 is easily and quickly made, the double inclined plane is that part on which the limb rests; but the third leaf allows of variation in the position of the trunk. In some cases of fracture of the neck in the old and feeble it may be sufficient to use the sound limb as a splint and to fix the other to it at knee and ankle, with wool pads intervening; the usual firm band should be buckled round the trochanters. A better plan, and no more trouble, is to maintain extension as shown. The leg may be fixed to the lower plane and the angle be so arranged that the body-weight maintains slight extension.

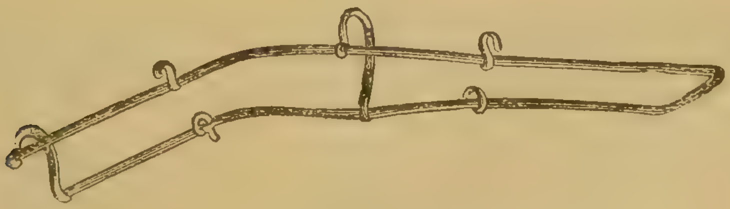

Fig. 301.—Hodgen's Splint.

The principle of the double inclined plane is carried out in a far less cumbersome way and with many advantages in *Hodgen's splint* shown in Fig. 301. The limb with extension stirrup and spanner attached is laid between the bars, and the cord is tied to the bottom of the splint so that the upper ends may lie comfortably below the anterior spine and groin. A few strips of flannel are passed behind the limb and fastened tautly with pins to the bar on each side. The slings are now fixed to the frame, and by drawing down the wooden stop on the cord the limb is slightly raised (Fig. 302). The tension necessary in each band can now be perceived, and they are rapidly put on so as to overlap well and form a firm cradle. By altering the tension of certain bands and changing the angle of traction, the lower fragment is raised to the upper, any prominence of which should disappear. When there is no angular deformity the two fragments rise together. Counter extension is obtained by the body-weight, the foot of the bed being raised. Excessive rotation out of the leg is opposed by the extension stirrup, friction of the flannel bands against the limbs, and slight flexion at the knee. In this splint the limb swings comfortably; fractures with and without angular deformity can be equally well treated in it, even the supracondyloid fracture with flexion of this fragment, if it be possible to overcome the deformity by splint only; the nursing is easy, for the patient soon learns to lift himself by his sound leg and arms up and away from the extension cord, or he can be lifted in this way, so as

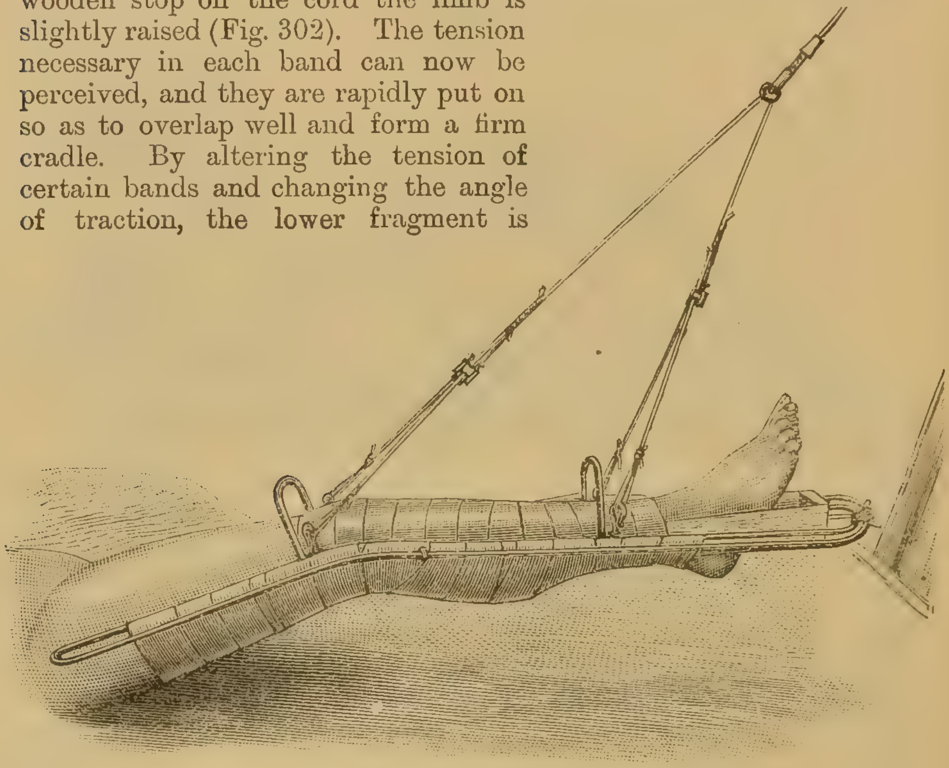

Fig. 302.—Hodgen's Splint applied. The splint used has a second bar to adapt it to Bloxam's cradle. The bandage round the limb only secures the extension stirrup, which is tied to the end of the splint. The limb is comfortably slung between the bars by the strips of bandage. The extension pole is shown, passing the foot of the bed, which is raised; the extension cords, from the splint, are attached to the pole high up.

to maintain constant, comfortable extension. It is an admirable splint for compound fractures, as it can be included in an antiseptic dressing, the bands being sterilised and changed one by one at the dressings. It appears to be clearly the best all-round splint for treatment of fractures of the thigh; though Thomas's knee-splint has the advantage that, in cases for which it is suitable, it gives greater freedom, the patient being able to get up, and even to walk.

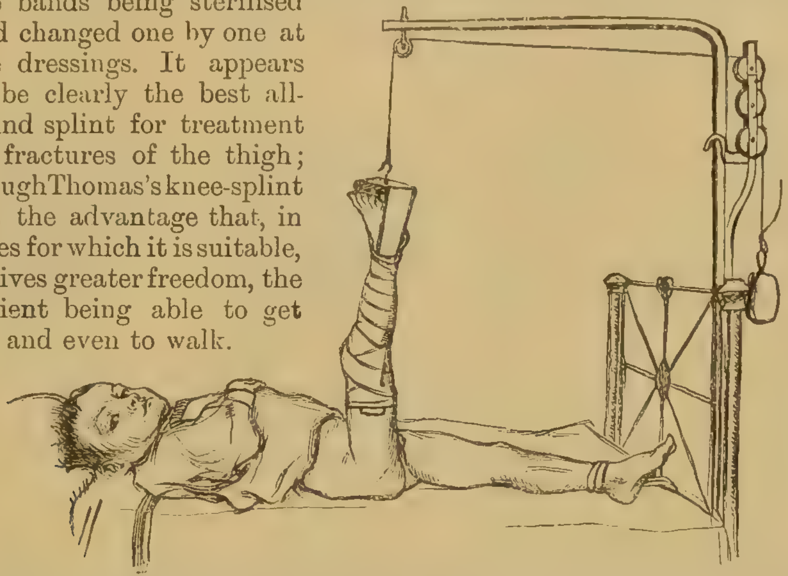

Fig. 303.—Bryant's vertical Suspension Method—weight-extension being used. The left leg is loosely fixed by a clove-hitch round the ankle and the shoulders are fixed by a webbing brace with arm-holes, buckled round the chest and tied to the sides of the bed-frame.

(6) *Nathan Smith's anterior splint* is a narrow wire framework, moulded to the front of the limb from groin to ankle with the knee moderately bent; two or three layers of boric lint form a pad. The limb is attached to the splint by a few bands and then slung to a hook above the limb so that little or no extension is obtained. Further support is given by more bands or by a continuous bandage. It is hard to imagine a case for which Hodgen's modification would not be better.

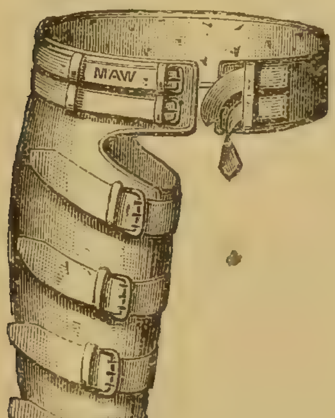

Fig. 304.—Leather Hip Splint, lined with chamois and provided with buckles.

(7) *Bryant's vertical suspension* is the most satisfactory mode of treating children. Fig. 303 explains most of the points. Bryant recommends that both limbs should be flexed to 90° at the hips with an extension stirrup on each, and that the stirrups should be tied together over a bar at such a height that the sacrum barely touches the bed. At the Paddington Green Children's Hospital only the injured leg is raised, and it is kept suspended by a weight sufficient, with slight assistance from the nurse's hand under the sacrum, to raise the pelvis from the bed: extension is thus maintained even when the child

is moved. The sound leg is fixed by a clove-hitch round the ankle to the bed-frame and generally the shoulders are fixed by a webbing band which leaves the arms free. A long back splint and short side splints of Gooch's material give additional security, but are not always necessary.

Fractures of the neck in old people which are not expected to heal may be treated by the pelvic band and either horizontal extension, the double inclined plane and extension in the line of the thigh, or Hodgen's splint. If all goes well, they may lie thus for three to four weeks, having the hip massaged till tenderness and pain on movement have gone. Then, with a leather hip-splint (Fig. 304), a starch spica getting a good grip upon the thigh, or a special splint with a joint that can be locked straight opposite the hip, with a high heel on the sound side and a pair of crutches, the patient will begin to get about. After two to three months some weight can be borne on the broken limb; the high heel should be removed and the heel on the injured side raised. Gradually a stick will replace the crutches. If circumstances require it, the move out of bed must be made at an earlier date.

All these splints except the last render sitting impossible; so, too, does a single Thomas's hip-splint, which can be applied with a water-glass bandage, and enables a patient to move about early. This seems to be the chief point—some exercise to ward off hypostasis.

Fractures of the neck in which bony union is expected are preferably treated with a pelvic band and Hodgen's splint, or with simple extension; eight to twelve weeks must be allowed for union; but generally after six weeks the patient may get up with the hip fixed by a Thomas's splint or starched bandage.

Impacted fractures of the neck should be carefully handled and comfortably placed till union has had time to occur. Great deformity might lead one, in fractures apparently of the base, to break down the impaction and treat as usual. Southam has recently carried out this treatment with good result.

Fractures of the shaft in adults are best treated with a Thomas's knee-splint, a Hodgen's splint, or with short splints and horizontal extension. In *young children* vertical suspension, in older children Thomas's splint, are preferable. Union takes five to six weeks in children, seven to ten in adults.

Supra-condyloid fractures.—If there be no angular displacement a Thomas's splint or horizontal extension between sandbags will give a good result. When the characteristic flexion of the lower fragment is present, the double inclined plane with extension in the line of the thigh occurs as suitable; some have apparently found it so, but the grip on the lower fragment may be insufficient to prevent marked shortening, and the lower fragment tends to drop back from the upper. Hodgen's splint is more likely to succeed. Should these methods fail, we must, on Bryant's suggestion, divide the tendo Achillis, and then treat by horizontal extension, or a

Hodgen, allowing the foot to drop. Treves speaks highly of this practice, but he uses the long splint after the tenotomy with heavy extension from it—sometimes as much as 40 lbs.

Fractures of the condyles and other fractures within the knee are best treated with a Thomas's knee-splint.

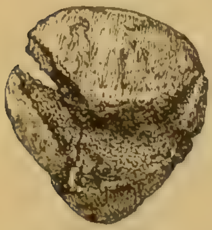

Fig. 305.

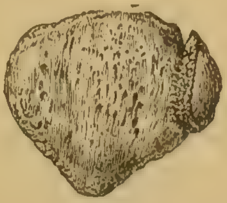

Fig. 306.

Fig. 305.—A Patella repaired after a "starred" Fracture which broke it into four pieces; all have united by bone after slight displacement, but union is weak everywhere, and at one spot, absent (University College Museum, No. 290.)

Fig. 306.—A Patella from the outer edge of which a Fragment has been broken by a longitudinal Cleft. Bony union has occurred. (University College Museum, No. 291.)

Fracture of the patella. — Fractures of the patella form 1·4 per cent. of all fractures. They are very rare under twenty, commonest up to fifty, but occur even in old age. They are far more frequent in men than in women (13 to 1). These fractures may be due to direct violence, when the bone may be comminuted, or "starred" (Fig. 305), or split longitudinally down the middle (rare), or a bit may be chipped from a lateral border (Fig. 306); the skin may be damaged even to laceration by the fall or blow, rendering the injury compound. Far more often they are caused by muscular action, bending and finally cracking the bone across the convex femoral condyles. When a person misses a step, or stumbles and makes a strong effort to pull himself up, the knee being already bent in the fall, the patella rests on the condyles by a comparatively narrow transverse surface; and the piece above this surface is a longer or shorter lever upon which the quadriceps acts. The resulting fracture is generally transverse, but may be markedly oblique. The fragments vary much in size, being sometimes equal, again very unequal; now the upper, now the lower is the smaller. In cases from direct violence the surrounding fibrous tissue (periosteum, fascia, and aponeuroses of the vasti muscles) are not much torn, the fragments do not separate much, and union by bone, though not very strong, is usual (Fig. 305). But muscular action tears the fibrous coverings of the bone, and the

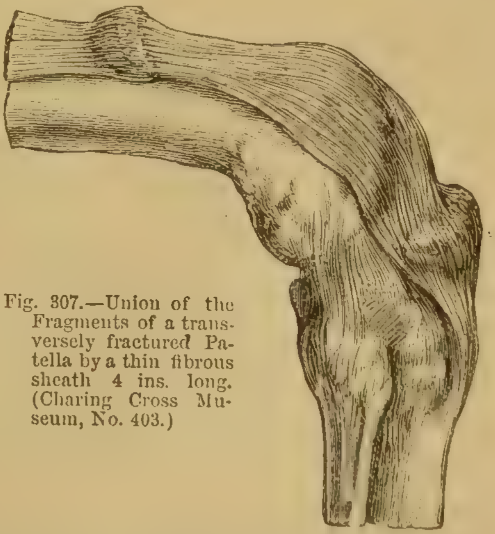

Fig. 307.—Union of the Fragments of a transversely fractured Patella by a thin fibrous sheath 4 ins. long. (Charing Cross Museum, No. 403.)

rent may extend into the fascia lata and the aponeuroses of the vasti when the force is great and continues acting after the bone has yielded, and whilst the knee is bending; the gap between the fragments varies accordingly from a narrow cleft to a gap of some inches, and union is almost always by a shorter or longer, thicker or thinner fibrous bond (Fig. 307). A patella may be broken twice and even thrice: usually one of the fragments breaks, not the fibrous bond, though this does happen and the skin has torn with it.

Symptoms.—A loud snap is often heard as the bone gives. Rising from the ground after the fall is a matter of difficulty. A groove indicating the gap between the fragments is often seen. But the joint quickly swells, as a rule, and the outline of the synovial membrane becomes clear; bloody fluid oozes through the fracture and forms a prominent rounded swelling in front of the patella: fluctuation between the intra- and extra-articular collections may be obtained; through this, and especially towards the margin, a transverse or otherwise directed fissure can be felt, and the fragments can generally be seized, approximated and rubbed together. Any more or less transverse gap is widened by bending the knee or by endeavouring to straighten it; as a rule there is no power of extension, but with starred and longitudinal fractures, and even with narrow transverse ones, accompanied by little tearing of fasciæ, the patient may be able to lift his heel from the bed. Standing is possible, for the knee extends beyond the right line and locks; but walking straight forwards is generally impossible—the patient finds that he must either go backwards dragging the extended leg after him, or go forwards, everting the damaged limb completely and bringing it forward with his adductors. On removing the fluid covering it, if necessary, the kind of fracture and the size and separation of the fragments are noted. In the transverse variety, the torn surface of both fragments is more or less everted, especially that of the lower.

A good deal of synovial irritation results, but not sufficient to excite even warmth of the skin. More or less of the blood clots —sometimes early, sometimes late. The fluid part is diluted by effusion from the synovial membrane, and as tension increases the fragments are forced apart. The fluid is then absorbed; the clot may become organised, and form adhesions. Clot adheres closely to the fractured surfaces, filling every chink, and becomes replaced by callus. The apparent causes of fibrous union in transverse fracture are: the separation of the fragments is usually considerable owing to muscular action, effusion and tilting on the condyles; the fibrous tissue on the surface of the patella is generally torn up in a flap connected with one or other fragment, and this flap falls into the gap, thickens and adheres quickly to the raw surface; lastly, immobility cannot be secured. But there must be something else, for transverse gaps are often very narrow, the bony union of starred and longitudinal fractures is often feeble (Figs. 305 and 306), and even after wiring, if narrow gaps occur from imperfect fitting, they may fail to fill with bone.

Treatment.—The usual plan is to elevate the leg to 45° with a back splint and to raise the back to 45° on a bed-rest, and thus throw the rectus out of action. Ice is packed round the knee to limit effusion, or pressure is made over a mass of cotton wool round the joint. After twenty-four hours, or sooner, if swelling has ceased, the fluid is drawn off with an aspirator with all cleanliness; the fragments are then gently pressed together, and an endeavour is made to retain them in position by U-straps, one above the upper, one below the lower fragment, drawn towards one another; by figure-of-8 turns of narrow bandage above and below the fragments taking a hold upon a couple of hooks or screws on the back or upon notches cut in the side of a back-splint; or by Sir A. Cooper's plan of laying a couple of strips of calico bandage on each side of the patella and of tying closely round the limb over these above and below the fragments a couple of wet strips of the same kind; the two longitudinal strips are then tied on each side of the patella so as to approximate the fragments. The circular constriction of the limb in Cooper's plan is objectionable, and all straps and bandages used to drag the fragments together must be watched lest they cut the skin. Some apply a plaster bandage over wool before swelling occurs, or after aspiration; the patient goes about on crutches after a week. A fixed splint is applied also after four to five weeks of the earlier-mentioned methods of treatment: it is worn for two to three months, then the patient is allowed to walk with a leather knee-cap (Fig. 308), which permits but little flexion, and this is worn for four months more.

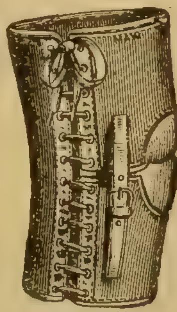
Fig. 308.—A Leather Knee-Cap.

In different hands the average result of the above methods is about the same—short fibrous union in most cases. The knee is stiff on removal of the apparatus, the limb often œdematous. Motion may return quickly, or slowly, and in either case the bond of union may stretch. No attempt forcibly to hasten the return of movement should be made; it will come in time. If massage were practised function would probably be earlier restored. The limb in the majority of cases is a very useful one, and for ordinary walking does well enough; but the thigh is smaller than its fellow and more or less weakness comes out in running, weight-carrying, climbing, descending stairs, etc. The limb is not so reliable as its fellow. Extension of the knee and swinging the leg forwards are the feeble movements. Though, as a rule, feebleness is proportionate to length of bond, it is not always so; some long unions have done excellent work, while some short ones have been useless.

Malgaigne's hooks (Fig. 309) have been used freely by some surgeons since antisepsis has enabled them to avoid the inflammatory troubles which occasionally started in the punctures. Aspiration should be performed. Then a tenotomy knife prepares the way down to the bone for each point, and the hooks are inserted, each taking a firm hold superficial to the synovial membrane; the hooks are then screwed together till the fragments lock, and are left in six weeks; the punctures are dressed with iodoform powder or paste (made with 1 in 20 carbolic solution) and a little gauze and wool over all. The union is generally fibrous, and the after treatment must be as above to prevent stretching.

In view of the length of time occupied by the above treatment, of the constant permanent inferiority of the injured limb leading with undue frequency to fracture of the other patella, and of the occasional very unsatisfactory results, Lister, in 1883, proposed that recent fractures from muscular action should be treated by wiring, with the proviso that no one should do the operation who could not keep the wound sweet. The operation is by no means easy in a simple transverse injury, and it becomes more difficult with oblique or irregular surfaces to obtain good coaptation. A flap, base in or out, should always be raised large enough to cover the whole deep wound; the joint is cleaned of all fluid and clot: clot is scraped from the broken surfaces and hanging tissues are cut away; if there is any difficulty in obtaining accurate contact two sutures (Fig. 310) should be passed, one on each side of the middle of the fracture. The bone should be bared where a hole is to be drilled and two artery forceps nipped on to the fibrous tissue reflected as guides to the spot. The drilling can be done with an ordinary bradawl, but is more likely to be accurate if done with less effort by a dental engine. A Thomas's hollow needle or an aspirator needle of suitable size is useful for passing along the channel and withdrawing that end of the wire which must be passed from the broken surface; the sutures are of $\frac{1}{16}$-inch silver wire; they are drawn tight, twisted, cut short, and hammered down; any capsular rent is sewn up, the wound closed, the limb fixed in a Thomas's knee-splint, and a large compressive dressing applied. The splint may be removed on the third day. As soon as the wound is healed, the patient should be drilled daily into moving the limb. Bony union is obtained, and the patient is walking in five to six weeks.

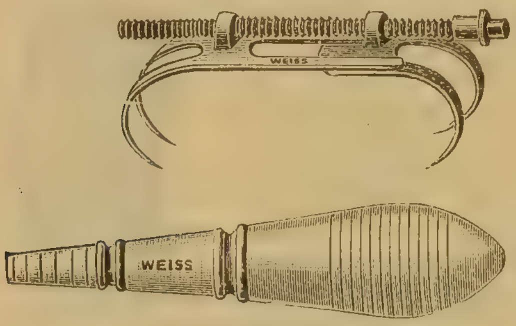

Fig. 309.—Malgaigne's Hooks.

The results of the operation vary greatly with the surgeon. Many cases have suppurated, great dangers have been run, knees have become ankylosed, or amputation has been required; even where all has gone well, prolonged pain and stiffness have resulted; re-fracture, too, has occurred from imperfect union, the skin sometimes tearing. But strong bony union has generally resulted, the limb soon becoming as good as ever. Each surgeon must decide for himself whether and when he will undertake in recent cases an operation having such possibilities. It is a different matter in old cases of disabled limbs. Some attempt must be made to relieve by resecting the fibrous bond and bringing the fragments together after sufficiently free division of retaining tissues. But for septic failures, the results of these difficult operations have been good.

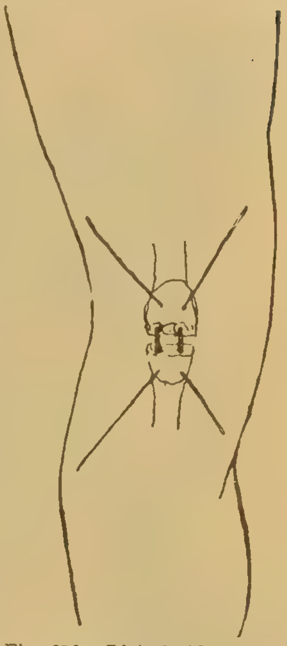

Fig. 310.—Lister's Method of wiring the Patella. Wires *in situ*, ready for drawing tight. (A. E. Barker.)

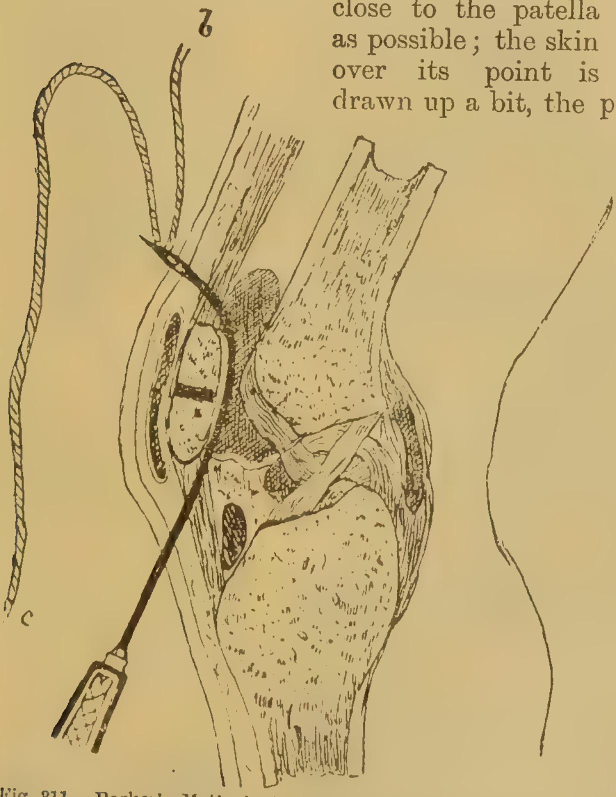

Fig. 311.—Barker's Method of suturing the Patella: the needle passed beneath the patella and threaded with silk (*b*, *c*). (A. E. Barker.)

To avoid an open operation many ingenious plans have been suggested, of which the following are the best: Scrupulous cleanliness is as essential in them as in the open plan. A. E. Barker passes a narrow scalpel into the knee through the lig. patellæ, and in withdrawing it cuts firmly on to the lower edge of the patella; a well-curved pedicle needle is introduced and pushed through the quadriceps tendon as close to the patella as possible; the skin over its point is drawn up a bit, the point is cut upon and a knife passed into the joint between the needle and the bone, laying the bone bare as it is withdrawn; the needle is threaded with wire or the strongest sterilised silk (Fig. 311) and withdrawn; the needle is now passed between the skin and patella from puncture to puncture, the upper end of silk threaded (Fig. 312) and withdrawn; the two fragments are rubbed together till clot and soft tissues are removed, and then the ends of

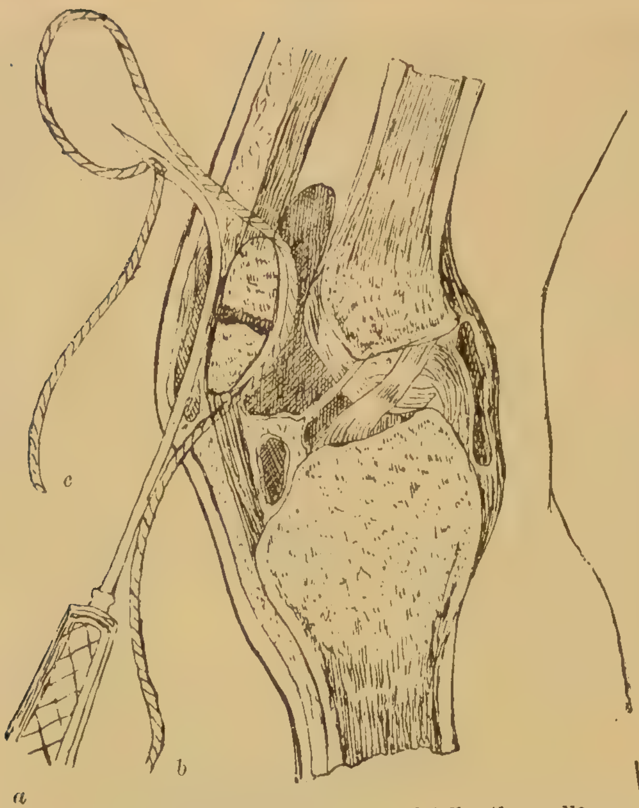

Fig. 312.—Barker's Method of suturing the Patella : the needle (*a*) passed between the patella and the skin, and threaded with the upper end of the silk (*b*, *c*), which lies behind the patella. (A. E. Barker.)

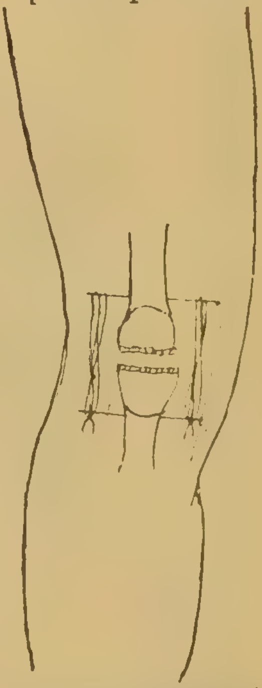

Fig. 313. — Mayo Robson's Method of approximating the fragments after transverse fracture of the patella. (A. E. Barker.)

silk are drawn tight, firmly knotted and cut short. The patients walk in five weeks, apparently with bony union. G. E. Twynam "tyres" the patella, *i.e.* throws a ring of wire or silk round it in the attached fibrous tissues : in some comminuted cases with separation it may be useful ; without a special needle it is a somewhat difficult suture to pass. Mayo Robson passes a bonnet-pin through the quadriceps tendon and another through the ligamentum patellæ, each touching bone : the projecting ends are wired together (Fig. 313). Robson believes that he gets bony union. In three cases I obtained close fibrous union.

Compound fractures should be treated by disinfection and suture of some kind, unless other bones are comminuted or soft part seriously injured, when excision or amputation must be considered.

Fractures of the leg bones. Separation of the upper epiphysis of the tibia.—The upper epiphysis of the tibia is of large area, but not deep ; it sends a triangular process downwards in front, which ends in the tubercle. Ossification begins just after birth as a rule, and ends at 22. It is much less frequently displaced than the lower epiphysis of the femur, owing to the attachment of the semi-membranosus and int. lateral ligament partly to it, partly to the shaft. Hutchinson collected ten cases, ages 12 to 16; cause, violent wrenches; displacement either slight and forwards or altogether absent. The knee joint is sure to suffer in so severe an injury to one of its constituent structures.

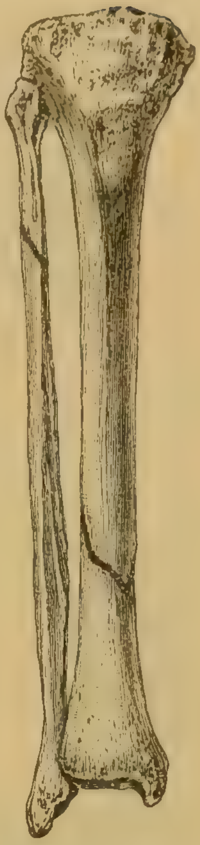
Fig. 314.—The ordinary oblique Fracture of the Tibia from behind downwards, forwards, and inwards at the junction of the lower and middle thirds (en bec de flûte). Also a fracture, correspondingly oblique, near the neck of the fibula. (Charing Cross Museum, No. 405.)

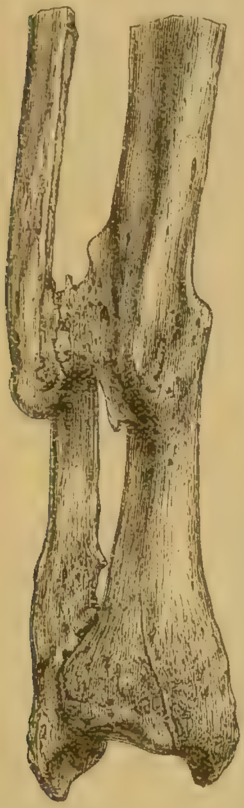
Fig. 315.—Fracture of both Bones of the Leg at the Junction of the middle and lower Thirds. The lines of fracture ran from before downwards and backwards. The lower fragments were drawn up in front and to the inner side of the upper fragments. Strong union has occurred, callus bridging the interosseous space. The fibula sustained a second fracture lower down which united without displacement. The tibia shows a fissure which starts on the articular surface near the malleolus and ascends on the posterior aspect. It presents little sign of healing. This injury resulted almost certainly from direct violence. (Charing Cross Museum, No. 410.)

The quadriceps has occasionally pulled off the tubercle of the tibia, which may grow from a separate centre. The accident has generally resulted from jumping or vaulting the "horse."

Treatment. — Reduction of the displacement and fixation of the limb. Hutchinson quotes a case of arrested development from Volkmann; Stimson figures another, which he attributes to Bryant. The tubercle could be easily fixed by a pin or two driven through the skin.

Fractures of the tibia and fibula form 10·2 per cent. of all fractures; they are, perhaps, the commonest fractures. Fractures of the tibia alone form 1·8 per cent., and of the fibula alone 2·0 per cent. They occur at all periods of life, but are most common during the period of greatest activity and danger.

Indirect violence is a commoner cause than direct, but both are frequent. Several cases are known of separation of the head of the fibula (an epiphysis up to 24) by violent contraction of the biceps, due to an effort to prevent falling. Other fractures from muscular action must be very rare.

Direct violence causes fracture, often with comminution and wound over the sharp tibial edge, at the point struck; if both bones are broken they yield at about the same level.

Fractures of the upper end of the tibia, of the tibia alone, and of the fibula above its lower third, are generally due to direct force. Indirect violence, especially falls upon the feet, gives rise to the common oblique fracture—"en bec de flûte"—of the tibia downwards, forwards, and inwards at its weakest spot, the junction of middle and lower thirds. The fibula generally yields immediately after the tibia, either at the same level or at the neck (Fig. 314). To falls upon the feet are due also spiral fractures, starting on the inferior articular surface, and winding up the shaft (Figs. 207 and 210). The sharp point of the upper fragment of the tibia close beneath the skin, as it usually is, is often driven through the skin by an attempt to stand, or by dropping of the foot in improper lifting.

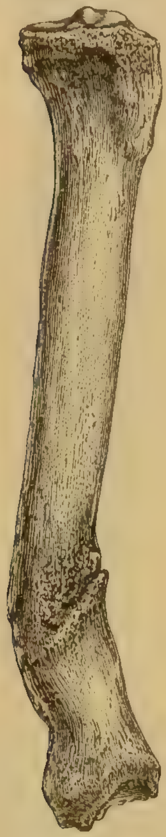
Fig. 316.—A Tibia which has been broken at the usual Situation, and the Line of Fracture running from before downwards and backwards. The lower fragment has been displaced forwards, so as to produce an angle, salient backwards, with the upper fragment. This is very unusual. (Charing Cross Museum, No. 410.)

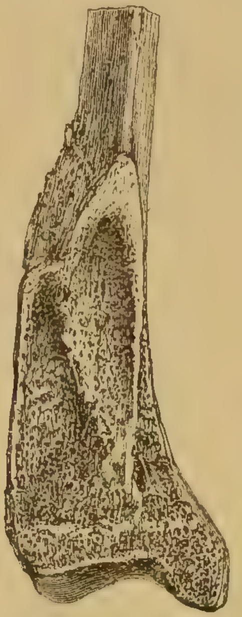
Fig. 317.—The lower End of a right Tibia repaired after Fracture from without downwards and inwards, and Impaction of the Shaft into the lower Fragment. The lower fragment has rotated so that its articular surface has been carried inwards. Firm union has occurred and the medullary canal has been almost restored. (University College Museum, No. 315.)

Signs.—All the signs of fracture are present or easily elicited in fractures of both bones as a rule; fracture of the tibia, owing to its subcutaneous surface, generally causes no difficulty. But fracture of the shaft of the fibula may be very obscure; localised tenderness and pain, constantly referred to a spot, not struck, when the leg bones are pressed together at a distance from it, are very suspicious. Separation of the head of the fibula is easily recognised.

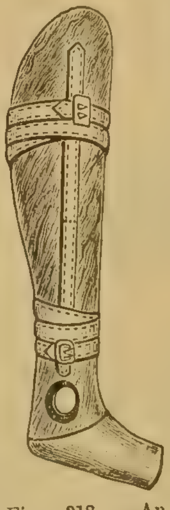
Fig. 318. — An outside Cline's Splint, with webbing bands and buckles coiled round it.

When only one bone is broken the other acts as a splint to it, and deformity is slight or absent. When both suffer, the calf muscles cause shortening, with angular deformity if the fragments interlock; but usually the surfaces are oblique, and the lower fragment glides up and back and out behind the upper (Fig. 314). In high fractures the quadriceps often seems to cause prominence of the upper fragment, but it is impossible to eliminate the effect of dropping backwards of the lower fragment. Other displacements are illustrated in Figs. 315, 316, 317.

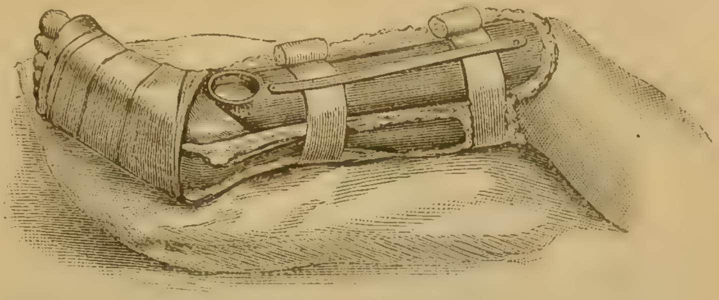

Fig. 319.—Cline's Splints applied with webbing Bands. A bandage has been begun round the foot, and may be carried up the leg, but is not necessary. The limb has been laid on its outer side upon a pillow.

Treatment.—When there is displacement, setting is best accomplished with the knee fully bent to relax the calf muscles. Extension is made from the foot; counter-extension from the lower third of the thigh, which is firmly grasped by an assistant. Rotatory deformity is best avoided by comparing the injured with the sound limb, and endeavouring to copy the form of the latter.

A couple of lateral plaster splints, shaped like Cline's splints (Fig. 318), may be moulded to the limb at once and fixed with a bandage; this can be cut up in front, should much swelling appear, and the splint be reapplied with a fresh bandage.

Or for the first few days movable splints may be used. None are better than Cline's for fractures at or below the middle. Two outside Cline's are more secure than an outside and inside, unless it is desired to produce marked inversion of the foot. The splints are lined with boric lint, and buckled on as shown in Fig. 319. The limb may be left free or slung; at first the latter is better. If there be much tendency to dropping back of the lower fragments, Cline's splints do not prevent it. Then an Arnold's or a MacIntyre's splint may be used. One or other of these should be employed for fractures above the middle, unless plaster is used.

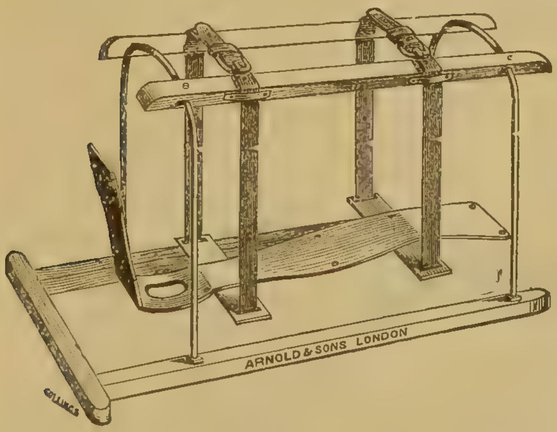

Fig. 320.—Arnold's Splint (wooden side pieces not shown) and Cradle.

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Arnold's (or Neville's) *splint* is shown applied in Fig. 321. It consists of an iron foot piece and back splint (Fig 320) with two wooden lateral splints. The limb is placed on the padded back splint and secured to its foot piece and lower part; any extension required is now made, counter-extension being provided at the thigh; whilst this is maintained, a bandage is carried from the fracture up to the top of the splint. The side pieces are now buckled on, and the splint is slung in a special cradle by means of straps passed through lateral projections from the back splint. It should be placed so that the toes point up and out in the position of rest; or the foot may be fixed in this position across the foot piece, and the splint hung straight.

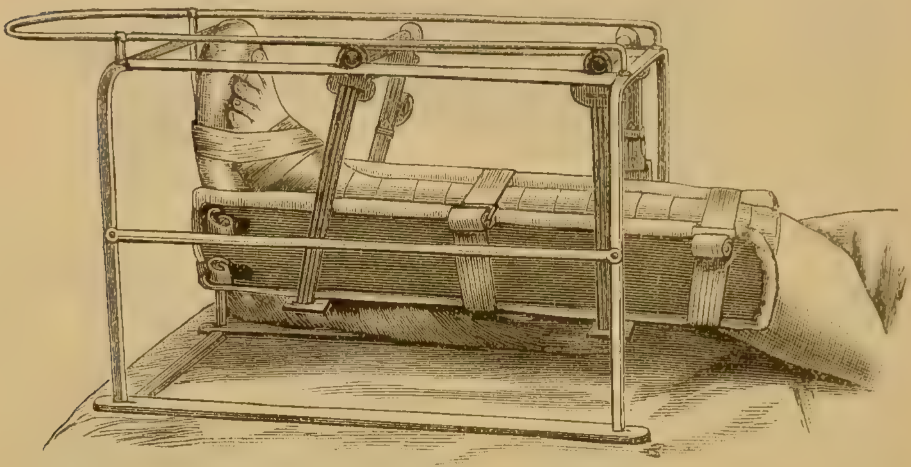


Fig. 321.—Arnold's Splint applied and slung on Bloxam's Cradle. The eversion of the foot shown is quite insufficient.

MacIntyre's splint (Fig. 322) is a posterior gutter splint of iron, with a screw arrangement for varying the angle between thigh and leg pieces, and with a vertical foot piece, sliding in lateral slots for purposes of extension, and fixed at any spot by a screw nut. The foot piece is left loose and pushed well up; the back splint is padded. A sock having a tape attached on the sole aspect is drawn on to the foot, and the limb is placed on the splint. The tape is carried over the apex of the foot piece and fastened round a button on the lower surface; the foot is thus held up without much pressure on the heel. The leg above the fracture and thigh is fixed to the splint; the foot is raised to the proper level, the angle of the splint regulated to remove all angular deformity; the toes are properly pointed across the foot piece (the

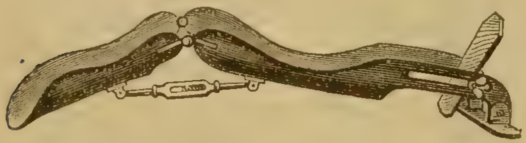


Fig. 322.—MacIntyre's Splint.

other leg must be similarly flexed to judge) and bandaged or strapped firmly to it. Extension is now made as shown in Fig. 323, and whilst it is maintained the foot piece is fixed by the nut, and the bandage is carried on up to the fracture. The lower end of the splint may rest on a block, or it may be slung in Salter's cradle (Fig. 324).

Weight extension on a pillow between sandbags is often a useful way of treating these fractures.

The difficulties in managing an oblique fracture of both bones with marked shortening are many and great. Lane has recently expressed the opinion that a man has lost 75 per cent. of his working value on recovery from such an injury; and as a result of this view he advises that all such cases should be cut down upon and screwed together in good position.

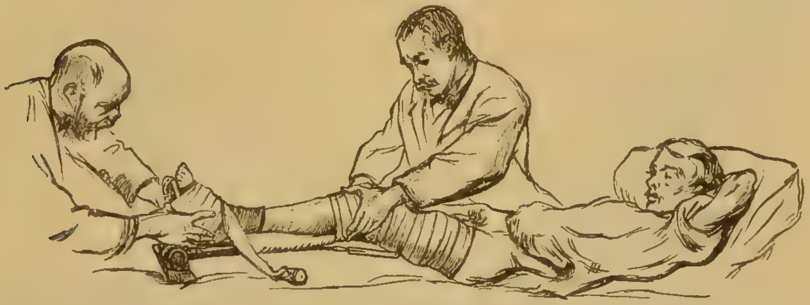


Fig. 323.—The Application of MacIntyre's Splint. The thigh has been fixed by bandage to the splint; the hands of an assistant grip the limb and splint about the knee. The foot in its sock is fixed by tape to the button on the foot piece and by bandage. Extension is being made by drawing down the foot piece (and foot) in its slots. The screw nut would then be tightened and the bandage carried up to the fracture.

In fractures of the tibia or fibula only, without displacement, no splints are needed; it is sufficient even at first to place the limb in a soft cushion, and tie this up round it. Massage should be undertaken at once. Union of the fibula takes six weeks, of the tibia seven, or of both bones, eight weeks.

In *compound fractures* the dressing should be surrounded by moulded plaster splints. Injuries to vessels are specially frequent complications at the upper part of the leg; and the peroneal and ant. tibial nerves are specially liable to injury in fracture of the neck of the fibula and of the tibia respectively.

Fractures of the lower ends of the tibia and fibula.—Direct violence may, of course, act here, but the typical fractures of the part result from twists of the foot of various kinds, jumps from a height or from a carriage in motion, etc., bringing indirect violence to bear upon the malleoli. Though the mechanism differs, the anatomical results of inversion (supination) and of eversion (pronation) are much the same.

Perhaps the commonest accident here (1) is a *fracture of the fibula* 3 inches above its lower tip, due to excessive pressure of the astragalus on the malleolus, and recognised by swelling, tenderness, and pain—the latter increased by pressing the fibula high up towards the tibia—and perhaps slight mobility and crepitus in moving the foot.

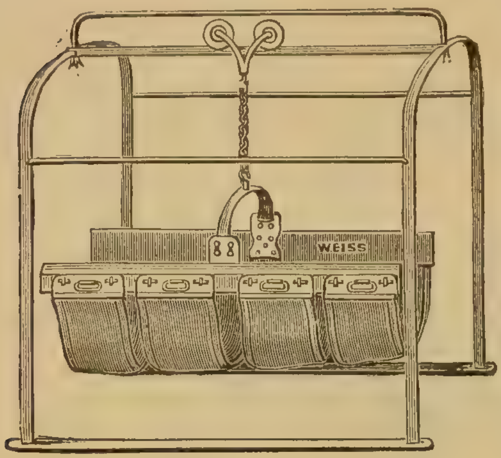
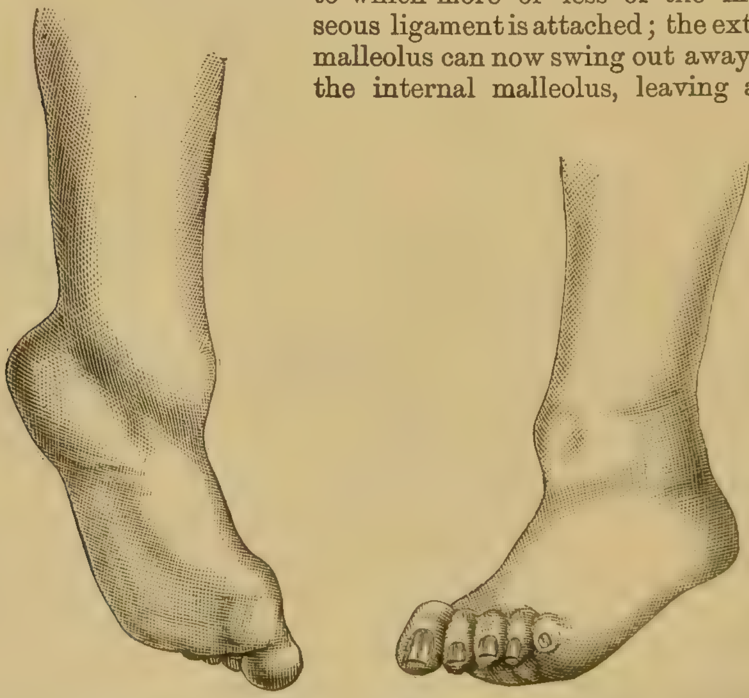


Fig. 324.—Salter's Cradle.

(2) Next comes *Pott's fracture* (Figs. 325, 326, 329), due to greater but similar violence. The fibula is broken 3 inches up, the internal malleolus is torn off, or the internal lateral ligament of the ankle is torn through. The foot is markedly everted, and often carried backwards as well; there is a hollow over the fracture of the fibula, soon obliterated by hæmorrhage; the internal malleolus or its sharp stump presses strongly against the skin, and not infrequently cuts its way through. (*See also* page 1018,)

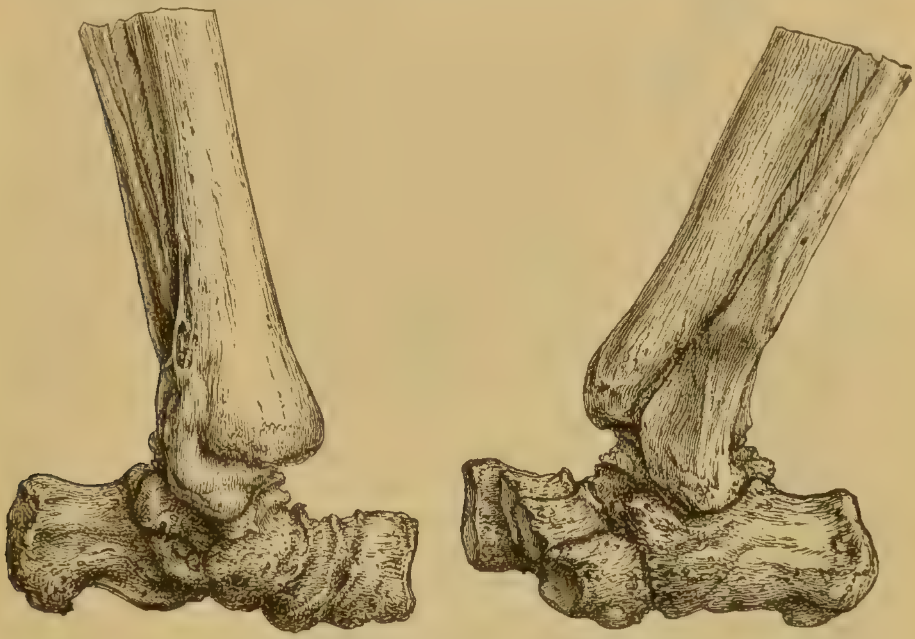
(3) Rarely the fibula, in breaking as above, carries with it the fibular margin of the tibial articular surface and a fragment of bone, to which more or less of the interosseous ligament is attached; the external malleolus can now swing out away from the internal malleolus, leaving a gap



Figs. 325 and 326.—From Photographs of a recent case of Fracture of the internal Malleolus and of the Fibula 2 to 3 inches up; with displacement backwards and outwards of the foot.

into which the astragalus, outer edge first, can ascend (*Dupuytren's fracture*). The foot is greatly everted, but the chief feature is the increase in width at the level of the malleoli. (*See also* page 1018.)

(4) In catching the heel, alighting from a height or from a carriage in motion, the leg-bones tend to move on after the foot has been arrested, and a dislocation backwards of the foot is likely to occur; sometimes the astragalus and os calcis carry both malleoli with them (Fig. 330); the heel projects markedly; the lower end of the tibia forms a prominence in front; the tendo Achillis is stretched, and the astragalus is depressed below the often fractured posterior edge of the tibial surface, hence the toes are pointed downwards. The displaced malleoli can be felt.



Figs. 327 and 328.—Views, from the inner and outer Sides, of the Bones from an old case of Pott's Fracture. The internal malleolus has been broken and carried backwards; so has the external malleolus, the fibula having yielded one inch up. The astragalus has been displaced backwards, and the foot bones have rotated outwards beneath it, flattening the arch. A chronic inflammation has obliterated the ankle and some other joints. (Charing Cross Museum, No. 416.)

The lower epiphysis of the tibia ossifies in the second year and joins at 18 or 19. Separation of it with fracture of the fibula, or, very exceptionally, with separation of the lower epiphysis of the fibula, almost replaces Pott's fracture in the young, and is caused by similar violence. The epiphysis and foot are generally dislocated backwards or back and out, while the diaphysis tends to protrude, or protrudes in front. Reduction has not as a rule been difficult; and in compound cases the result has been good. Bryant thinks that sprains in the young may often involve loosening of this epiphysis and that getting about too early on the foot may result in inflammation, impaired growth, and shortening of the limb.

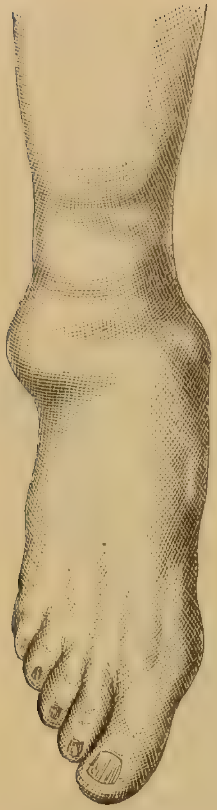


Fig. 329.—From a Photograph of a recent Case of Fracture of the internal Malleolus, and of the Fibula 1½ inch up, with Displacement inwards of the Foot. Early swelling about the ankle is shown.

The lower epiphysis of the fibula is rarely separated. In one case suppurative epiphysitis occurred. Arrest of growth with curvature of the tibia is a conceivable result.

Treatment of fractures of the lower ends of the leg-bones.—In all cases in which dislocation exists it must be corrected with the knee fully flexed to relax the tendo Achillis. Some are very difficult to overcome, and the attempt to do so has been abandoned after section of the tendo Achillis had failed. But, with antiseptics to help us, failure should no longer be the case. Most of these deformities from unreduced fracture dislocations have been operated upon after many months, and with good result: they would be easier to deal with after the early extravasation has subsided. No directions can be laid down for such operations; success in each case depends on a correct appreciation of the state of matters coupled with a good deal of ingenuity and operative skill.

Cases without deformity, or in which deformity has been reduced, should be put up with one or other of the splints used for fractures of the leg, or in a large mass of cotton wool firmly bandaged. In cases of marked eversion some use Dupuytren's splint (Fig. 331). This, which is a "long splint" in miniature, should be padded with increasing thickness from the knee down to the ankle; here the pad should be thick enough to permit inversion of the foot without contact with the splint. The splint should be fixed below the knee first; then, the deformity being undone, the foot should be bandaged to the splint as shown. Some first bind the foot to the splint and use the latter as a lever to undo the deformity — brute force instead of skill.

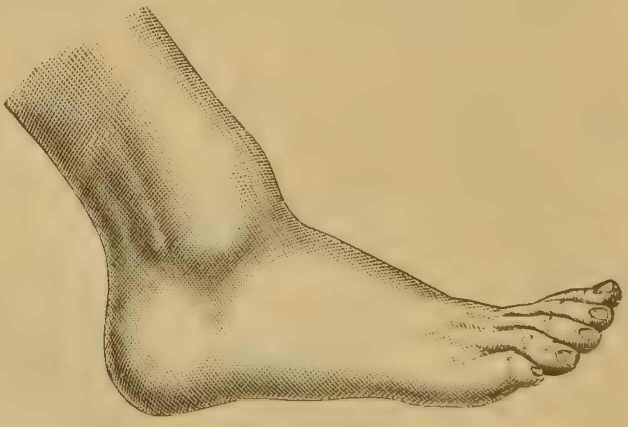


Fig. 330.—Fracture of the two Malleoli with Displacement backwards of the Foot, causing a prominence above the ankle in front, projection of the heel, and extension of the foot. (From a cast in the University College Museum, No. 324.)

Fractures of the bones of the foot.—Fractures of bones of the foot constitute 2·66 per cent. of all fractures. Almost all arise from direct violence (run over, falling objects, caught between spokes), or great indirect violence (falls and jumps from a height); rarely one of these bones is broken by force which appears quite inadequate. By crushing force any or all the bones may be pulverised; but the os calcis and the astragalus suffer much more frequently than the other tarsal bones, and often alone; of the metatarsal bone the fifth and first seem to be broken more frequently than the central three; commonly two or more are broken.

Os calcis.—This bone is more often broken than any other tarsal bone, usually by falls and jumps on the heels (Fig. 209). Fracture from this cause occurs not uncommonly on both sides, and more or less comminution often results, especially of the inner side upon which the astragalus chiefly rests. Sometimes the result of the accident is a transverse vertical fracture between the tuberosity and the posterior astragaloid surface. In this case the fragment is more or less drawn up by the calf muscles, and if the fragment is a long one the arch of the foot falls. In the first case—of extensive comminution—flattening of the antero-posterior arch and more or less eversion of the foot are common; the malleoli are nearer to the ground, and the os calcis feels widened; the heel may appear lengthened or shortened, if a piece is displaced upwards. Swelling soon comes on, more or less limited to the region of the os calcis. Crepitus is sometimes very obscure.

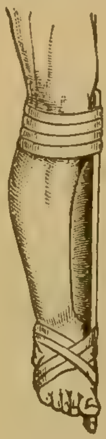


Fig. 331.—Dupuytren's Splint.

Forcible inversion occasionally causes the tense middle band of the external lateral ligament of the ankle to tear off a scale of bone which can be felt at the tender, painful, and swollen spot. Very rarely the same movement forces off the sustentaculum tali, and then, the middle band of the internal lateral ligament being detached, the os calcis rotates out and the foot becomes everted. A good many cases are on record of avulsion of the tuberosity (ossifying in the tenth, and joining in the sixteenth year) before or after union, chiefly after; displacement may be slight or great, 4½ in. in one case, in which it persisted, yet the patient is said to have recovered with perfect use of the limb.

Treatment.—If there be no displacement, elevation and ice and then a plaster shoe. If there be displacement, flex the knee fully, place the foot carefully at 90° to the leg, with the sole so that it will touch the ground all along its outer border; endeavour to restore a flattened arch. Plaster over wool, if there be much swelling, makes the best splint. Avulsion of the tuberosity necessitates flexion of the knee and extension of the ankle, a position which can be maintained by putting a slipper on the foot and attaching the heel by a cord to a broad band round the lower end of the thigh;

or the fragment may be pinned on to its proper surface. Fracture of the sustentaculum should be treated in a position of moderate inversion. In bad cases some eversion and flattening of the arch are almost certain to persist.

The **astragalus** is broken by similar violence to the above. It may be comminuted, especially in its anterior half; or the fracture may run through it transversely, longitudinally, or obliquely so as to separate the tibial surface from the rest of the bone. These considerable fragments, particularly the anterior half, or the tibial surface with more or less of the bone below it, may be dislocated widely, even through the skin; more or less dorsal or plantar flexion, with inversion or eversion, results according as the fragment goes backwards or forwards, inwards or outwards. On the other hand, the bone may be comminuted, and yet there may be no deformity and no crepitus may be obtained. Occasionally, after such an injury an acute suppurative inflammation of the ankle comes on; the abscess is opened, the finger discovers the fragment or fragments; their removal is likely to be followed by recovery with a good movable foot.

Treatment must be conducted as for the os calcis; but an anæsthetic is likely to be required if fragments are dislocated. If they cannot be reduced, and do not seem likely to do harm, they may be left for removal when the injured tissues have recovered to some extent; but if they are causing deformity or pressing on the skin, or if the application of a splint would endanger the skin over them, they must be removed, Much care must be taken as to the position of the foot. Pressure over a mass of cotton wool is the best way of controlling swelling and fixing the joint. In a few days plaster may be applied. In compound cases the wound must be cleansed, dislocated fragments removed, and a large wool dressing applied.

The **metatarsal bones** are almost always broken by direct violence. Pain, increased by pushing towards the tarsus the corresponding toe, mobility, crepitus, with little or no deformity are the signs; naturally they are best marked in the first and fifth.

The **phalanges,** more commonly than those of the hand, are broken by crushes. A little strapping wound round in a simple, or gauze in a compound, injury, supplies the necessary rest: the adjacent toes may be used as splints.

All the above injuries are frequently compound and may require primary amputation for damage to soft parts; but, as in the hand, if there be doubt as to the condition of any important parts, the whole should be cleaned and wrapped in an antiseptic dressing until it is clear whether the suspected parts are going to live or to die.

XXX. DISEASES OF BONES.

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OSTEITIS.

The same changes that take place in inflammation of other structures occur also in inflammation of bone. Owing, however, to its density, the same exudation and multiplication of cells cannot take place without a coincident absorption of the osseous trabeculæ; and from the intimate relations existing between the bone and its periosteum on the one hand, and the medulla on the other, both these structures are necessarily involved. Periostitis is, therefore, often stated to be the nature of the disease when really it is primarily a case of osteitis.

Pathological anatomy.—The bone, periosteum, and medulla become, when inflamed, more vascular. Their vessels dilate, and exudation takes place into the connective tissue within and around the bone. The Haversian canals enlarge, capillary loops form, and the embryonic cells increase in number. The osseous lamellæ are also eroded from absorption, forming Howship's lacunæ, which, communicating with the enlarged Haversian canals, convert the compact tissue into large irregular spaces similar to that of the cancellous tissue of bone. In the granulation tissue which now occupies these spaces are found a large number of multinuclear cells, called "osteoclasts," which are thought to produce this absorption of bone. This is the condition known as **rarefying osteitis.**

Meanwhile, coincidently with this rarefaction, new bone is being formed in the outlying parts beneath the periosteum and in the medulla. The small nucleated cells which take part in this formation are called "osteoblasts." These two processes of formative and rarefying osteitis are at first constantly going on together. Subsequent changes must vary with the conditions which produce the disease. In favourable circumstances, the inflammatory exudation undergoes resolution, and the bone returns to its original condition. If the irritation, whatever it be, has been long continued, complete resolution does not take place, and the bone remains larger and harder than it was before. A still further development of this latter process may ensue, and the osteitis then terminates in

osteosclerosis. In this condensing osteitis, not only does new bone form beneath the periosteum, increasing its size, but the granulation tissue within the Haversian canals organises into new bone, filling up the spaces which had been previously formed, and partly obliterating these narrow channels. The bone, therefore, becomes denser and harder than in the normal condition, and may even be converted into a tissue as dense almost as ivory. If the vessels be completely obliterated, the death or necrosis of a considerable area of bone may take place.

Rarefying osteitis may terminate in another way. If the irritation be more acute, the granulation tissue may form pus. This is the ordinary termination of osteitis around a piece of bone already dead, as will be described under *necrosis* and in compound fracture. In tuberculous osteitis of cancellous tissue the same softening process may ensue, producing the well-known **chronic abscess of bone** (page 891). If the inflammation be still more acute, and the infiltration take place before time has been allowed for the absorption of bone, the vessels in the Haversian canals may be compressed, and the circulation arrested over a considerable area of bone, causing its death. This is one of the ways in which **acute necrosis** arises (page 876).

Ætiology.—Injuries of various kinds are the most common exciting causes of osteitis, such as fracture (simple and compound), gunshot wounds, amputations, contusions, and burns. Trivial accidents in a subject already predisposed to inflammation of bone may give rise to the same condition. The diseases in which such predisposition exists, apart indeed from any injury, are syphilis and tubercle. Rheumatic affections also give rise to chronic inflammation of bone. Subacute osteitis terminating in abscess is not uncommon during the convalescence of typhoid fever. The other exanthemata, such as measles and scarlet fever, are often forerunners of both acute and chronic osteitis.

Symptoms.—If the compact tissue of bone is the primary seat of inflammation, it is, as a rule, a more or less chronic affection, beginning slowly and insidiously, with obscure dull aching pains, which are worse at night and in wet weather. It may make considerable progress before the patient seeks advice, complaining then only of persistent pain, which generally comes on in the evening, whilst sitting over a fire or after he has become warm in bed. On examination of one of the long bones, which is generally the part complained of, and more frequently the femur or tibia than any other, the bone will be found larger than its fellow, and tender on pressure. A part only of the shaft in its length is generally affected; but at this spot the whole circumference and thickness of the bone appear to be enlarged. At the margins of the inflamed area the enlargement tapers off to the normal condition of the healthy bone. It is thus easy by passing the finger and thumb down the sides of the bone from one end to the other to recognise the condition. If syphilis be the cause, the corresponding bone in the opposite limb may be similarly enlarged and tender; but it is scarcely likely to be of exactly the same size, nor is the enlargement likely to be in exactly the same part of the shaft. Other bones may also be affected. Except in the case of wounds and compound fractures, the soft parts are not involved in the inflammation. On the inner surface of the tibia, where the bone is quite superficial, there may be some slight œdema and "pitting" on careful pressure.

In the *diagnosis* of osteitis from tumours of bone, it may be observed that the endosteal growths are generally more globular in outline, whilst the periosteal scarcely ever completely surrounds the shaft. In both forms of tumour the ends of the long bones are the parts most frequently affected, and more than one bone is not likely to be involved. (*See* page 907 *et seq.*)

Treatment.—Iodide of potassium in gradually increasing doses, five, ten, or even fifteen grains, three times a day, should always be at once tried. In most cases this drug gives instant relief to pain in the early stages of chronic or subacute osteitis. The more nearly the inflammation approaches the acute type the less likely is it to give relief. In these cases especially, if accompanied by any febrile symptoms, an incision should be made through the periosteum over the centre of the swelling, and the trephine applied. If the swelling be long and fusiform, without any definite spot of acute tenderness on pressure, a groove may be made in the bone with a gouge, and deepened till the medulla is reached. Even if no pus is found, relief may be obtained, and the bone subsequently return to its normal condition. Suppuration and necrosis may sometimes in this way be avoided. In the more chronic cases, and especially where syphilis is likely to be the cause, iodide of potassium must be pushed and persevered with for some weeks or months, and mercury in some form should be added if the former drug fails to reduce the swelling and relieve the pain.

PERIOSTITIS.

Three varieties will be described, namely: (1) Simple acute periostitis; (2) acute diffuse, or infective, periostitis; and (3) chronic periostitis.

1. Simple acute periostitis.—This trouble may arise from an injury, such as a blow. Syphilis also gives rise to periosteal nodes, which are sometimes quite acute. Tubercle may also, in rare instances, be responsible for some softening nodes on the shafts of long bones, but is generally then an accompaniment of a tubercular osteomyelitis. Acute articular rheumatism, typhoid fever, measles, and scarlet fever are also sometimes followed by an acute periostitis.

Pathological anatomy.—The periosteum is swollen and vascular, easily separable from bone beneath, and infiltrated with inflammatory products. In favourable circumstances the parts involved may return to their normal condition; but, on the other hand, suppuration may take place. The abscess which forms has less tendency to burrow far before making its way through the fibrous layer of the periosteum than in the case of the diffuse form of periostitis. The cavity may heal as soon as the pus is evacuated, or a small portion of the compact tissue of the bone may die and be removed by exfoliation.

Symptoms.—The patient complains of pain, which is worse at night than during the day; and on examination of the painful part, an acutely tender swelling is found over one of the more superficial bones, such as the tibia. It will be fixed to one aspect of the bone, which will be free from any general enlargement, except at this spot. The temperature may be raised, but not to any great extent, and the general constitutional disturbance is of a mild type.

Treatment.—If syphilis be the cause, iodide of potassium will probably give immediate relief. In other cases rest in the recumbent position is the first and most essential point in the treatment of the inflammation. Hot fomentations should be applied, and frequently changed. Leeches are undoubtedly of service, but are objectionable if an incision has subsequently to be made, for they are liable to produce wound infection. A spare diet and a brisk purge complete the treatment for the early stage in a healthy person. If the swelling steadily increases and the pain continues, an aseptic incision should be made down to the bone before suppuration has taken place. Careful aseptic or antiseptic dressing should then be employed, to lessen the risk of a subsequent necrosis. If typhoid fever or any other exhausting illness be the cause of the trouble, a generous diet, stimulants, and tonics will be required, provided, of course, the original fever has passed away.

2. Acute diffuse (infective) periostitis, or acute necrosis.—The latter name is more generally used, as necrosis invariably follows, and there is, as a rule, osteomyelitis as well as periostitis. This is a most dangerous disease, and requires prompt and energetic treatment if limb and life are to be saved.

Pathological anatomy.—The long bones of the extremities are more frequently affected than any other parts of the skeleton, whilst the flat bones are very rarely attacked. It is also far more common in the lower than in the upper extremity. Commencing at the epiphysial line at either end of a long bone, such as the tibia, by an inflammatory effusion beneath the periosteum, it rapidly terminates in suppuration. The pus thus formed spreads far and wide between the periosteum and the bone, and unless a timely incision be made, may stretch from one epiphysis to the other, and completely encircle the circumference of the bone.

Coincidentally with this formation of pus beneath the periosteum, an extensive suppurative osteomyelitis may take place, the inflammation within and without the bone being directly continuous at the epiphysial line. The process thus begun stops at the next epiphysis, but it will result, if the suppuration be as extensive as described above, in the total necrosis of the shaft. Intermediate stages between this and the mere exfoliation of a portion of the cortex need not now be described, as the subject will be again considered under Necrosis (page 884). The periosteum separated from the shaft begins to throw out new bone, whilst the pus makes its way through several apertures to the surface. The new bone is called the invaginating sheath, whilst the apertures are spoken of as *cloacæ.* This disease sometimes attacks the epiphysis itself, and extends to the joint instead of spreading along the shaft. In that case a purulent synovitis as a rule, ensues, with all the dire consequences of a suppurative arthritis, which is described in the article on DISEASES OF JOINTS (Art. XXXIII.). It is rare for both the diaphysis and the epiphysis to be involved at the same time, but it does sometimes occur (Fig. 332). Pyæmia may arise from septic emboli, but it is not so frequent as in primary diffuse osteomyelitis. The means by which Nature rids itself of the dead bone will be described under Necrosis (page 884).

Ætiology.—Little is as yet known for certain as to the exact cause of this most interesting disease. It is known to be an acute infective disease, and believed to be due to the invasion of pyogenic organisms, which must in that case be supposed to act with a special virulence and through the blood. Or it may be that the vitality of the tissues is lowered by some local and trivial injury, and unable successfully to resist the invasion. By experiments on animals it has been shown that a simple fracture may be made to suppurate by feeding the animal on putrid meat. The disease is most common in young and growing boys between eight and eighteen years of age. Some slight injury appears oftentimes to be the immediate exciting cause, but it is frequently so slight as to have been scarcely noticed, and may even be entirely forgotten. Those that are rather feeble and delicate or ill-nourished seem more liable to be attacked than the strong and healthy.

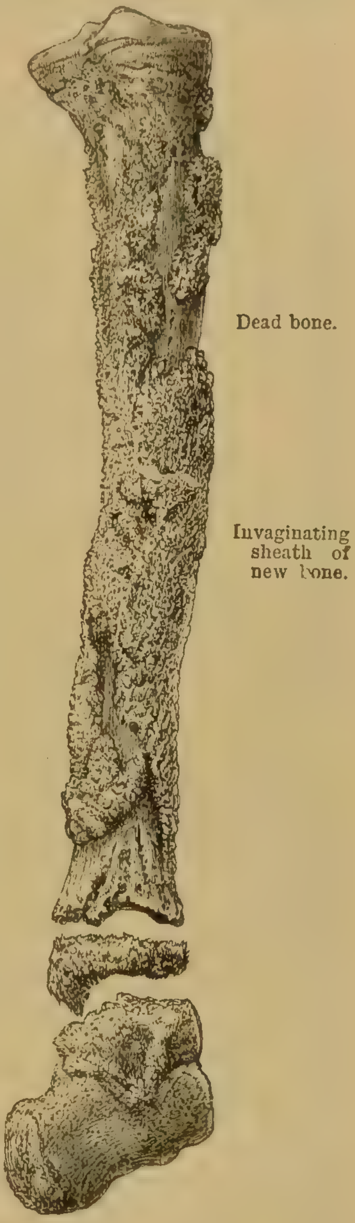

Fig. 332.—Acute Necrosis of a Tibia in which both the Diaphysis and lower Epiphysis have been involved, with consequent destruction of the Ankle Joint. (St. Thomas's Hospital Museum, No. 531.)

Symptoms.—Shivering, or even a rigor, with a temperature of 102° to 104° Fahr., may usher in the attack. General malaise, headache, loss of appetite, and even vomiting and diarrhœa, may also be present. For a time the patient often makes no complaint of any local trouble, and is thought to be sickening for some acute specific fever. In a day or two, however, the doctor's attention is drawn to an acutely tender and swollen part. A difficulty now arises as to whether this is due to acute rheumatism, cellulitis, or acute diffuse periostitis. *Cellulitis* may, as a rule, be excluded if there be no wound of the surface for the entrance of infective organisms ; and if there be such a wound, the inflammation rapidly spreads in all directions, beyond the next joint and over the adjoining bone. The tender swelling in the disease now under consideration is likely to be situated over the end of one of the long bones, such as the tibia, and therefore near a joint. In *acute rheumatism* the joint will probably be in the centre of the swelling, and more than one joint is likely to be involved. In *acute diffuse periostitis* the centre of the swelling—in the early stage, at any rate—will be either above or below the joint. More than one bone is also rarely attacked at the same time. If there be any doubt, an anæsthetic should be given, and deep fluctuation sought for in the usual manner. An exploratory incision may also be made if proper antiseptic precautions be taken, and is far safer treatment than leaving the case in doubt, and possibly finding at the next visit that the swelling has extended some distance up the shaft. Occasionally, too, under the anæsthetic, it will be found on handling the limb that separation has taken place between the epiphysis and the shaft.

Treatment.—As already suggested, early and free incisions are here imperatively needed to save the patient from necrosis of the whole shaft. It is far better to have made an unnecessary exploratory incision than to be a day too late in giving relief to pus pent up beneath the periosteum. From the severity of the constitutional symptoms and the presence of a local swelling, coupled with the absence of any other explanation to account for the patient's condition, it would be criminal not to give him the chance even in a doubtful case of arresting the progress of this acute infective disease. An incision having been made and pus found, the opening should be enlarged so as to permit a complete and thorough examination with the operator's finger.

The extent to which the bone is already denuded of its periosteum can thus be accurately estimated, and the opportunity taken of making openings in the best possible position. The incisions should be long and in the axis of the limb. Wide gaping of the wound takes place, which makes the introduction of a drainage-tube unnecessary, except where muscles have had to be divided, and tend to overlap or fill up the deeper part of the wound. After the cavity has been thoroughly washed out, a copious antiseptic dressing and a convenient splint are applied. The patient is at once relieved from pain, and the temperature falls. Frequent dressings and fresh incisions may from time to time be necessary, according to the progress of the case, which may be estimated so far as these points are concerned by the temperature and the amount of discharge. If a joint becomes involved, it must be treated in the same way, by free incisions.

The general condition of the patient must not be neglected. A most trying and exhausting illness is before him. It is, therefore, necessary to support his general health by a generous and easily assimilated diet. Stimulants, as a rule, are advisable, and morphia at times may be given to allay pain and lessen the irritability which is so commonly seen in this disease. Amputation may have to be considered, especially where a joint is involved, and the patient shows signs of sinking from the severity of the strain which is necessarily thrown upon his vital powers. The absence of sleep and inability to take freely of nourishment are, as a rule, the indications which warn the surgeon of the impending danger. The occurrence of pyæmia may also necessitate amputation, as by that means the source of further septic emboli is removed.

The extraction of dead bone resulting from this disease can only be accomplished when it has become detached from the living, and is lying loose in its periosteal shell. The mode of separation and the method of removal will be described under Necrosis (page 887). There is, however, one exception to this rule. In cases of total necrosis—*i.e.* the death of the whole circumference of a long bone, a condition which has probably been estimated by the surgeon's finger at the first examination—the dead part may be twisted off from the epiphysis without waiting for the invaginating periosteal sheath. It is not necessary, or in all cases advisable, to do this at once, for it is better in the case of the femur, which has no parallel bone to maintain the length of the limb, to wait to see how the patient stands the exhausting nature of his illness. If it is clear that he will have a hard battle for life, and especially if it be the tibia or fibula which is involved, subperiosteal re-section should be undertaken. If the whole length of bone has been bared of its periosteum, it may be sawn across in the middle, and each end removed by twisting it off from the epiphysis. If total necrosis has affected only one end of the diaphysis, the saw is applied at the junction of the dead and living bone, and the necrosed part removed from the epiphysis in the same manner. The discharge will, in suitable cases, at once diminish, and the patient be relieved from a very serious drain upon his constitutional powers. Means must be taken to maintain the length of the limb by extension, and new bone will form of equal strength to that which has been removed.

3. Chronic periostitis.—Simple uncomplicated chronic periostitis is rare except in syphilis, but as a part of a general osteitis it is common enough. (Osteitis, page 873, and Syphilitic Diseases of Bone, page 892.) As regards the *diagnosis* of chronic periostitis from osteitis, the enlargement involves only the surface of one aspect of the bone, whilst in osteitis the whole thickness of the bone should be increased. The inflammatory exudation takes place beneath the periosteum, and slowly develops into new bone, which may be arranged in layers parallel to the surface or in nodulated masses. The former eventually becomes sclerotic, as in condensing osteitis. When the latter assume definite size and outline, they are sometimes called *osteophytes.* This condition is seen round old rheumatic joints, or in the disease generally known as osteo-arthritis.

Beneath a chronic ulcer of the leg, a thick layer of bone (Fig. 333) may be produced upon the surface of the tibia. This would appear to arise from simple irritation; but at the same time it is more common in syphilitic subjects and in a bone that is already affected with a general osteosclerosis.

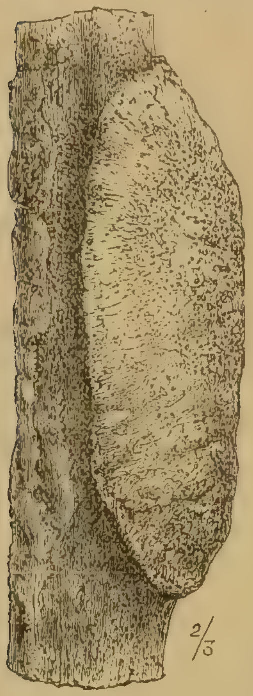

Fig. 333. — Osteoplastic Periostitis of a Tibia. An oval eminence of new bone, with a sharply defined border, has formed beneath a chronic ulcer of the leg. The specimen also shows chronic periostitis on other parts of its surface. (St. Thomas's Hospital Museum, No. 398.)

OSTEOMYELITIS.

Osteomyelitis is an inflammation of the medullary canal and of the tissue lining the cancellous parts of bone. It may be divided into acute and chronic osteomyelitis, of which the former has two very distinct varieties.

1. Simple acute osteomyelitis occurs most frequently after some definite injury, such as a fracture (simple or compound), gunshot wounds, and after amputation. It may also take part in a general osteitis, as previously described. The inflammatory exudation in the tissue of the medullary canal may undergo the same changes as in other parts: namely, resolution, suppuration, and organisation. In this simple variety of osteomyelitis the inflammation is local and does not spread, and is, therefore, probably not due to any infective organism. For example: in simple fracture it forms the internal callus, which is again re-absorbed. In an amputation a simple osteomyelitis closes the medullary canal, and in doing so may cause a very small and limited necrosis.

There are no special symptoms or treatment to detail apart from that of the particular injury in which it occurs.

2. Acute diffuse or infective osteomyelitis is a most serious and often fatal disease. As an accompaniment of acute diffuse periostitis or acute necrosis, it has already been described. It now remains for us to mention those cases in which the inflammation arises in the medullary canal, apart from the more general diffuse osteitis previously described.

Pathological anatomy and ætiology.—After amputation, compound fractures, and gunshot injuries, it was in former times of fairly frequent occurrence. It has now become comparatively rare; and this improvement must be undoubtedly attributed to the antiseptic treatment of wounds.

The open medullary canal lying exposed in a suppurating wound becomes infected with the same organisms: namely, the pyogenic bacteria, which have given origin to the suppuration of the wound or stump. Decomposition of pent-up discharges in an irregular and lacerated wound undoubtedly facilitates the growth of these organisms. The ptomaines of the putrefactive bacteria, if these be present, injure the vitality of the surrounding tissues, and so lessen the resistance to the pyogenic organisms. (*See* page 25.) The disease thus started in the medullary canal may spread throughout its whole length, and the products of this inflammation finding no outlet, and confined within bony walls, may so arrest the circulation as to cause necrosis (Fig. 334). (*See* page 885.)

This condition is also specially prone to give rise to embolic pyæmia, for the veins within the medullary canal are unable to collapse and assist in the formation of an adherent and healthy thrombosis.

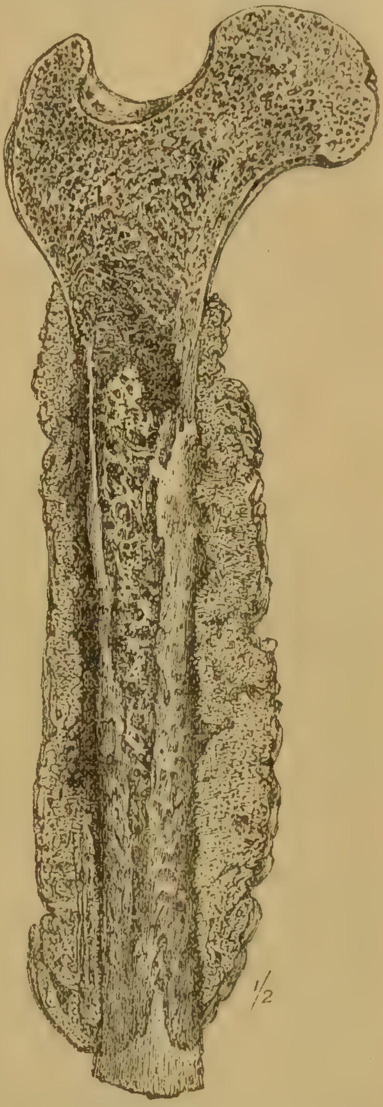

Fig. 334.—Necrosis of Stump from infective Osteomyelitis. (St. Thomas's Hospital Museum, No. 577.)

Symptoms.—Deep-seated swelling of the soft parts covering the bone affected, whilst the skin remains almost in its natural condition, severe dull aching pain, and much tenderness on pressure are the chief local signs. An increase of the discharge from the wound or stump, which has become offensive, is also likely to occur. If the bone is exposed and capable of examination, the periosteum will be found swollen and easily separated from its attachment. The bone itself when visible, as in the case of the skull, will be dry, yellow, and dead. If a probe can be introduced into the medulla it will encounter no resistance, and no bleeding will be produced till healthy marrow be reached. But the constitutional symptoms of an infective osteomyelitis are the most important, and lead to a careful examination for the local signs above enumerated. Shortly, they are those of septicæmia and pyæmia. A man who has had a compound fracture, or whose leg has been removed by amputation, may show signs of septic poisoning by a high temperature with considerable fluctuations, profuse sweats, and much depression. The surgeon naturally looks for some explanation, and if he finds the local condition before described, he at once suspects the presence of osteomyelitis. Should a rigor take place with a temperature of 105° Fahr., he feels convinced that such is the case. But even without this last evidence of general infection, the continuance of septic poisoning for a few days would induce him to open the wound and examine the bone in the manner detailed under treatment. If this is not done, and the case be one of osteomyelitis, the fever will continue, the rigors may be repeated, and the patient will die of exhaustion from septic poisoning or the metastatic abscesses of embolic pyæmia.

Treatment.—Such a condition ought not to arise under our present method of dressing wounds. It is now happily rare, and will become still rarer as the principles of aseptic and antiseptic surgery are more and more enforced upon the younger generation of practitioners. If, however, it has arisen, as it must do sometimes, notwithstanding all precautions, especially after gunshot injuries, or a suspicion be entertained that osteomyelitis may be the cause of the patient's condition, then the bone must be thoroughly exposed and examined.

If the periosteum and the medulla be in the condition above described, amputation above the seat of injury should be at once performed. A difference of opinion might be entertained as to whether it is necessary to amputate above the leg in osteomyelitis of the lower third of the tibia. Those who believe that the whole bone should be removed, if it can be safely done, would amputate at the knee-joint or through the thigh. On the other hand, it is quite possible that an amputation through the upper third of the tibia would be above the highest extension of the osteomyelitis; and if that is found not to be the case, the medulla may be effectually scraped with a sharp spoon till healthy tissues are reached, and the cavity treated with iodoform powder. In the case of infective osteomyelitis of the femur, an amputation at the hip-joint would be almost necessarily fatal, and, therefore, amputation high up in the shaft, and scraping out of the remaining medulla if it is found to be diseased, would be the best line of treatment. The removal of the suppurating medullary tissues by scraping has been very successful, provided, of course, pyæmia has not been already firmly established. It is, therefore, probable that with increased experience this method of treatment will be preferred to a high amputation, even when the patient's condition admits of its successful performance.

3. Chronic osteomyelitis takes place most frequently in conjunction with chronic osteitis and periostitis. It leads to a gradual narrowing of the medullary canal, and the bone undergoes the same changes as in osteosclerosis. (*See* page 873 and Fig. 342.) There is a tuberculous variety of osteomyelitis which is, as a rule, chronic or sub-acute in its progress, and often terminates in the so-called "chronic abscess of bone." This will be described under Tuberculous Disease of Bone (page 889).

EPIPHYSITIS.

Acute epiphysitis is an infective disease of the same nature as that already described under acute necrosis and acute osteomyelitis, occurring in the epiphysial end of a long bone frequently opening into the adjacent joint, but rarely extending along the shaft. Many of the cases recorded by T. Smith as "acute arthritis of infants" were cases of this disease. (*See also* Article XXXIII., on Diseases of Joints.)

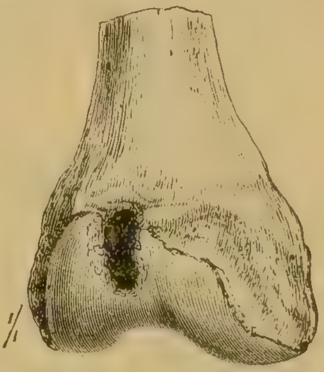

Fig. 335.—Acute Epiphysitis of Femur opening into Knee Joint. From an infant one month old. (From St. Thomas's Hospital Museum, No. 442.)

Pathology and ætiology. — In most instances it is impossible to trace the origin of the infection. In babies a few weeks old it might possibly arise as a pyæmic process during the separation of the umbilical cord. In older children the ulcerated throat of scarlet fever or diphtheria may in a similar manner give rise to this affection. It must be remembered that in acute necrosis and osteomyelitis we are often unable to trace the source from which infection has arisen. There does not seem to be any clear reason why this disease should be separated from those just named, except that its occurrence is almost confined to infants and young children. The acute inflammatory infiltration may involve the whole epiphysis and cause its necrosis. It may spread across the axis of the bone, along the epiphysial line, and cause the separation of the epiphysis from the shaft. Pus may open laterally through the periosteum, without involving the joint; but in certain joints, *e.g.* the hip, it must necessarily also in doing so make its way into the articulation. The pus may open through the articular cartilage directly into the joint (Fig. 335), or extend some distance into the medullary canal of the shaft. On the other hand, it occasionally works its way along the surface of the shaft away from the joint.

Symptoms.—The mother first notices that the child keeps one limb absolutely at rest, whilst the others are thrown about in a normal manner. On examination an extremely tender swelling is found close to one or other joint of that limb. In the first instance, the joint is itself not involved, but may rapidly become so in the course of a few days. The centre of the swelling is over the epiphysial line of one of the bones forming the articulation, and is, as a rule, most marked on one side, although the whole circumference of the limb at this spot may be swollen and tender.

Whilst moving the limb, grating may be felt or lateral mobility discovered. Separation of the epiphysis from the shaft would then be known to have taken place.

The constitutional symptoms are severe, and are those of acute septic poisoning.

Treatment.—No time should be lost in making an incision down to the bone in the centre of this swelling. To wait for fluctuation is to run a risk of the pus opening into the joint. For those cases in which the joint is already involved, the reader is referred to the article on DISEASES OF JOINTS (Art. XXXIII.). The usual aseptic or antiseptic dressings should be applied, and in an infant the whole limb should be wrapped in an abundant layer of wool, to act as a splint. Extension of suppuration should be most carefully watched for and treated in a suitable manner.

NECROSIS.

1. Necrosis with suppuration.—The death of bone *en masse* is termed Necrosis, whilst molecular disintegration, where the bone is insensibly removed or absorbed, is styled Caries. These terms, therefore, correspond to sloughing and ulceration of the soft parts.

Ætiology.—The inflammatory conditions which give rise to necrosis have already been considered. They act chiefly by cutting off the circulation to a given area of bone, which consequently dies from want of nutrition. Acute inflammation of the periosteum and medulla is the common antecedent of an extensive necrosis, but chronic osteitis and osteosclerosis may also terminate in this way, from the gradual obstruction and filling up of the Haversian canals.

Inflammation, too, of the soft parts around may spread to the periosteum and bone, and give rise to a limited necrosis, as in whitlow. Injury is also an important factor, but is of less moment than it was in the days before the antiseptic treatment of wounds.

A simple fracture is very rarely followed by necrosis. And in compound fractures and gunshot wounds, where formerly more or less necrosis was always to be expected, it is not at the present day so common for bone to die provided suppuration can be prevented. It must, therefore, be concluded that the inflammation and suppuration consequent upon the entrance of organisms is the usual cause of necrosis if it follow an injury. Syphilis and tubercle may both give rise to the death of bone, but the exact manner in which they do so will be described under separate headings. (Syphilis, page 892. Tubercle, page 889.)

Exposure to the fumes of *phosphorus* amongst lucifer matchmakers is also a potent cause of necrosis of the jaws. (*See also* page 928.) It is, however, now comparatively rare, although Mr. Treves records three cases in six months at the London Hospital from his out-patient room in 1882, and again in 1892 he records another of the upper jaw in which a cerebral abscess arose. The poison is supposed to enter through carious teeth. It slowly induces a gradually extending necrosis from the socket to the alveolus, and from the alveolus to the body of the jaw, till nearly the whole of the maxilla may be destroyed by necrosis. *Mercury* may also be the cause of necrosis of the jaw, involving more or less of alveolus from which the gums have retracted by the specific action of the drug.

Pathological anatomy.—Necrosis varies very much in its extent. It may only involve the cortical part of the bone, and is then called *peripheral necrosis.* The cancellous tissue alone is rarely affected except in a localised osteomyelitis, which is generally tuberculous, as in chronic abscess of bone (page 891). This is called *central necrosis.* The septic osteomyelitis of a stump after amputation involves rather more than the central portion of the shaft. Where the whole thickness of the bone is destroyed, as in some cases of acute infective periostitis and osteomyelitis, it is called *total necrosis* (Fig. 336). A portion of bone being dead from one of the causes previously described, we have now to consider the process by which Nature gets rid of the useless part. If inflammation and suppuration be the cause of the necrosis, the same process continues in the adjoining living bone, but falls short of producing strangulation of its own blood supply. If not already present, a rarefying osteitis commences converting the denser bone into cancellous. (*See* page 873.) This is continued, producing absorption of the living bone, till that immediately in contact with the dead is converted into a line of granulation tissue. The dead bone is then loose, and is called a *sequestrum.* Meanwhile, new bone is produced around by the periosteum over the area of necrosis and upon the surface of the adjacent living bone. This ensheathing bone is perforated here and there by openings for the escape of pus, which are called *cloacæ.* This invaginating bone is generally absent in peripheral necrosis of superficial bones, such as the inner surface of the tibia or the outer table of the skull. The removal of dead bone in such circumstances is spoken of as "exfoliation."

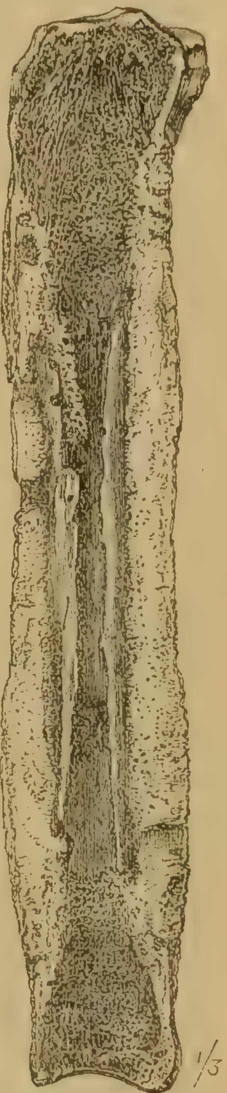

Fig. 336.—Total Necrosis of Tibia. (St. Thomas's Hospital Museum, No. 512.)

Symptoms.—These at first are those of the disease which produced the necrosis. Presumably, therefore, an incision has been made, and pus has been evacuated. The surgeon's finger introduced through the wound has probably also made sure of the nature of the case.

Supposing, however, this advantage has not been obtained, a suspicion that there is dead bone will be entertained from the history of the case, from the presence of enlarged bone beneath and from the appearance of one or more sinuses discharging thick pus.

The sinuses are button-shaped, with pouting florid granulations. The introduction of a probe will at once confirm the suspicion if it strike with a clear ringing sound upon the hard non-vascular sequestrum.

One or two points have now to be settled—the character of the necrosis, and the time at which its successful removal may be anticipated. If small and superficial—peripheral necrosis—there will be only a small amount of discharge, and not much enlargement of the bone beneath. Moreover, it can be felt and seen quite close to the surface. In the case of a large extent of both surface and thickness, as in total necrosis, there will be a very copious discharge, a large development of new bone, and several sinuses at some distance from one another; and on examination, the probe will pass through new bone for some distance before it strikes upon the sequestrum.

Its fitness for removal will depend upon whether the whole area of necrosis has become detached from the living bone by the line of granulation tissue. Some time must elapse before this can be fully accomplished. And this will vary with the age of the patient, the extent of the necrosis, the nature of the disease which produced the necrosis, and the particular bone affected. In the very young, when the bone is very vascular, the process is more rapid than in the young adult. The rapidity diminishes with age till the full adult period is reached. In the old, separation is sometimes very slowly accomplished, on account probably of the feeble circulation. Superficial necrosis is more quickly separated than the central or total necrosis. An exception may possibly be made for some cases of total necrosis after acute diffuse periostitis. If the whole shaft has died from this cause up to the epiphysial line, it will often be very quickly separated from its connections. The periosteum has already been stripped from the bone, and the vascular cartilaginous epiphysis is easily disconnected from the shaft. In necrosis from syphilis, many months, and sometimes years, may elapse before the whole of the dead bone has been separated from the living.

As regards particular bones, necrosis in the bones of the upper extremity is always more rapidly loosened than in those of the lower, on account, probably, of their greater vascularity. In the young adult two to three months will be about the average length of time for dead bone to become loose in the lower extremity, whilst in the upper the average would be about six weeks to two months.

Some estimate may be made as to its fitness for removal by an examination with the probe. When thoroughly loose, the probe on striking the sequestrum produces a hollow note. Two probes may also be introduced at some distance from one another, and by alternate pressure, a movement of the sequestrum may be effected if it has become detached. In the case of exfoliation, a director may often be inserted beneath the edge of the sequestrum, which is then tilted up if it is loose, and felt or seen to move.

Treatment.—Incisions may from time to time be necessary for the evacuation of pus at convenient spots to procure efficient drainage during the process of suppuration attendant upon the loosening and removal of dead bone. Absolute rest should be obtained by a splint or by a pillow and cradle. The antiseptic method should be employed on the well-known principles, if for no other reason than this, to avoid the frequent change of dressings which would otherwise be necessary. A rise of temperature should always lead to a careful examination for any fresh collection of pus. A very sudden rise of temperature and a sharp fall, with or without a rigor, should make the surgeon think of the possibility of pyæmia, especially if there be nothing in the appearance of the wound to account for this condition. Amputation may then be the wisest course for the patient.

General treatment must be attended to, for the continued suppuration is very exhausting, especially in the cases of acute infective periostitis. Good wholesome nourishment should be frequently given, wine or brandy being added according to the particular case in hand. (*See* Treatment of Acute Necrosis, page 878.)

When the time comes for the removal of sequestra, to which allusion has already been made, an anæsthetic must be given, and the dead bone exposed by enlarging the sinus or laying one sinus into another. If it is still overlapped by the invaginating bone, and cannot be easily withdrawn, the opening may be further enlarged. Sometimes the division of the sequestrum into two parts facilitates its extraction, and may avoid the further opening up of the sinuses. After it has been removed, the surgeon's finger should explore the cavity which is lined with soft granulations, feeling like velvet, so that no fragment of dead bone may escape detection. The wound having been syringed to remove blood clot and *débris*, it is packed with aseptic gauze. A layer of wood wool tissue and a bandage firmly applied would conveniently complete the dressing, which need not be changed, if the temperature remain normal, for some days.

An Esmarch's bandage, which renders the operation almost blood less, is in many cases of great assistance. But the oozing after it has been removed somewhat counterbalances this advantage. Still, in difficult cases, where a prolonged search is anticipated, it would be wise to employ this method.

2. Quiet necrosis, or necrosis without suppuration.—This is a very rare condition, but one that is now well known by the records of a few important cases.* A portion of bone having died, the amount of inflammation produced for its separation from the living bone around is so limited, that scarcely any pus is formed, and none works its way to the surface. Consequently, the enlargement of bone has most frequently been mistaken for some form of endosteal new growth. There appear to be two classes of cases—one in which the death of bone is produced by some specific disease, such as syphilis or tubercle, and the other in which an acute infection of the tissues outside the bone has produced an abscess which has subsequently healed, but in doing so has destroyed the vitality of a portion of the adjacent bone. It is probable that in the latter case the pyogenic cocci have never entered the bone at all, which has not, therefore, the same irritating qualities of most sequestra. The inflammatory barrier around the original abscess has been of such intensity as to cut off the circulation to a certain portion of the bone beneath, and has then produced healthy granulations, which have quickly closed the original abscess or wound. The sequestrum is then in an analogous position to an aseptic foreign body, which becomes encysted. In the former case of syphilis or tubercle it is well known that necrosis may arise in both these diseases from obstruction to the circulation of a given area of bone. If the organisms of suppuration do not gain access to the part affected, the inflammation may be only sufficient to separate dead from living, and not enough to involve the surrounding tissues. There is then no external sign of suppuration, and the case may resemble one of endosteal new growth.

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* Paget: Clin. Soc. Trans., vol. iii.; Morrant Baker: Med.-Chir. Trans., vol. lx. Charlewood Turner: Path. Soc. Trans., vol. xxxv.; Watson Cheyne: Path. Soc Trans., vol. xli.; Shattock: Path. Soc. Trans., vol. xli.

Diagnosis.—The diagnosis may present considerable difficulty. Where there is a history of a previous abscess or a septic wound, of which the scar remains to tell the tale, there will be less difficulty than one in which the enlargement of bone appears to have been of spontaneous origin.

In syphilis it would be rare for one bone only to be affected, and there would possibly be a history of other manifestations in the mouth, throat, or skin. The shaft of a long bone is also more likely to be the part enlarged, and not the articular extremity, where new growth is more common. The length of time would be greater in syphilis than in any malignant disease. The swelling might also be much diminished by iodide of potassium and mercury. In Morrant Baker's case the symptoms of disease in the shaft of the femur had existed for ten weeks, and spontaneous fracture had occurred four weeks before the amputation. After removal of the whole limb at the hip-joint for what was thought to be malignant disease, it was found to be a case of necrosis of a large part of the shaft of the femur from chronic osteitis.

A tuberculous osteomyelitis of the extremity of a long bone is, however, the condition most likely to give rise to mistakes, although, as a rule, there is no difficulty in the diagnosis of this disease, on account of the inflammatory exudation into the soft parts over the bone. But when such inflammatory exudation has not taken place, and the bone is large and hard, without any softened spots to throw light upon the nature of the case, there may be some difficulty in arriving at a positive diagnosis. Tubercle is slow in growth compared with malignant disease, but not slower than myeloid sarcoma. It never, however, reaches to any considerable size, and is unaccompanied by the special signs of new growth which will be described under Tumours of Bone.

Treatment.—This should consist in applying the trephine to the centre of the bony swelling. In this way the diagnosis may be confirmed, and the opening then enlarged for the removal of the sequestrum. If, on the other hand, the case prove to be one of new growth, amputation or re-section may be necessary. (*See* Tumours of Bone, page 907.)

TUBERCULOUS DISEASE OF BONE.

Pathology.—Miliary tuberculosis of bone is extremely rare, and need not be considered here, as it is an accompaniment only of a general tuberculosis.

Tubercle in a localised form attacks the cancellous tissue of bone, such as the small bones of the carpus and tarsus, the phalanges, the bodies of the vertebræ, and the ends of the long bones. In the latter position it enters largely into the causation of Joint Diseases, and will be described in the article devoted to that subject. Sub-articular caries was the former designation of this particular variety of tuberculous osteitis. On either side of the epiphysial line of a growing bone it is also fairly common. In the hip it must almost of necessity involve the joint if it reaches the surface of the bone, as the epiphysial line is within the capsule. In other bones, however, the disease may open externally without involving the joint, or may creep down the shaft and cause a more or less diffused tuberculous osteomyelitis.

Tubercle in bone commences as a rarefying osteitis by enlargement of the Haversian canals, thickening of the periosteum, and the development of a granulation tissue which shows the characteristic structure of the tuberculous disease. Two or more patches of varying sizes may exist within a short distance of each other, and subsequently coalesce; or there may be only one such small area of tuberculous growth. The changes that then ensue are very variable, and have led to the use of such terms as *caries fungosa*, *caries sicca*, *caries necrotica*—names which indicate the different terminations of this morbid process. The tuberculous deposit is surrounded by an area of inflammation, which at first shows itself in the form of rarefying osteitis. The granulation tissue occupying the spaces resulting from the absorbed trabeculæ may itself be invaded by tubercle, and undergo the same change as in the first deposit; or, for a time, it may partially organise and produce sclerosis of bone. A small portion of this may again be encircled by rarefying osteitis and tubercular infiltration, and being cut off from the circulation, may become a tuberculous sequestrum. Caseation and softening of the tuberculous deposit occur in the central part, whilst these changes are taking place in the periphery. As the process extends the inflamed bone becomes tuberculous, and again softens till the surface of the bone is reached or the joint invaded. Thus caries fungosa and necrotica are combined in the same tuberculous area, for small particles of bone may be isolated by the invasion and irregular growth of tubercle, and be cut off from their vascular connections. The tuberculous deposit may, on the other hand, be walled in by a healthy vascular growth in the surrounding rarefied bone, and slowly undergo partial absorption and calcification, whilst the surrounding inflammatory infiltration ossifies, and becomes sclerosed. This is the condition spoken of as caries sicca. If this occurs at the epiphysial line of a long bone, it may cause premature ossification and a shortened bone; or, on the other hand, in rare instances it may fall short of sclerosis, and merely irritate the growing bone to greater activity, and result in elongation. (*See also* Art. XXXIII.)

Chronic abscess of bone is a variety only of the tuberculous osteomyelitis, occurring in the cancellous tissue at the end of a long bone. Its pathological history is the same as the above, but its clinical features will require a separate description, which will be given later. Caries, which is essentially a tuberculous process, has usually been described as ulceration of bone. It will be admitted that we do occasionally meet with tuberculous disease of the surface or cortex of bone, but it is certainly rare; the term caries, if employed at all, should be used in this sense only.

Symptoms and diagnosis.—Tuberculous diseases of bone are insidious in origin, slow in progress, and very tedious in recovery. Pain or lameness is often the first indication of the disease, and the former is more severe at night than at other times. But some complication may be the first sign pointing to the nature of the case.

Enlargement of bone and tenderness on pressure must be looked for in the situations already mentioned, namely, the ends of long bones, the phalanges, and the tarsus or carpus. The signs of the disease in the vertebræ are considered in a separate article. In time the superficial tissues are involved, and œdema, redness, and a small area of fluctuation become evident. Or without any signs of inflammation, a collection of fluid may be felt some distance from the surface or beneath the skin.

The latter is generally called a "chronic abscess," and may remain indefinitely of the same size, or slowly increase till the skin becomes thin, and the fluid is evacuated spontaneously or by the surgeon's knife. If the cavity has been opened, a certain definite line of treatment is followed, which will be described presently. If it open spontaneously, a sinus is left which is of itself an indication of the nature of the case. The fluid is thin and watery, containing often gritty particles of bone, unless infection by pyogenic organisms has taken place, when true pus may be present. The granulations are œdematous and "pouting," instead of being florid and vascular, as in the true suppuration of necrosis from pyogenic infection. On the introduction of a probe, it will pass through soft and friable bone, meeting with irregularities of surface, but no hard and ringing sequestrum so characteristic of ordinary necrosis. The sinus will remain in this condition for many months, or years, unless some treatment be adopted to shorten its progress. And during this time infection by the micrococci of suppuration may take place, and render it the more difficult to bring the case to a satisfactory issue. Complications may arise, such as involvment of a neighbouring joint, amyloid disease of viscera, or exhaustion from profuse suppuration. General tuberculosis may take place, but it is comparatively rare as a sequel to tuberculous disease of the bones of the extremities.

Treatment.—An enlargement of bone from tuberculous disease, if seen sufficiently early, may for a time be left without any operative measures for its relief. Meanwhile, general treatment should be adopted, such as good air, good food, and absolute rest to the part affected. A splint of some kind should, if possible, be applied to ensure this result. In the case of the foot, a knee-rest is invaluable, as it prevents any pressure on the diseased part, and yet enables the patient to be out in the fresh air as often as his strength allows.

So soon as softening of the tuberculous growth is thought to have taken place, an incision should be made, and, if necessary, the bone perforated with a drill or gouge. If a cavity be found or granulation tissue be present, a Volkmann's spoon should be used to remove thoroughly the caseous products and tuberculous tissue. If a large cavity is left, a drainage-tube may be introduced, but should not be retained for more than twenty-four or thirty-six hours. In most cases a drainage-tube may be dispensed with altogether. The wound, in favourable circumstances, will heal, and if there be no recurrence or further growth of tubercle, the case will be at an end. On the other hand, in a few weeks or months a further collection of fluid may be found beneath the cicatrix of the first operation, when the same treatment must be again employed. If a sinus already exists, or pyogenic infection has obviously already taken place—a condition which would be recognised by the presence of true pus—then the same operation of erasion is undertaken, but without the expectation that primary union of the skin incision will be obtained. Of course, in all these operations it is understood that the most careful methods of operating and dressing on aseptic principles are enforced. The same care must be taken to prevent the entrance and growth of the pus microbes as in joint operations. The early termination of the case will be found to depend almost entirely on the absence of pyogenic infection.

Chronic abscess of bone, although not differing materially from the disease above described, requires a few words. It is more chronic in its progress, more localised, and more certainly relieved by operation than other tuberculous diseases of bone. It occurs most frequently in the young adult, and nearly always in the articular extremity of a long bone, and by preference in the upper end of the tibia. But no long bone can be said to be exempt, for it has been found in the lower extremity of the femur, in the radius, humerus, and ulna. It very rarely involves the neighbouring joint, being, as a rule, situated at some little distance from the articular cartilage, and circumscribed by a healthy zone of chronic osteitis. There is a history in most cases of some preceding accident or injury, followed by persistent pain in a definite spot, which is always worse at night and in wet weather.

On examination, an enlargement of bone is found in the position previously indicated, with one spot perhaps not bigger than a shilling or sixpence, which is exquisitely tender on firm pressure. There may be œdema and redness around, with a soft, almost fluctuating, area in its centre.

In the absence of the small soft spot indicating perforation of bone, the excessive tenderness limited to quite a small space is the indication of the position to which a trephine should be applied. If this is done and no pus found, the walls of the trephine opening should be perforated by a drill, in the hopes of reaching the small collection of pus which is thought to be present. A small sequestrum may also be found with the pus, as in other tuberculous diseases of bone. If neither pus nor dead bone be found, the patient will probably be relieved by the operation of a troublesome chronic osteitis, which was, after all, perhaps not tuberculous. The relief which is obtained in such a case is due in all likelihood to the removal of tension from the rigid Haversian canals.

SYPHILITIC DISEASES OF BONE.

1. Early periostitis.—In quite the early stage of acquired syphilis, whilst the eruptions are still present, and when even the primary lesion may not have ceased, it is not uncommon for the patient to suffer from small *periosteal nodes.* He generally complains of pain, especially at night, and tenderness at many different parts of his body. On examination, there will be found on the superficial aspect of the tibiæ, clavicles, sternum, ribs, or skull small and excessively tender swellings attached to these bones. Relief is almost at once given by iodide of potassium in 10-grain doses. But the mercurial treatment, which has already been given, must on no account be omitted, as on this depends the ultimate success of the treatment for syphilis. (*See also* the Article on SYPHILIS, page 407.)

2. Late periostitis.—Several years after the primary manifestations of syphilis one large periosteal node may be found on one or other of the long bones. It is generally accompanied by a chronic osteitis, and often ends in a more or less diffused osteosclerosis. Iodide of potassium gives only temporary relief, and, as a general rule, a permanent enlargement remains, which occasionally reminds the patient by attacks of pain and tenderness of his previous trouble.

3. A diffused osteitis, terminating in osteosclerosis of nearly the whole shaft of a long bone, may, however, be present without any definite node, as above described. When this is the case, many bones are affected, and generally in a symmetrical manner. They are as intractable as in the former instance, but relief of the severer symptoms may be obtained by large doses of iodide of potassium, with or without the addition of mercury, according to the effect produced by the former drug.

4. **Gummata of bone** are nearly always associated with periosteal nodes or a more or less diffused osteosclerosis. Some portion of the inflammatory infiltration, whether it be in the periosteum or in the bone, softens, and presents the ordinary characters of a gumma. (*See* page 408 and Fig. 82.) The overgrowth of connective tissue which at one part organises and at another undergoes degenerative changes is the most characteristic feature of syphilis when it attacks the bones, as it is when other organs are affected. An enlargement of bone, painful at night and tender on pressure, is the first indication of this, as of other diseases of bone. The special feature of its gummatous nature is the presence in some part of the swelling of a small area of fluctuation. If this remains untreated, the skin will eventually give way, discharging a thin watery fluid, and leaving an aperture with an irregular, perhaps crescentic, margin, like the gummatous ulceration of soft parts, leading to diseased bone. If the original swelling has been a softening periosteal node, the portion of bone affected will be the cortex only ; but if the whole thickness and more central parts of the bone be the primary seat of the trouble, a very irregular cavity and area of disease may be found to occupy the parts below the surface. If a large piece of dead bone is found, it is spoken of as *syphilitic necrosis,* which is, however, more common as the result of syphilitic ulceration of the parts covering a thin bone, such as the septum nasi and the irregular bones at the base of the skull. (*See* page

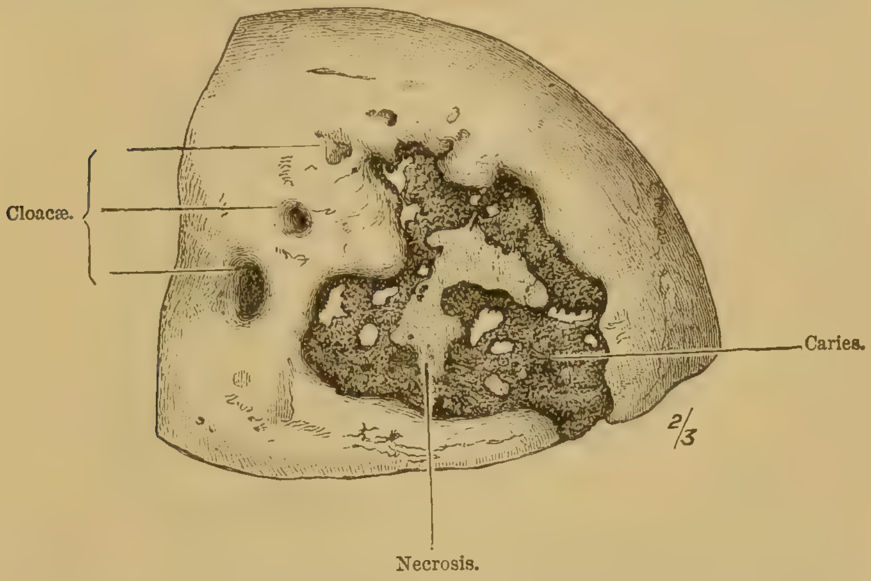

Fig. 337.—A Portion of the Frontal Bone showing its outer Surface. An irregular sequestrum of which a large portion is carious, and involving both tables of the skull unequally, has been completely detached from the surrounding sclerotic bone.
(St. Thomas's Hospital Museum, No. 559.)

408.) In osteosclerosis, ossification of the inflammatory products may result in a widespread necrosis, as has been explained before. The peculiarity of syphilitic necrosis by which it may be distinguished from other forms of necrosis, is the slowness with which it is separated from the living bone, remaining for many years attached to the surrounding parts. It is probable that this is due to the want of vascularity in the adjoining bone, in which the Haversian canals may be almost completely filled up with new material which has ossified. More frequently a gumma in bone results in what is called *syphilitic caries:* that is to say, the cavity is rough and irregular, and more nearly resembles ulceration of the soft parts. Particles of dead bone there may be, which come away in the discharge, but there is no appreciable "sequestrum." The spaces between these irregular walls and floor are filled with granulation, which shows no tendency to organise into healthy bone, probably from the same reason given above in speaking of syphilitic necrosis—namely, that the surrounding bone is sclerosed and, therefore, non-vascular. Caries and necrosis are also not uncommonly present together, differing as they do only in the extent to which the circulation has been cut off from the part affected. The skull and sternum, especially the former, are the bones most frequently affected with this intractable form of syphilis (Figs. 337, 338). Our museums are full of specimens showing its inveterate nature. They have been collected for the most part by our predecessors, before the treatment of syphilis was so well understood as at present. This present comparative immunity is apparently in part due to the lesser virulence of the disease; for the virulent forms of primary syphilis in cases where no treatment has been adopted are much less frequent than in days gone by. (*See also* Art. XXXIII. page 1031.)

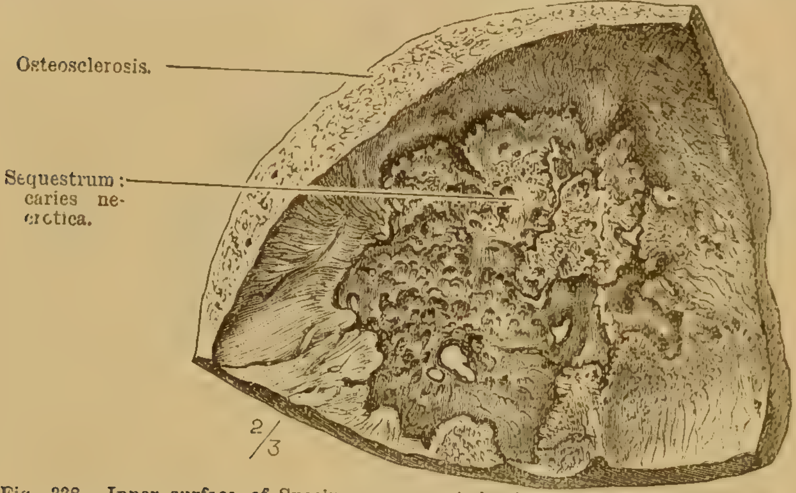

Fig. 338.—Inner surface of Specimen engraved in Fig. 337, showing that the sequestrum involves more of the inner than the outer table, and is thus locked within the skull. The living bone around is sclerotic. (St. Thomas's Hospital Museum, No. 559.)

Treatment.—Enlargement of bone from syphilis is always treated by iodide of potassium, whatever be the exact anatomical and pathological condition present. It is desirable, however, to enforce the doctrine that a gummatous lesion of bone, even if the skin be red and almost perforated, should not be opened. Again and again such swellings have been known to subside under iodide of potassium, in gr. x., gr. xv., or gr. xx. doses three times a day. When they have already opened, the same drug is given in the same or still larger doses for many months. An improvement is produced, and in some cases a cure effected. Occasionally, mercury is found necessary before this result is obtained.

But in many cases, if a sinus already exists, drugs appear to have little or no effect. Possibly this is due to the previous entrance of pyogenic cocci, which, under the peculiar circumstances of the surrounding sclerosis, cannot be properly dealt with by a healthy granulating barrier. What result a rigid antiseptic dressing would produce it is impossible to say, as the writer knows of no series of cases treated in this way from the moment when contamination could occur. For no argument would be serviceable for this purpose if the sinus has been exposed some time, even although an operation, such as erasion, has been undertaken. Gummata in other organs have been successfully dealt with by erasion and subsequent careful aseptic dressing. But the peculiarity of the bony structure before mentioned around the central lesion does not make the comparison of much service.

Bony lesions of inherited syphilis.—These do not differ much from those of the acquired disease. (*See* pages 424 and 428.) Large *periosteal nodes* are not uncommon, but are more likely to be multiple and symmetrical. They are, as a rule, less painful and less amenable to treatment. They are less frequent on the skull, and on the shafts of long bones are often accompanied by a diffused osteitis of the whole bone. (*See* Fig. 90, page 429.) They sometimes lead to a general enlargement, slight uniform curvature, and elongation of such a bone as the tibia. The *symmetrical lengthening of the tibia* is most characteristic, and the diagnosis can generally be confirmed by old interstitial keratitis, the physiognomy of the patient, and the presence of the teeth so familiar to all students of hereditary syphilis. These changes are most frequently seen between eight and eighteen years of age. In very young children it is difficult to distinguish between rickets, syphilis, and tuberculosis, so far as the bones are concerned, and the diagnosis is usually made by the history and by other lesions which may be present. No definite rule can be laid down for the treatment of these bony lesions of hereditary syphilis. Iodide of potassium and mercury are likely to be of service, but sometimes cod-liver oil and iron do more good than anti-syphilitic remedies. (*See* pages 426 and 430.)

Syphilitic dactylitis occurs both in acquired and in hereditary syphilis. Slight enlargement of one or more phalanges is the first indication of the disease. The whole bone is generally swollen and slightly tender. It is most likely to be mistaken, in children especially, for tuberculous dactylitis. In the latter the whole bone is not at first enlarged, and the deposit quickly softens down and approaches the surface. Anti-syphilitic remedies, as a rule, produce an improvement, if not complete resolution, in syphilitic dactylitis, whilst in the tuberculous form such remedies cause no alternation, and the enlargement slowly softens down into caseous *débris*.

HYPERTROPHY AND ATROPHY OF BONE.

Hypertrophy.—This is a term which should not be applied to an enlarged bone, unless the whole is of normal shape and density. For instance, chronic osteitis may produce an enlargement of bone, and in young subjects may even result in lengthening of the shaft of a long bone; but if this is only a part of a general osteitis with resulting osteosclerosis it should not be called hypertrophy. For the same reason, osteoporosis, osteitis deformans, leontiasis ossea, and acromegaly cannot strictly be cited as hypertrophy of bone.

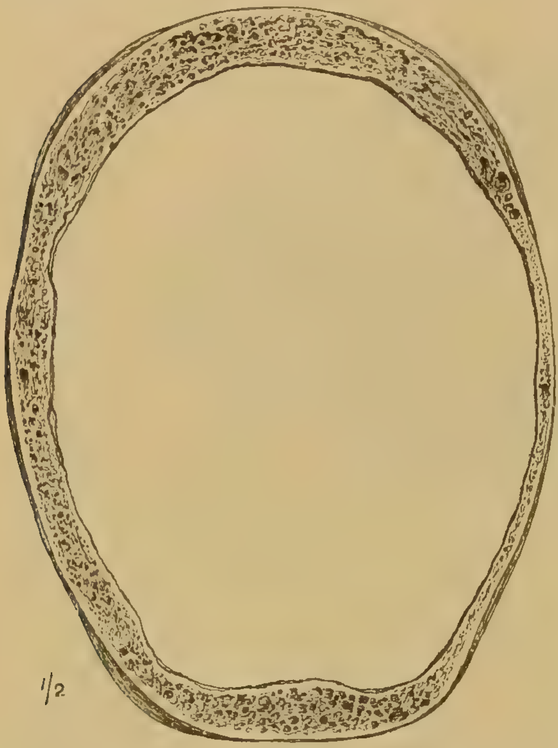

Fig. 339.—Horizontal Section of a Skull, showing Hypertrophy of the Bones of the left Side, accompanied with a corresponding Diminution in the Cranial Capacity. From a man who had suffered from epilepsy and became a criminal lunatic. (St. Thomas's Hospital Museum, No. 342.)

True hypertrophy of bone from excessive use in an extremely muscular subject is, perhaps, the best instance of this condition. Here the bones generally and the ridges for the attachment of muscle are found enlarged.

There are some rare cases of congenital hypertrophy of a whole limb in which the bones of that limb are correspondingly large and long. They may be classed as cases of true hypertrophy of bone. In elephantiasis of one leg in the young subject the bones are undoubtedly longer than those of its fellow, and appear also to be larger, but I know of no evidence to show that the bones in such a case are of normal density. The lymphatic obstruction has led to increased growth, but from the condition of the skin one could not argue that the bone was likely to be of normal consistence.

Atrophy.—This condition, as contrasted with hypertrophy, is comparatively common, arising from such simple causes as want of use or the changes incidental to old age. The bone becomes light and porous, the cancelli and the medullary cavity being enlarged and filled with fat (Fig. 340). The cortical layer is reduced to a mere

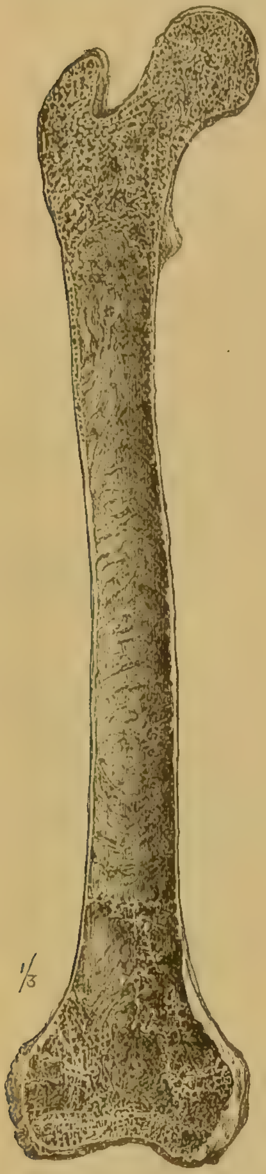

Fig. 340.—Atrophy of Femur, from a woman who had long been bedridden. (St. Thomas's Hospital Museum, No. 343.)

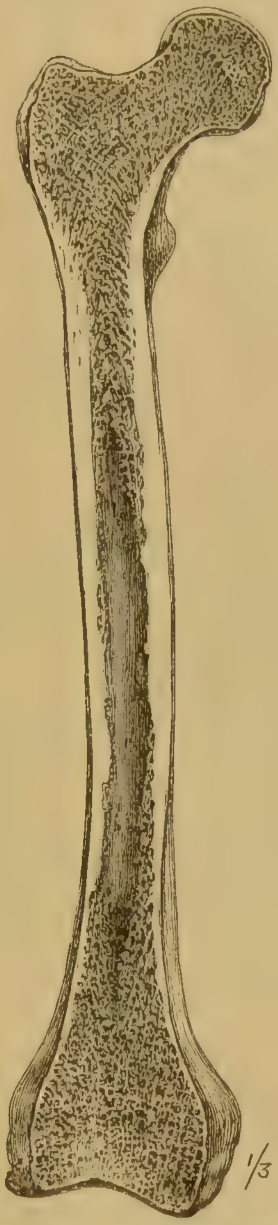

Fig. 341.—Normal Femur. (Royal College of Surgeons Museum.)

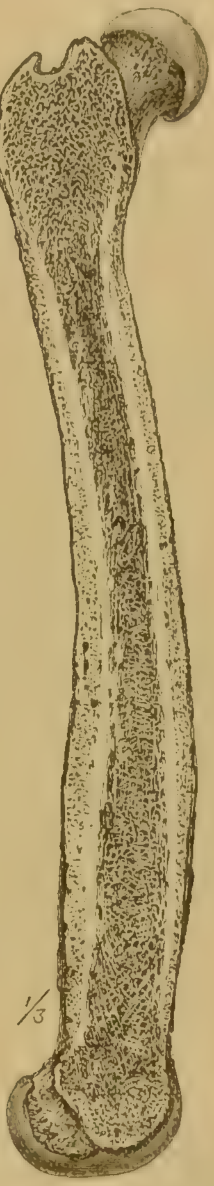

Fig. 342.—Chronic Osteomyelitis and Osteosclerosis. (St. Thomas's Hospital Museum, No. 405.)

shell in advanced cases, and in such circumstances may be very brittle, and break with the slightest degree of violence. Of general causes, senile degeneration is the most common, but it has been shown by Professor Humphry that the bones of aged people after fracture unite as readily as those of the young.

Long confinement to bed from any cause undoubtedly predisposes to atrophy, and possibly in part accounts for the occasional occurrence of fracture of the opposite thigh-bone on getting up after prolonged rest in bed from a previous fracture of the femur.

The cachexia of malignant disease is also thought to induce atrophy of bone, from malnutrition. Certainly such bones break easily without the secondary deposit of new growth.

It has long been a question of speculation whether the bones of the insane are also more brittle than those in health. They often show signs of atrophy, but whether this is peculiar to the insane or what might be seen in those who lead a very inactive life from other causes it is difficult to say. As regards their spontaneous fracture or fracture from slight causes, there would generally be a little difficulty in accepting the evidence of an attendant, whose interest it may be to underrate the force he has been induced to use.

Other general causes there must be of which little or nothing is known. For example: a girl of fourteen is recorded by Stanley as a case of fragilitas ossium, and is said to have had thirty-one fractures, all of which readily united.

Local causes for atrophy of bone are of practical importance to the surgeon, as the knowledge will lead him to deal gently with bones when such a condition may be suspected to exist. During the disuse of a limb from disease, such as disease of the knee-joint or a badly-united fracture in which weight cannot be borne on the limb, the bones undoubtedly atrophy and become very brittle, so that when the time comes to employ passive movement of the joint the femur is liable to fracture. Infantile paralysis may also be cited as another cause for these local changes. It is well to bear in mind that, in all these cases of atrophy of bone, union takes place as readily after fracture as under more healthy circumstances. (*See also* page 726.)

OSTEOMALACIA.

This is a disease of the fully-formed adult bones in which bending or fracture of one or more parts of the osseous framework of the body is liable to occur with very great deformity. As regards its actual causation, very little is known. It is more common in females than males, in the proportion of about ten to one; and in females it would appear that pregnancy must be the most important exciting cause, for a very large proportion are shown to be first affected during this period. The situation of the disease is also remarkable, for in those connected with pregnancy, the pelvis and lower part of the spine were the first to give any indication of the disease. The great majority of the cases analysed by Durham, in Guy's Hospital

Reports (vol. x.), began to suffer between the ages of twenty-five and thirty-five. It would be out of place here to discuss the theories that have been advanced to explain the reason why the bones should be so extensively diseased whilst the rest of the tissues remain to all appearances unaltered. (*See also* page 729.)

Pathological anatomy.—The disease consists essentially in a softening of the bony tissue. The earthy matter is in the first place absorbed, leaving the organic matrix with a faint indication of its lacunæ and canaliculi, producing an appearance somewhat similar to a microscopical specimen of bone which has been treated with hydrochloric acid. The parts decalcified are gradually transformed into a soft gelatinous tissue, without any osseous trabeculæ. The process goes on till the whole of the interior of the bone is converted into a substance like jelly, leaving, however, a thin layer of cortex beneath the periosteum. The vessels, no longer contained in rigid walls, allow their blood to escape, so that the semi-fluid pulp-like substance previously described is always more or less blood-stained.

Symptoms.—Nothing definite is complained of except pains like rheumatism, till some very obvious deformity points out the nature of the case. Most frequently the discovery is made by the family doctor from an examination of the pelvis during parturition. The pelvic .bones may at first be alone affected. The promontory of the sacrum is thrust forward by the weight of the body, whilst the sides of the pelvis are made to approximate by pressure acting through the two femora. The pelvis thus assumes the beaked form so characteristic of this disease, as distinguished from the rachitic pelvis. (*See* Fig. 75, page 369.)

The bones in osteomalacia, being soft, may be bent and altered in shape by the hands of the medical attendant: a change which cannot possibly be accomplished in the deformity produced by rickets. The spine may also be very seriously deformed, and chiefly by a very great increase of its natural curvatures. The thorax may be distorted by the bending or fracture of ribs from the action of the respiratory muscles. The bones of the extremities may be bent and broken in many places, allowing the feet to touch the head, or the limbs to assume the most fantastic shapes. The resulting deformity is the most exaggerated and most extreme condition of which the human skeleton is capable. At the same time it should be remembered that the changes may be confined to the bones of the pelvis and lower part of spine, and never progress beyond this portion of the body.

The urine has almost always, when examined, contained an excess of lime salts, pointing to the conclusion that the inorganic constituents of bone are eliminated through the kidneys.

The disease does not of itself appear to be fatal, but a large proportion of recorded cases have died from mechanical difficulty arising at parturition. Apart from this cause of death, the patients may live a very long time in a crippled and bedridden condition, and die only from some intercurrent malady. If the ribs give way, and cause compression of the lungs, or the softening of the bodies of the vertebræ interferes with the integrity of the spinal cord, a fatal termination may quickly supervene, and cut short an otherwise long and tedious complaint. Recovery or obvious improvement is stated, however, to have taken place in twenty-two out of the 145 cases recorded by Durham; but they appear to have been nearly all connected with pregnancy in which the disease was confined to the pelvis and the lower part of the spine.

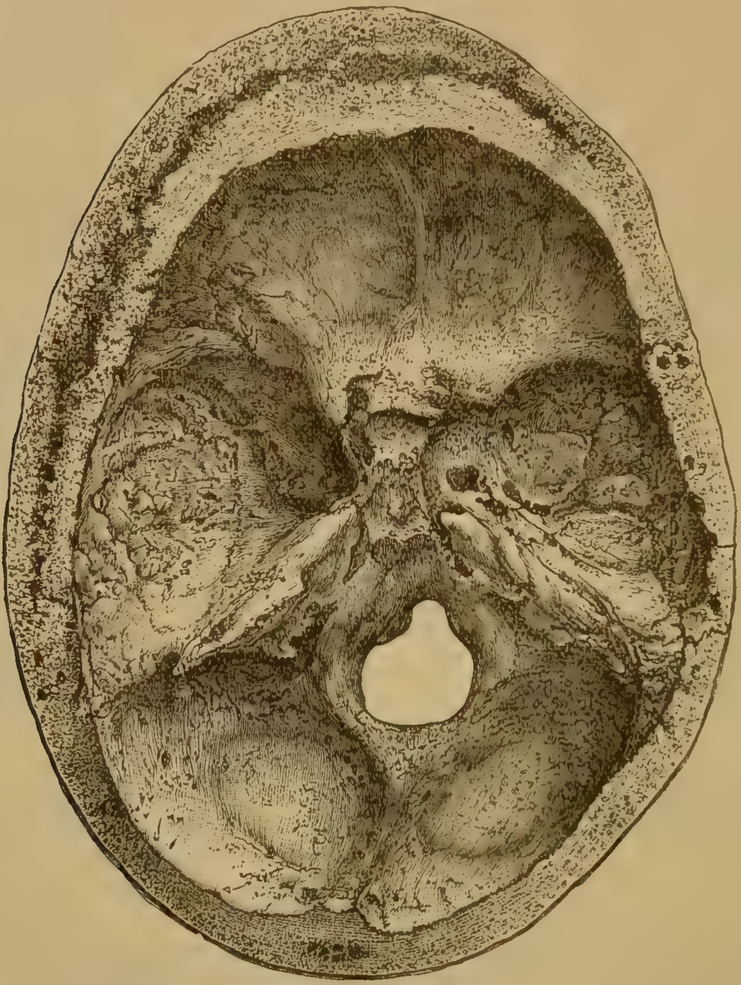

Fig. 343.—Base of Skull from a Case of Osteitis Deformans. (St. Thomas's Hospital Museum, No. 422.)

Treatment.—Beyond the ordinary and judicious treatment that would be suggested for any chronic and incurable complaint, there is really nothing to be done. In the early stages fractures might unite, but later in the disease this result could not be expected to take place.

OSTEITIS DEFORMANS.

To Sir James Paget we are indebted for the first clear account of this interesting disease. His earliest series of cases was published in vol. lx. of the Medico-Chirurgical Transactions, 1877, and seven others in vol. lxv., 1882. Since the publication of these cases a large number have been recorded by other observers, but without much addition to our stock of knowledge. Single bones previously labelled in our museums as osteoporosis, in which the life-history was incomplete, may now, we think, be regarded as cases of osteitis deformans. If this be so, one more obscure bone disease has been reclaimed by these observations from the unclassified group of osseous deformities.

Causation. — Nothing whatever is yet known as to its ætiology. Cancer may now be excluded as having any direct influence in this respect; and no one constitu-

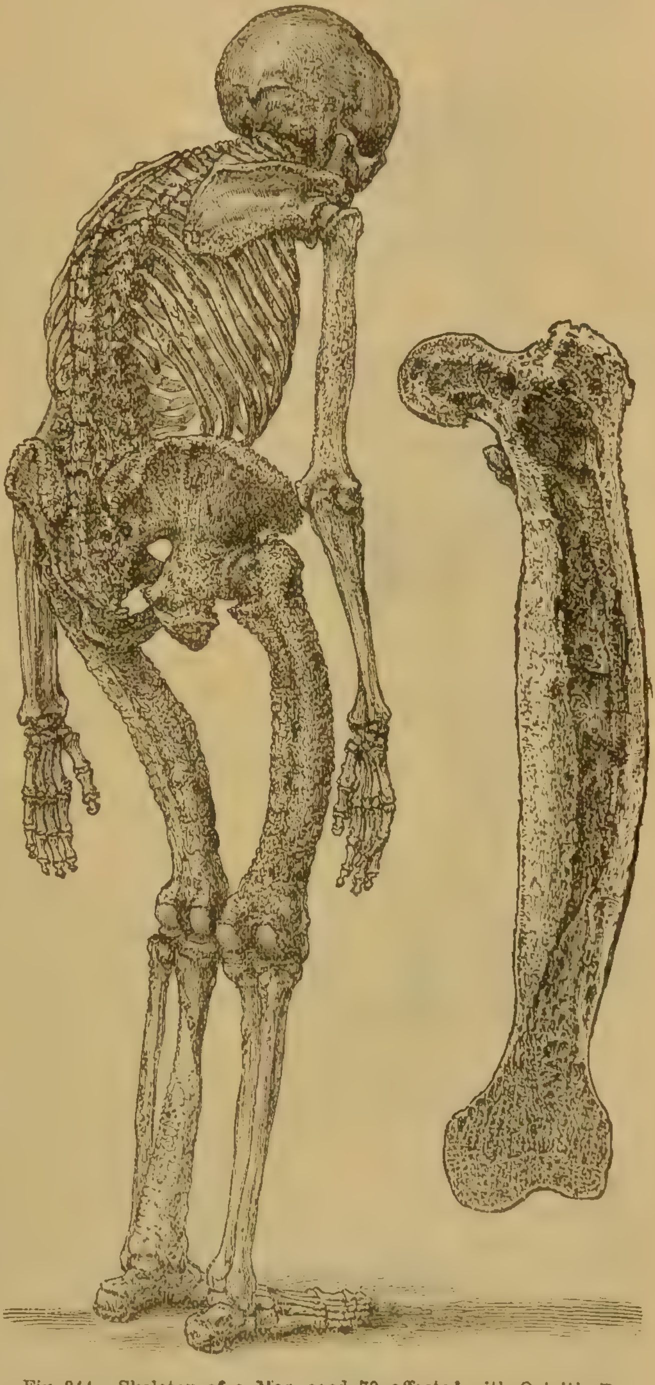

Fig. 344.—Skeleton of a Man aged 70, affected with Osteitis Deformans. The section of femur is from the same specimen. (St. Thomas's Hospital Museum, Nos. 416, 417.)

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tional diathesis or disease can be traced through a sufficient number of cases to make it probable that they have anything to do with its origin.

Pathological anatomy.—Chronic inflammation especially affecting the long bones and those of the skull, beginning in mid life or in old age, appears to be the most probable pathological condition through all its stages. Commencing as a chronic and rarefying osteitis, in which the normal compact tissue becomes finely porous and reticulated, and in which also fresh bone is formed beneath the periosteum, it slowly undergoes that hypertrophic and partially sclerotic change that we see so well developed in the later stages of the malady. The medulla is also filled up with the same porous bone (Fig. 343). In Sir James Paget's words: "The bones enlarge and soften, and those, bearing weight, become unnaturally curved and misshapen." The curvature of the long bones is certainly seen quite early in the disease, and it is fair to assume that this arises from the weight of the body acting through soft and yielding bone (Fig. 344). Late in the disease, when the bone can generally be examined *post-mortem*, the density of the new formation is such as to contradict any idea of its yielding to pressure. Another question arises as to this hypertrophic rarefying osteitis. Does the bone also lengthen? A few specimens seem to indicate that such may be the case.

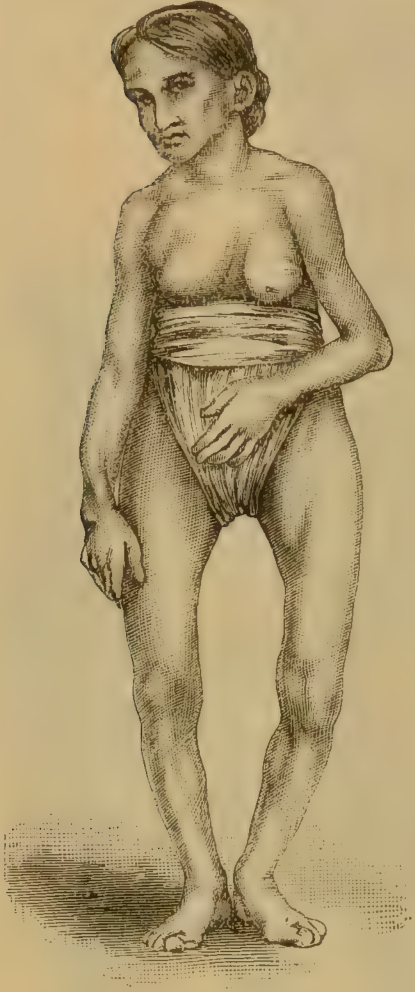
Fig. 345.—Osteitis Deformans, from a case under the writer's care. (From a photograph.)

In the accompanying illustrations of a case recorded by the writer in the Pathological Society's Transactions (vol. xxxix.), it will be seen that, besides the ordinary symptoms of osteitis deformans, some single bones are enlarged and curved, and, being unsymmetrical, show evidence of elongation (Figs. 345, 346, 347). In the right fore-arm the radius is very much curved and enlarged, especially in its lower third, whilst the ulna is of normal size and quite straight. The radius must here be increased in length, for otherwise the hand would not be displaced in the opposite direction to the curvature of the radius, the curvature alone somewhat reducing the length of a bone. The left humerus is also, notwithstanding its curvature, from an inch and a half to three-quarters of an inch longer than the right humerus, the measurement being taken along the arc of this curve (Figs. 346, 347).

Symptoms.—After middle life or in old age the first symptoms of the disease are generally noticed. The loss of height, stooping posture, and low position of the hands in the erect position are, perhaps, the most remarkable features. The curved lower limbs held apart, with the knees slightly bent and overhanging the ankles, are still more characteristic on careful examination of the nude figure. The vault of the cranium is comparatively large for the face, and on inquiry it will often be found that larger and larger hats have of late years been ordered by the patient. The upper part of the spine will be seen to be quite fixed in its bowed position, whilst the ribs are crowded together and immovable. The breathing is, in such circumstances, entirely diaphragmatic, in a manner exactly similar to that of spondylitis deformans. In the latter disease, however, the immobility of the spine and ribs is alone observed, combined possibly with general osteo-arthritis. But there is no enlargement or curvature of the long bones, and the skull is entirely unaffected. On careful examination of the lower limbs in a case of osteitis deformans, the long bones will be found very much enlarged, and uniformly curved outwards and forwards from one end to the other. Other bones may also be affected, and generally in a symmetrical fashion; the case previously described (Figs. 345, 346, 347) being quite exceptional.

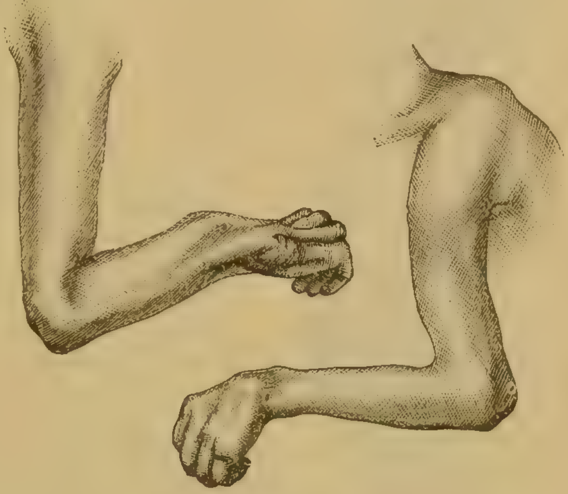

Fig. 346.—The upper Extremities of the Case shown in Fig. 345.

The patients often complain of pain in the lower limbs and spine, but it is not severe, and is generally described as rheumatism. The general health may remain entirely unaffected, except from such indisposition as may arise from want of exercise. Life is not materially shortened, except possibly from the onset of such an intercurrent malady as bronchitis, when the fixity of the chest would materially impair the patient's powers of breathing.

Treatment.—Iodide of potassium has not been found of much value in osteitis deformans, either for the relief of pain or to check the progress of the disease. There is no particular line of treatment to recommend other than what common sense would dictate for each special case.

ACROMEGALY.

This is a name given by M. Marie* to a disease which is now recognised as having special characteristic features, but for which no definite pathological cause has as yet been discovered.

Symptoms.—Acromegaly may make its appearance at almost any period of life, but is perhaps most frequent in the young adult. It commences so insidiously that the patient is unable to give any exact account of its origin, his friends often being the first to notice the change in his appearance. Enlargement of the hands and feet, which is chiefly due to hypertrophy of the soft parts, is the most characteristic symptom of this curious affection. The face is elongated and deformed, from the increased size of the facial bones, especially the upper and lower jaw, the latter being the most obvious, from its projection forwards and increased vertical depth at the chin. The superciliary ridges are also more prominent, producing a frowning aspect; the soft parts are more bulky, especially in the lower lip; but the nose, ears, and eyelids are all noticeably thick and heavy-looking. The general effect of the appearance is that of a dull, heavy, coarse-featured person. The muscular development is good, and in some cases seems to be above the average till quite late in the disease, so that the patient is able to continue his or her occupation, unless the deformity of hands or feet is in any way a hindrance in the work they may have to do. The mental functions are, as a rule, unimpaired, although persistent headache and gradually increasing blindness have been noticed in some of the cases. The progress of the disease is very slow, and is shown by a gradual loss of muscular power, so that the body is no longer held erect, the spine becoming kyphotic or scoliotic. The bones of the extremities from the periphery towards the trunk gradually increase in size, the

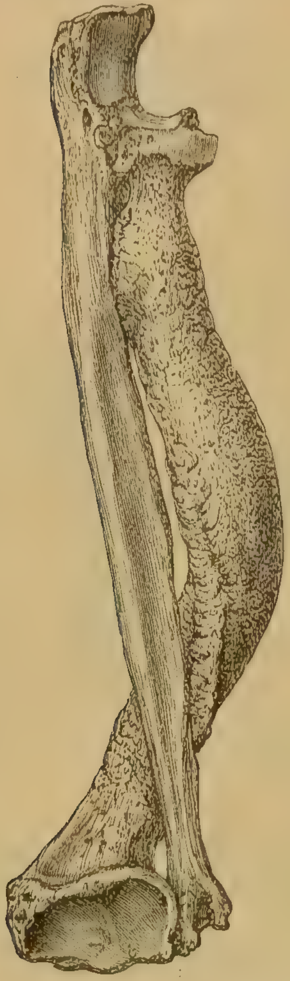

Fig. 347.—Osteitis Deformans. Bones of right fore-arm, from case shown in Figs. 345, 346. The radius is increased in length and enlarged.

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* *Revue de Méd.*, April, 1886.

fore-arms before the arms, and the legs before the thighs, but this change is unaccompanied by any curvature of the long bones.*

The patient gradually becomes exhausted from general debility, or dies from some intercurrent disease.

Pathology.—As regards the pathology of acromegaly, it must still remain in obscurity. Enlargement of the pituitary body, which has been frequently observed, may be a symptom rather than the cause of acromegaly, inasmuch as it is not always enlarged. Atrophy of the optic discs, which is present in so many of these cases, is undoubtedly caused by the pressure of this body. The thyroid gland has also been found altered, sometimes enlarged, sometimes atrophied, and in others normal. Persistence of the thymus has also been held responsible for this disease; but no one of these three organs can be said to be the cause, for in some instances none of them were affected.

Diagnosis.—The differential diagnosis has to be made from myxœdema, osteitis deformans, leontiasis ossea, and arthritis deformans. From *myxœdema* the enlargement of bone, if it can be detected in the early stage of acromegaly, would be sufficiently distinctive. The skin in acromegaly shows no alteration, and the hypertrophy of the soft parts is confined to the hands, feet, and face. The hair also is unaffected.

In *osteitis deformans* the long bones invariably become curved, there is no special tendency to enlargement of the distal ends of the extremities, and the face is small, whilst the skull is enlarged.

Leontiasis ossea is an affection only of the facial bones and skull, which may become excessively and irregularly enlarged, much more so than in any case of acromegaly, but the soft parts are normal, and the distal extremities are unaffected.

Virchow has pointed out that in acromegaly, as in *arthritis deformans* (or osteo-arthritis), there are osteophytic outgrowths at the articular ends of the long and short bones, and also along the margins of the vertebræ and the large flat bones, such as the ilium. "But there is here also a striking difference, for in acromegaly the joint as such is not affected." "The changes are extra-articular, and have their seat outside the ligamentous and capsular constituents of the joint."

LEONTIASIS OSSEA.

Virchow, in his work on Tumours, applied this term to a rare group of cases which vary much in their extent, but are similar in their pathological anatomy. Hyperostosis of the facial and cranial bones is the distinguishing feature of this disease. Large bosses of bone, often symmetrical, are developed from the bones of the face and cranium, which slowly and steadily increase in size and produce the most hideous deformity. The osteoma, with which it may be compared, is an isolated outgrowth of bone, whereas hyperostosis is an enlargement of the whole bone, all its processes being involved in the growth. As it increases in size, it becomes very irregular, and other bones in the neighbourhood become similarly affected. The eyes are often displaced, from the gradual diminution of the orbital cavity, and the nerves are compressed, from the narrowing of the foramina through which they pass. Pain, blindness, and deformity are the principal clinical features with which we have to contend. (*See also* Article XXXVII., on DISEASES OF THE HEAD, Vol. II.)

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* For description of skeleton, see Broca: *Archives Générales de Méd.*, Dec., 1888; Virchow: *Berliner klinische Wochenschrift*, Feb., 1889; Henry Alexis Thomson: *Journal of Anatomy and Physiology*, vol. xxiv., 1890. For numerous cases, see "Essays on Acromegaly and Bibliography," with cases. New Sydenham Society, vol. cxxxvii., 1891.

Many years may elapse before these symptoms justify any operative interference; and then only when it is more or less unilateral is it possible in such cases to interfere with advantage to the patient.

TRANSPLANTATION OF BONE.

Under this heading may be included re-implantation of bone which has been removed, as in trephining operations, and true grafting, or transplantation, in which a piece or pieces of bone are removed entirely from one part to another or from one patient to another.

With regard to the first, it was formerly the practice to divide the disc of bone that had been removed into small fragments before it was re-implanted. In young bone certainly this has been found to be unnecessary if the disc is not very large. In old bone, whatever its size, it would probably be wisest to drill one or more holes through it, to prevent the possibility of necrosis.

Macewen has pointed out that in returning large slabs of bone after trephining, the best practice to ensure its continued vitality and to maintain drainage is to punch or drill holes a quarter-inch in diameter, so that instead of solid bone, a mere lattice-work is left.

There is no doubt about the possibility of successfully transplanting a piece of human bone into a freshly-made wound. Macewen published a remarkable case in the Proceedings of the Royal Society in May, 1881, which illustrates this line of practice. Wedges of bone were removed from the tibiæ of children who required osteotomy for deformity, and were planted between the muscles of a boy's arm whose diaphysis had been lost a year previously from infective periostitis. The two epiphyses were eventually successfully united, and the boy obtained a useful arm. The wedges of bone were minutely divided, irrespective of the periosteum, and placed in a groove previously made between the muscles of the boy's arm. The operation was performed on three separate occasions, two wedges being used each time.

Young and vascular bone may not require such minute subdivision; but if subdivided, care should be taken not to crush the bone, which should be cut as cleanly as possible. The part most likely to be successful for transplantation is that immediately beneath the periosteum in a young subject, on account of the osteoblasts.

The writer has tried transplanting portions of a long bone with its periosteum from a puppy for a case of un-united fracture with deficient bone formation. The wound suppurated, so that, although a successful union was obtained after many previous failures, it is doubtful how far the result was due to the transplantation. More will be done in this direction now that wounds can be made to heal without the intervention of strong antiseptics, which must injuriously affect the young cells in the transplanted bone.

TUMOURS OF BONE.

The general pathology of tumours is given at page 443, but it is necessary here to go more fully into the clinical history and treatment of these diseases in relation to the bones.

Osteomata. — Of the innocent tumours, we may refer first to the osteomata. We know nothing of their ætiology except the fact that in the multiple osteomata, occurring about the epiphysial lines of the long bones, they appear very frequently to be hereditary. There are two varieties: the ivory and the cancellous osteoma. (*See* page 455.)

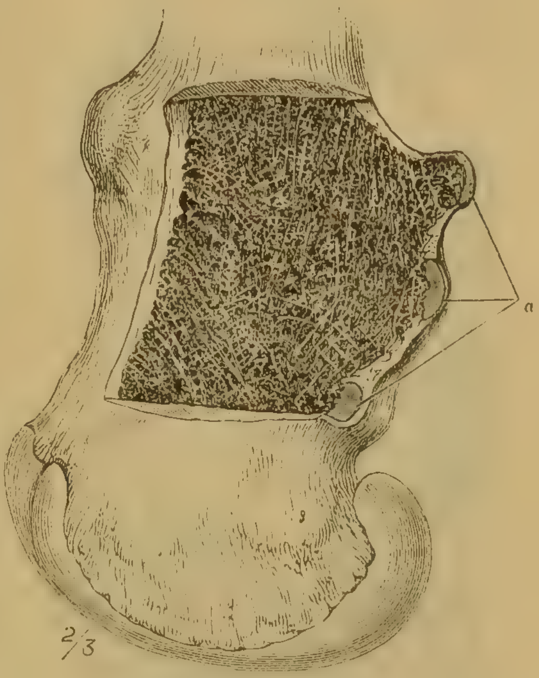

Fig. 348.—Osteomata (*a*) close to Epiphysial Line of Femur. Each is capped with cartilage, whilst its base is continuous with the cancellous tissue of the shaft. (St. Thomas's Hospital Museum, No. 601.)

The **ivory osteoma** occurs chiefly on the flat bones, such as those of the skull orbit, scapula, and pelvis. It is of remarkable density, resembling throughout its structure, to whatever size it may attain, the cortical bone from which it springs. It is generally single, and causes little inconvenience except in special localities, such as the frontal sinuses, orbit, auditory canal, or the interior of the skull. (*See* Fig. 101.) It may attain a large size, and totally obstruct a canal like that of the ear; or, distending the frontal sinus, displace the eye; or, growing within the skull, interfere with the functions of the brain. But except in this way, from slowly increasing pressure on important parts, it has no direct influence upon the life of the patient.

The **spongy or cancellous osteoma** is a far more common trouble, is most frequently multiple, and most often first seen before the complete ossification of the skeleton. It is always capped with cartilage, from which it appears to grow, and is nearly always placed close to an epiphysial line (Figs. 348, 349). An osteoma of this class will be seen on section to be composed of open cancellous tissue, which is directly continuous with that of the bone on which it is placed, the cortical tissue at that spot being absent. The removal of such a growth was, for this reason, formerly looked upon as a dangerous procedure, as the operation exposed the medullary tissue to wound infection, which was then the *bête noir* of surgery. It is not uncommon to find a large number of these osteomata, which are then more or less symmetrically distributed at the epiphysial ends of the long bones, especially the femora, the tibiæ, and the humeri. In such cases there is generally, as before stated, a family history of similar growths. They are most frequently seen about the age of puberty or earlier, and one or more may assume large proportions. They generally cease to grow when complete ossification has taken place in the bone to which they are attached. The cap of cartilage then ossifies, and becomes continuous with the cortical bone of the shaft. When subjected to pressure or friction, a bursa is frequently found upon its summit. An osteoma may, however, arise much later in life, and is then, as a rule, solitary. In such circumstances it may grow to a very large size, producing an irregularly lobulated tumour of bone, with a comparatively small peduncle.

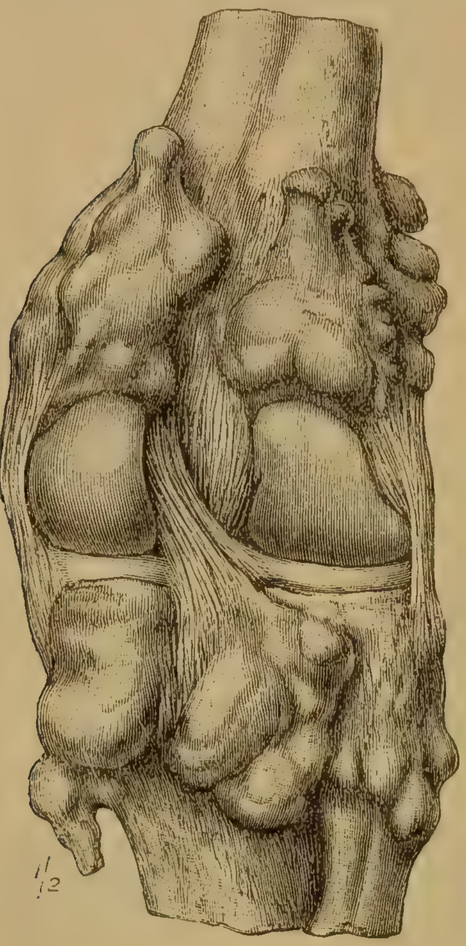

Fig. 349.—View from behind of the Specimen shown in Fig. 348.

From its position, a cancellous osteoma may give rise to a very grave inconvenience. It may interfere with the movements of a joint, or, being placed on the inner side of the knee, prevent the patient from grasping the saddle in riding, or, growing from the inner side of the humerus, project the arm from the side by its contact with the thorax. A patient may thus really require relief from a growth, large or small, which would otherwise be considered quite unimportant.

Treatment.—The ivory or compact osteoma may, if small and situated in a place where it cannot interfere by pressure on important parts, be left alone. On the other hand, it may, from its proximity to a nerve, cause such pain as to necessitate its removal; or, from its growth into the cavity of the orbit, so displace the eye as to render an operation highly desirable. There is no reason at the present day why its removal should not be attempted. Formerly the chisel and saw were the instruments employed, and they were generally unsuccessful. But with the "dental engine," a carefully conducted operation may succeed in removing the whole of the offending part without too great violence to the surrounding tissues. It must, however, be remembered that in the skull the growth may extend into the cranial cavity in the same way as it projects upon the surface. The operation must, therefore, only be undertaken when something more than the mere symmetry of the part or improved personal appearance is desired. The cancellous osteoma can be very easily removed with the chisel, but when there are a large number the surgeon would naturally hesitate before he recommended the removal of them all. He would select only those which were causing inconvenience or much deformity. Most of them are capped with cartilage, and cease to grow with the ossification of this cartilaginous cap. Moreover, those which appear in the adolescent period of life generally undergo this change, with the complete ossification of the skeleton. It is, therefore, undesirable to operate with the belief that more trouble will ensue than at present exists. It must be borne in mind that such osteomata are generally placed about epiphysial lines, and, therefore, in close proximity to joints. Their removal will also necessarily open up the cancellous tissue of bone. For these reasons it is essential that the best antiseptic or aseptic methods of wound treatment be employed.

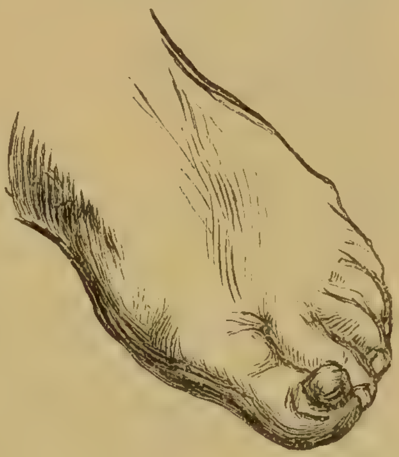

Fig. 350.—Subungual Osteoma in which the Nail has disappeared. (From a cast in St. Thomas's Hospital Museum.)

Of other special outgrowths of bone, the auditory osteomata or exostoses will be considered in Art. XLI., Vol. II., and the ossification of tendons in Art. XXXIV., Vol. II. (*See also* page 455 and Fig. 102.) The subungual osteoma (exostosis) is capped with cartilage, from which it continues to grow like the other cancellous osteomata. It is attached to the ungual phalanx of the big toe, and projects through the matrix of the nail at the tip of the toe

(Fig. 350). The free border of the nail is turned upwards, and the soft parts over the bony growth are frequently ulcerated from friction with the boot. It varies in size, but is scarcely ever allowed to assume large proportions, on account of the pain and inconvenience it causes in walking. It is very easily removed by a sharp spoon or gouge, but care should be taken that all the tissue covering it is also removed. It is otherwise liable to return, probably on account of some portion of the cartilaginous cap being left *in situ*.

Fibromata of bone are very rare except in relation to the jaws and base of skull. In the latter position they have been described as one form of naso-pharyngeal polyp. In other positions fibromata of bone are probably intermuscular or parosteal tumours which have only secondarily become attached to bone. They are recognised by their exceedingly slow rate of growth. The uniform consistence and smooth surface of such a tumour, coupled with the absence of infiltration and freedom from pain, would be additional arguments in favour of a fibroma.

But it must be remembered that some of the more chronic parosteal sarcomata present these features, and if treated in the way in which fibromata are ordinarily treated elsewhere, by removal from the tissues in which they grow, would be almost certain to show their malignancy by an eventual recurrence. If, therefore, a tumour apparently of this nature be removed from bone, it is essential that a microscopical examination be made before allowing the patient to imagine that he is safe without amputation of the limb.

Parosteal lipoma.—For cases of this extremely rare variety of growth attached to bone, the reader is referred to Bland Sutton's work on "Tumours, Innocent and Malignant," 1893, where a series of records in the Transactions of the Pathological Society are alluded to. A deeply-seated fatty tumour is always difficult of diagnosis; and if, further, it be beneath the deep fascia and fixed to bone, it may be impossible to assert its nature without exploration. (*See also* page 451.)

Chondroma of bone.—Some of the cartilaginous outgrowths are mere overgrowths of normal cartilage, and are called *ecchondroses* (page 454). These are especially common in the costal cartilage of young women, and require no treatment. They are also found on the laryngeal cartilages, and may give rise to dangerous symptoms when they project on the inner surface of the larynx. Along the margins of the articular cartilages they are seen as small rounded nodules, or "lip growths," in the osteo-arthritis of mid-life. At the base of the triangular cartilage of the nose they have of late received more attention than they deserve, for unless they produce obstruction they can do no possible harm.

The *true chondroma* of bone arises in the medullary cavity, close to the epiphysial end of a long bone, or between the walls and the periosteum. It is an innocent tumour, and does not return after complete removal. (*See* page 453.) With our present knowledge of histology we may safely assert that if it does return after complete removal, it is almost certainly a chondrifying sarcoma. It is, as a rule, mainly composed of hyaline cartilage, but may have a varying amount of fibrous tissue in its matrix, or may be mixed with other embryonic tissues. In the latter case it is probably a chondrifying sarcoma. Calcification or ossification may take place, and the tumour be converted into a cancellous osteoma, which is, as before described, covered with a cartilaginous cap. Fatty or myxomatous change may also ensue, especially in the larger growths, when the tumour is called a chondro-myxoma, in which distinct cystic cavities are often found.

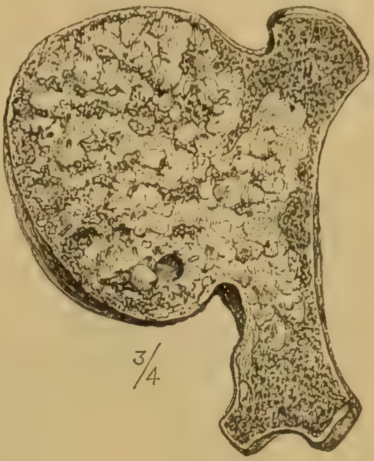

Fig. 351. — Chondroma of Metacarpal Bone, arising from the Medullary Canal, and covered with a thin shell of bone continuous with that of the shaft. (St. Thomas's Hospital Museum, No. 586.)

Situation.—In the hands and feet they are frequently multiple, arising in the medullary tissue of the phalanges and metacarpal or metatarsal bones (Figs. 100, 351). But they are far more frequent in the hands than in the feet. A complete shell or plate of bone may be found upon the surface. When multiple they do not individually grow to any great size, but cause great inconvenience from interference with the functions of the hand or foot (page 453).

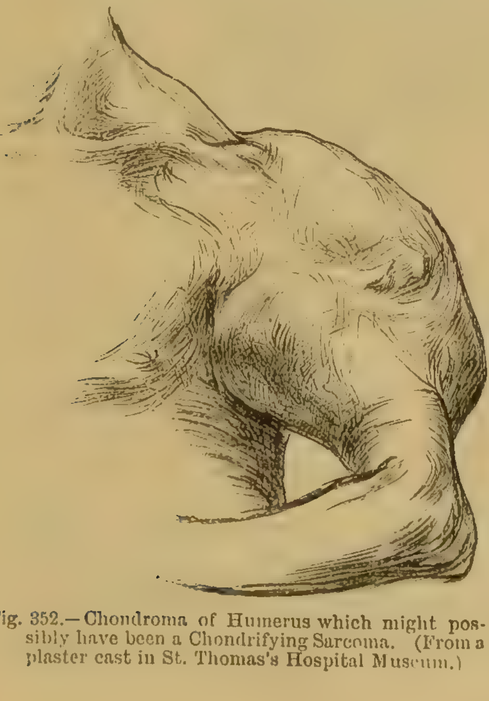

Fig. 352.—Chondroma of Humerus which might possibly have been a Chondrifying Sarcoma. (From a plaster cast in St. Thomas's Hospital Museum.)

When a chondroma arises from one of the larger bones, it is commonly single, and may then assume enormous proportions (Fig. 352). The scapula, pelvis, or the ends of one of the long bones are the usual sites for one of these growths, but scarcely any bone in the skeleton can be said to be free from the possible development of a chondroma. (*See* Fig. 100.) In these situations the tumour generally grows from the periosteum, and not from the medulla; but the bone may be partially absorbed by pressure. Many of these enormous cartilaginous tumours would at the present time be classified as chondrifying sarcomata; and the possibility of this being the case in any large chondroma should be borne in mind by the surgeon in charge of the patient.

Age.—The multiple tumours of the hands first make their appearance in children, about puberty, or in the young adult, and increase slowly in size for some years, and then remain stationary.

The large single chondroma arises much later in life, and continues, as a rule, indefinitely to increase as long as the patient lives, but without any ulterior consequence except that of inconvenience.

Diagnosis.—A cartilaginous tumour is globular in outline, with a slightly lobulated surface. Its consistence is dense, but elastic, and may in parts suggest the presence of fluid beneath. There is no infiltration of the surrounding tissues, the skin and muscles moving freely over the tumour. It merely pushes aside the overlying and adjacent structures, without invading them in its growth. As a general rule, it is perfectly painless, unless some nerve be accidentally stretched or pressed upon. It is inconvenient from its weight and size, and may impair the function of the part involved. But beyond this it has no influence on the duration of life. It is exceedingly slow in its progress, taking many years in attaining any great size. If, on the other hand, a tumour which is thought from its consistence to be cartilaginous, should be more rapid in its growth, some doubt may be entertained as to the possibility of its being a chondrifying sarcoma. The rate of increase is, therefore, the crucial point in diagnosis between a pure chondroma and a chondrifying sarcoma.

The chondromata of the hand, as a rule, give rise to little difficulty in diagnosis. The age of the patient, the particular bones affected, their endosteal origin with plates of bone on the outer surface, their slow growth and freedom from pain and tenderness, make the diagnosis tolerably easy. Tubercular dactylitis, with which at some stages it might be confused, is tender on pressure and accompanied by some inflammatory infiltration; and in the end it softens and discharges, and generally results in a shortened or stunted bone. Syphilitic dactylitis produces a uniform enlargement of the whole bone, with little or no tendency to softening or suppuration. It never produces the globular outline of a chondroma, nor its elastic sensation on pressure. It is, moreover, probably accompanied by a history or other signs of syphilis.

Treatment.—The illustration (Fig. 353) shows how impossible it would be to deal with some cases of multiple chondromata of the hand without sacrificing the part by amputation. And yet in this case it would have been distinctly wrong to have removed his hand, for he could hold a book and materially assist his right hand, which was the less deformed, in his daily occupation. He was eighteen years of age, and had had these tumours since the age of two. Till the left hand was absolutely useless it would have been a misfortune to him to have lost it. In other cases, however, a finger may be removed to increase the usefulness of the whole hand. Occasionally the tumour may be excised from the bone in which it grows.

On two occasions the writer has succeeded in removing a chondroma which projected only on the flexor aspect of a metacarpal bone, leaving the cortex on the dorsum perfectly free. The tumour grew from the medullary cavity, as is usual in such cases, and when removed, a thin strip of cortical bone only was left *in situ* (Fig. 351). No recurrence took place, and the patients recovered, with the perfect use of their hands.

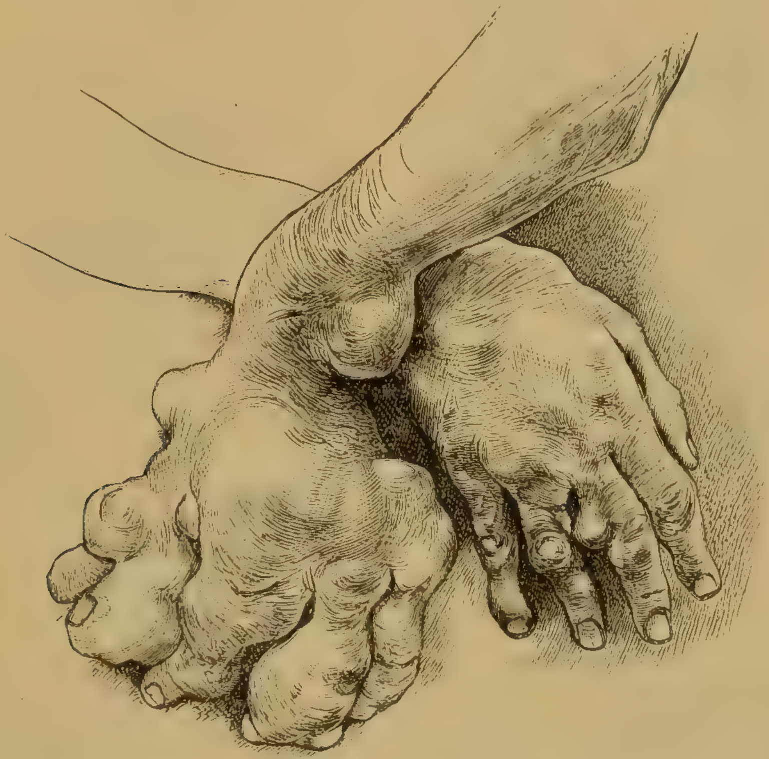

Fig. 353.—Multiple Chondromata of Hands in a Lad of 18. (From a photograph by Burgess.)

With regard to the large single chondroma of the long and flat bones, no definite rules can be laid down. One that is steadily, although slowly, increasing in size should be removed, for no one can estimate the size to which it may grow, and the very magnitude of any subsequent operation may in the end deter the operator from incurring the risk to life from shock. It may sometimes be removed from the bone to which it is attached, or the shaft may be so seriously damaged as to necessitate amputation. An exploratory operation must be done, with permission for the surgeon to act in each case as he thinks best for the patient. If there be any reasonable doubt as to its innocent nature, amputation would, of course, be the safest course to pursue.

Sarcoma.—A primary malignant tumour of bone is always one of the varieties of sarcoma, and must necessarily require amputation or very serious mutilation of the part involved. (*See* page 466.)

As regards the structure, little need be added to what has been said elsewhere (page 466), except that a large proportion of sarcomata of bone are attributed by the patients to some definite injury.

The most important distinction has to be drawn at the very outset between the central and peripheral sarcomata.

Central and peripheral sarcomata.—The former start, as the name indicates, from the medullary tissue of the bone, and may be called *endosteal*. The latter arise, as a rule, in the osteogenetic layer on the surface of bone, and are generally described as *subperiosteal.*

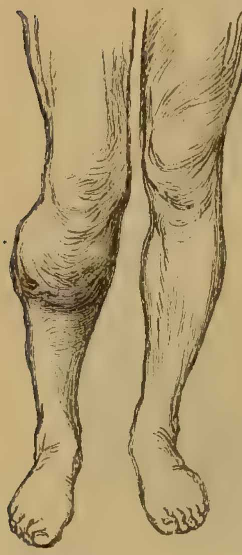

Fig. 354.—Man, aged 35, from whom the Specimen shown in Fig. 355 was removed by Amputation.

There is another and rarer variety of peripheral sarcomata, usually designated as *parosteal.* These arise in the tissue immediately outside the periosteum, and only secondarily invade bone. But, inasmuch as the treatment is almost the same as for the subperiosteal sarcomata, it is desirable to mention them in reference to this subject.

Pathological anatomy.—Both central and peripheral sarcomata may be round-celled, spindle-celled, or a combination of both. In the central alone, if we except an epulis, is a true myeloid sarcoma ever found.

Secondary changes may ensue in them all, the periosteal having an especial tendency to chondrify, ossify, or calcify, whilst the central are more liable to degenerative softening, and may become cystic, fatty, or myxomatous. The central are also often exceedingly vascular: a condition which may at one time lead to hæmorrhages in its substance, and in another to distinct pulsation.

With regard to the coarse anatomy of these sarcomata, the subperiosteal growth shows in most cases a distinct radiation outwards from the bone to which it is fixed. Round these columns or lines ossification or calcification takes place, so that the central portion is often composed of fan-like radiating bone. As the growth increases, the periosteum is farther removed from the bone, so that the area in which the radiation may occur becomes larger and occupies a greater part of the tumour. But bone is never found in the peripheral part of such a sarcoma, being confined to the central portion, if found at all. Soft new growth may also be found outside the periosteum and within the medullary canal. All these points are well shown in Fig. 357.

The central sarcoma starts in the medullary canal, and gradually approaches the surface by destruction and expansion of the bone within which it grows. The periosteum, irritated by its presence, produces new bone, so that a complete shell or plates of bone are always found upon its surface. This is the chief distinguishing feature, both in the clinical and naked-eye examination of the central sarcoma of bone. (*See* Figs. 355, 356.)

The malignancy of sarcomata of bone is shown by the tendency they exhibit towards local recurrence after removal, by the frequent occurrence of deposits of the same growth in distant organs, especially the lungs, and by the occasional invasion of the lymphatic glands. The usual method of dissemination is by the veins; hence the frequency with which the lungs are invaded. On the other hand, the lymphatics are very rarely involved; and when they are so, it is generally on account of the position of the primary growth. In a pelvic sarcoma the glands may be very early invaded by the simple and direct extension of the growth without travelling along lymphatic vessels. But having once gained access to a lymphatic gland, it may be distributed further from gland to gland by lymphatic vessels.

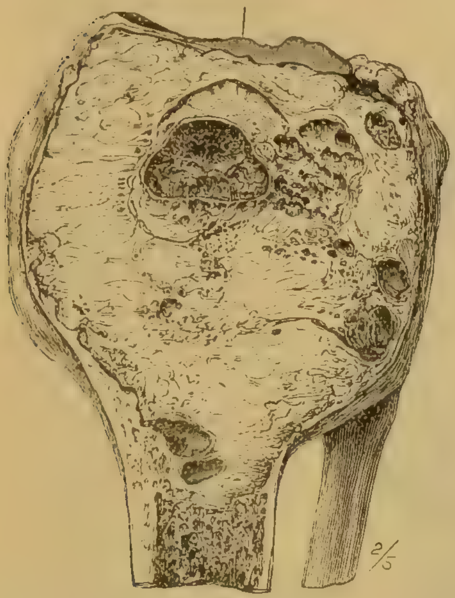

Fig. 355.—Giant-celled Sarcoma of the upper End of a Tibia removed by Amputation from a man, aged 35, under the writer's care. He had noticed in 1878 some swelling after an injury five years before. No recurrence had taken place ten years after amputation. (St. Thomas's Hospital Museum, No. 662.)

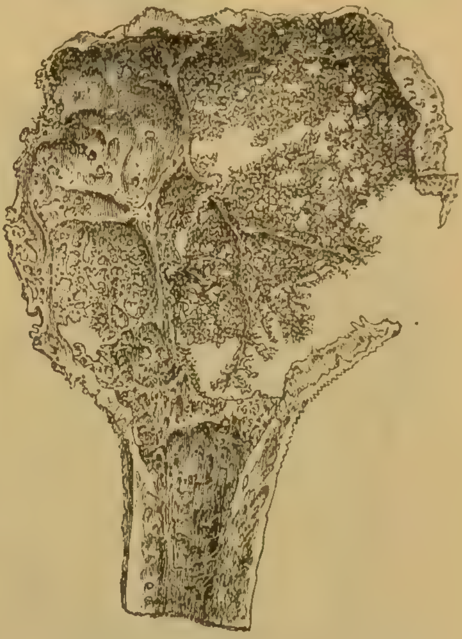

Fig. 356.—The outer Half of preceding Specimen macerated, and showing imperfect shell of new bone. (St. Thomas's Hospital Museum, No. 663.)

The sarcomata of bone differ very much in the degree of malignancy they present. Some invariably return locally, or appear elsewhere, no matter how early in the course of the disease or how far removed from the seat of the tumour the operation is performed.

On the other hand, the myeloid sarcomata so seldom return after removal that they may almost be looked upon as benign growths. But it is necessary here to state that such a tumour for so favourable a prognosis must not only have a few giant cells, but must be mainly composed of such cells to the exclusion of others. The periosteal spindle-celled sarcoma is probably the most malignant, and, speaking in general terms, the periosteal are all rather more malignant than the central sarcomata.

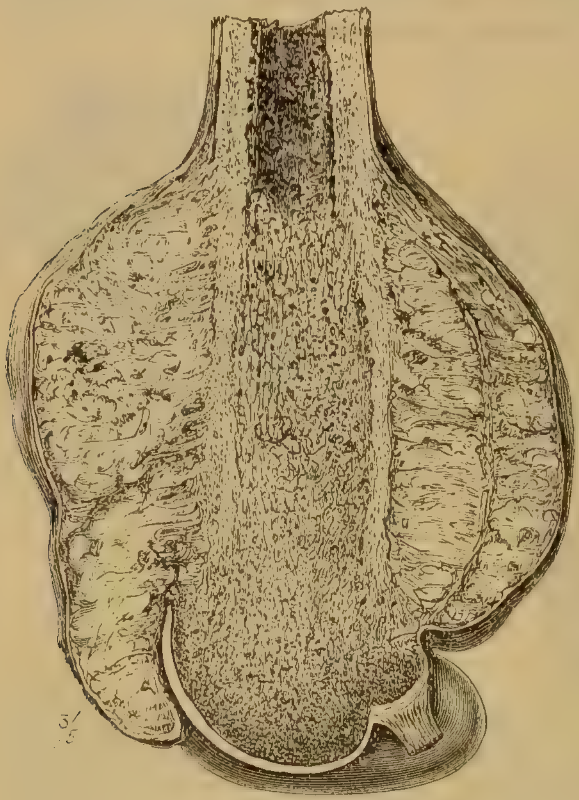

Fig. 357.—Subperiosteal spindle-celled Sarcoma of the lower End of Femur. Posteriorly, the growth has exceeded subperiosteal limits. It has also invaded the medullary canal, and projected into the joint. From a girl under the writer's care in 1886, aged 17, who was alive and well eight years after amputation. (St. Thomas's Hospital Museum, No. 637.)

The favourite seat of both central and peripheral sarcomata, if we exclude the jaws, which are considered in a separate article (*see* pages 932 and 938), is the articular extremities of the long bones; and some bones and some epiphyses are more likely to be attacked than others. For example: the bones of the lower extremities are more frequently attacked than those of the upper, probably on account of their greater liability to injury; and the lower end of the femur and upper end of the tibia are more commonly affected than the other extremities of these same bones, probably for the same reason that the knees are more often exposed to trivial injuries than any other parts of the lower limbs.

The age at which the sarcomata of bone most commonly arise is that of the young adult, but no age can be said to be exempt. The central tumours are more frequent in mid-life, and the subperiosteal in young children, if any distinction be made between the two as to this point of age.

Local signs and diagnosis.—The duration of the disease before coming under observation, and the rapidity with which the tumour has increased, are the first and most important points to which the surgeon directs his attention. As a rule, the more malignant a tumour the more rapidly it grows. The increase in size in the more innocent tumours may be measured by years; whereas in the malignant cases a few weeks or months may suffice to see a tumour double its original size. The rate of growth amongst the sarcomata of bone varies considerably. The subperiosteal increase more rapidly than the endosteal, and these again vary amongst themselves according to their different degrees of malignancy. Some opinion as to prognosis after operation may therefore be formed from this rate of growth before removal.

To distinguish between the central and peripheral sarcomata of bone is the next most important point in the diagnosis and consequent treatment. The central tumours are spherical in outline, whilst the periosteal are long and fusiform in the shaft and pyriform at the extremities of a bone, with the base or broader part directed towards the articulation. This gradual diffusion along the shaft of a long bone, with infiltration of the surrounding tissues, is very characteristic of a periosteal sarcoma, which may be at first localised on one side only of a bone. The central or endosteal sarcoma has a distinct bony capsule or plates of bone on its surface, and if the parts beneath are soft and yielding, will often give on pressure a curious rustling sensation or egg-shell crackling. The periosteal tumour, on the other hand, will be smooth or tuberous on the surface, with no bony plates, but a hard and unyielding centre.

Pulsation is of frequent occurrence in the central tumours of the long bones, especially in the articular extremities. It is also found in some of the multiple periosteal sarcomata of the flat bones. Pulsation over a small area of the upper end of the tibia was in one instance under the writer's observation the only symptom present of an endosteal sarcoma. There was no swelling and no expansion of bone. When submitted to operation, the interior of the head of the tibia was found to be replaced by a soft, almost diffluent, marrow-coloured growth, which on microscopical examination proved to be a giant-celled sarcoma.

Spontaneous fracture may occur in either the central or peripheral sarcomata; in the latter it depends upon the occurrence of an extension of the growth through the Haversian canals to the medulla. Occasionally it has been the first indication of the disease, when the tumour has then grown with very great rapidity.

Pain is almost always a prominent symptom, and is often the means by which the patient's attention is drawn to the presence of a local swelling. It is sometimes so severe as to induce the patient to submit to what might on other grounds be considered as an operation of doubtful utility. On the other hand, it may be entirely absent in a central myeloid sarcoma.

The *size* to which a sarcoma of bone may grow varies enormously, and no opinion as to its nature can be formed from this circumstance alone. In one case, whilst still quite small, general systemic infection may have already taken place, whilst in another the very largest tumour may be removed without recurrence of the disease.

The *diagnosis* of a sarcoma of bone from a simple tumour, such as a chondroma, is generally easily made from the history of its rate of growth, its varying consistence in different parts, and the presence of pain. A chondrifying sarcoma may, however, present some difficulty, and the rate of growth may be the only means of arriving at a definite conclusion. (*See* page 912.)

In very rare and exceptional circumstances some forms of inflammation of bone may be mistaken for sarcoma. (*See* page 887.)

A pulsating tumour of bone may at times present very great difficulty in diagnosis from an aneurysm. This is especially the case about the pelvis or groin or in the popliteal space. The points of importance would be the exact situation of the growth, which might be out of the direct line of the main vessel, the presence of bony plates upon the surface, or the ill-defined outline in the periosteal growth, the less impulsive character of the pulsation, and unaltered condition of the arteries below. But the most experienced surgeons have ligatured arteries, on the supposition that the swelling was an aneurysm when the subsequent course proved it to be sarcoma of bone.

Treatment. 1. *Subperiosteal sarcoma.*—Amputation at the earliest possible moment should be done for the most malignant of the sarcomata of bone : namely, the subperiosteal. At one time it was thought necessary to remove the whole bone from which the tumour arose : *e.g.* by amputation at the hip-joint in a case of subperiosteal sarcoma of lower end of the femur. The mortality from the operation alone was consequently very high, and the patient, if he lived, was still found to be exposed to the same risk of dissemination of the growth in the lungs as if he had been submitted to an amputation at a less dangerous spot. Provided there is sufficient room for performing amputation through tissues free from infiltration, there is no reason, with our more recent experience, for a removal so high as at the joint above the bone involved. There should, however, be plenty of room, and skin-flaps with circular division of muscles at the spot where the bone is sawn would be safer than any variety of muscular flap, for the periosteal growth is liable to spread upwards between the fibres of any muscle which is involved at the primary seat of infection. The sawn section of bone also, in such a case, should be carefully inspected, to see that there is no medullary growth from extension through the Haversian canals. With these precautions, an amputation may be done through the bone which is the seat of a subperiosteal sarcoma. If the disease be not eradicated, it is as likely to appear in the lungs as in the stump, provided that the amputation has been done at a fair distance from the tumour.

Occasionally the pain may be so severe and distressing as to induce the patient to submit to an amputation, even when he is assured that little hope can be entertained of a permanent relief from the disease.

Parosteal sarcomata, although less malignant, must be treated on almost the same lines as the subperiosteal, as they infiltrate the surrounding tissues, and cannot be dissected off the bones without the danger of local recurrence (Figs. 358, 359).

2. *Endosteal or central sarcoma.*—Being, as a rule, much less malignant than the subperiosteal growth, re-section of the portion of bone involved, or an amputation close to the disease, may fairly be entertained. If there be good reason to think, from its comparatively slow growth and pulsation, that it is a giant-celled sarcoma, re-section should always be done, provided that a useful limb will be left behind. This is certainly the case in the bones of the fore-arm, but in the femur and tibia some doubt may reasonably be felt as to whether the patient will not be better off after an amputation than after re-section, although by either method the disease may be eradicated. If the central growth proves after excision to be a round or spindle-celled sarcoma, the case must be most carefully watched, as local recurrence is much more likely to take place than after a giant-celled sarcoma. Amputation well above the limits of the disease must be at once performed on the first suspicion of such a recurrence of growth.

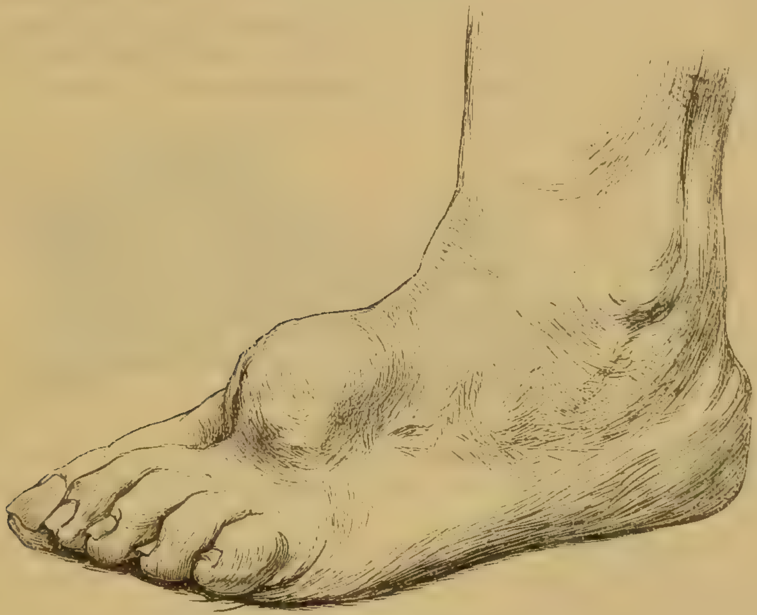

Fig. 358.—Parosteal Sarcoma from Foot of a Woman aged 40, under the writer's care. Four years' history of growth. (From a photograph.)

Sarcoma of individual bones. *Tibia.*—Periosteal sarcoma in the lower third of this bone will require amputation through the knee-joint by one of the various methods, at the option of the surgeon. In exceptional circumstances the amputation might be successfully accomplished just below the knee, through the tibia, but great care must be taken that the soft parts are really free from infiltration. If the disease be situated in the upper third of tibia, the amputation must be done through the lower third or middle of thigh, according to the extent of growth. An endosteal sarcoma of tibia may be removed by amputation, just above the disease. A re-section would leave a useless limb.

Fibula.—A periosteal sarcoma should be removed by amputation on the same lines as for the tibia.

An endosteal sarcoma of the fibula may fairly be submitted to re-section, and the case carefully watched.

Femur.—In the lower third of the femur a subperiosteal sarcoma would require amputation as high up towards the hip-joint as safety to the patient from shock would dictate. Skin-flaps and sawing through the neck of femur or trochanters would probably be as serviceable, so far as local recurrence is concerned, as disarticulation at the hip-joint. *See* Fig. 357, where success was obtained by a much lower amputation.

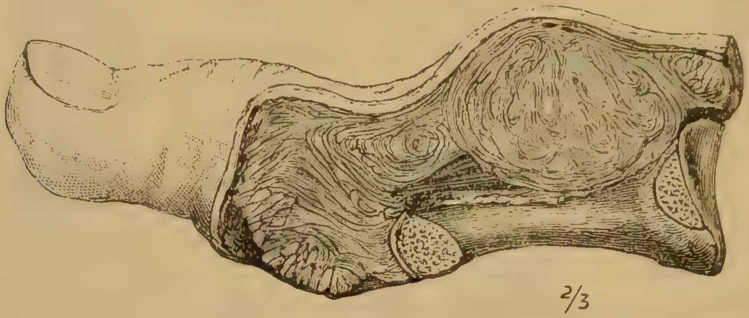

Fig. 359.—Parosteal spindle-celled Sarcoma between the first and second metatarsal bones. From the case shown in Fig. 358. Removed by Lisfranc's amputation, 1890. No recurrence four years afterwards. (St. Thomas's Hospital Museum, No. 2589.)

In the middle of the thigh nothing less than amputation at the hip-joint, with short skin-flaps, would be of any avail. And even this can only be successful quite early in the disease, or where it appears not to have spread rapidly along the shaft. A subperiosteal sarcoma in the upper end of the femur must be regarded as hopeless from an operator's point of view.

An endosteal sarcoma of the femur would require amputation immediately above the disease. In the head and neck of femur—where, however, it is exceptionally rare—re-section might be thought of on account of the dangers of amputation through the hip-joint. If there be a doubt—as there well might be—as to the nature of the tumour on account of its distance from the surface, an exploration might first be made. If found to be an endosteal growth, re-section would, perhaps, be the best treatment, especially if there were reasons to think that the case was one of giant-celled sarcoma.

Radius and ulna.—Periosteal sarcomata of the lower thirds of these bones must be treated by amputation just below or just above the elbow-joint, according to the extent to which the growth has invaded the soft parts of the fore-arm. In the upper third amputation through the middle of the humerus would probably be necessary, so as to be quite clear of the muscles arising from the condyles.

Central sarcomata of both radius and ulna should be treated by re-section. The writer has successfully removed both the upper and lower end of the radius for myeloid sarcomata, leaving in each case a useful hand and fore-arm.*

Humerus.—Disarticulation at the shoulder-joint or amputation through the surgical neck will probably be required for a subperiosteal sarcoma of the middle and lower thirds of the humerus. For the same disease in the upper third of the bone, the removal of the upper limb, with the scapula and a portion of the clavicle, may be the only effectual way of relieving the patient.

An endosteal sarcoma of the humerus should, if possible, be treated by re-section, for even if no union can be obtained after operation, some mechanical contrivance will render the hand serviceable.

Clavicle.—The circumstances in which this bone may be successfully removed for periosteal sarcomata are very rare. Bland Sutton† has removed the acromial end for a giant-celled sarcoma with success. (*See* Fig. 115.)

Of the flat bones, the *scapula* has been frequently removed in whole or in part for sarcoma. The lower segment especially can be successfully excised; but the removal of the whole bone, and with it the arm and outer half of clavicle, which would generally be necessary, is a formidable operation, almost comparable in its results with disarticulation at the hip. The new growth is also nearly certain to reappear in the lungs.

Portions of the *pelvis* may at times, in quite the early stages of the disease, be capable of removal, but the possibility of doing this will always be exceptionally rare.

If either the skull, sternum, or pelvic bones are invaded by sarcomatous new growth, it will generally be found, on careful examination, that there is more than one tumour: a condition which would quite preclude any operative interference. This is especially the case in the subperiosteal sarcomata of these bones. And more than one bone is then often simultaneously affected, when a wide and rapid dissemination may generally be expected.

The **prognosis** of sarcomata of bone may be gathered from what has been previously said as to their malignancy. But after the tumour has been removed and examined, a much safer opinion can be given as to the probable result. It would be wise, therefore, to withhold a decided opinion till the necessary operation has been performed.

Carcinoma of bone.—As a primary disease, it is almost unknown; in fact, it is believed by many to be impossible, as there are no epithelial tissues from which it could arise. Secondary growths are not uncommon, especially after scirrhus of the breast. In these cases carcinoma may develop in the cancellous tissues of any of the bones, and may give rise to spontaneous fracture or to persistent pain. In the latter the *post-mortem* examination may reveal secondary growths in the bodies of the vertebræ.

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* Clin. Soc. Trans., vol. xxvii., 1894. † Clin. Soc. Trans., vol. xxiv., 1891.

Epithelioma frequently involves bone by direct extension from the primary growth, especially in the case of the lip and tongue.

Cystic tumours of bone.—Those which are connected with the teeth will be found described under Diseases of the Jaws (Art. XXXI., page 935).

Both *sarcoma* and *chondroma* of bone may contain cavities filled with serous and bloody fluid. The large majority of cystic tumours of bone occur under one or other of these headings, and the reader must be referred to the preceding pages for an account of their origin.

Hydatids may also—although very rarely—produce cystic cavities in bone. Several specimens are preserved in the museums of the Royal College of Surgeons, St. Thomas's Hospital, and Guy's Hospital. The bone becomes thin and expanded, and gives rise to the symptoms of a central cystic tumour. If tapped, a diagnosis may be made from the examination of the fluid. But a spontaneous fracture is sometimes the first indication of the disease; and an operation for re-section has demonstrated its nature by the escape of hydatids. The treatment would consist in the removal of the cysts, leaving only, perhaps, a shell of bone. But under modern conditions of wound treatment this shell may be expected to fill with healthy granulations. Amputation would be rarely called for. (*See also* page 321.)

XXXI. DISEASES OF THE JAWS.

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In this section it will be convenient not only to consider diseases of the jaws, but also the affections of those structures so closely associated with them, namely, the gums and teeth.

Diseases of the gums.—The muco-periosteum in immediate relation with the alveolar borders of the jaws is liable to a variety of diseases.

Overgrowth.—This affection, often spoken of as "hypertrophy," consists of a general increase in thickness of the gums, which in some cases is so marked as almost to obscure the teeth. The mucous membrane, in severe examples, forms a vascular fold on the lingual and labial aspects of the teeth, especially the incisors and canines. It occurs in children, but the extreme cases are met with in young adults. The cause is quite obscure.

Treatment.—Scarification with a scalpel or Paquelin's cautery after the parts have been cocainised. When the gum hangs in pendulous flaps, the redundant tissue may be excised.

Lead staining.—In chronic plumbism a blue line exists at the dental margins of the gums, due to a deposit of sulphide of lead. The metal, contained in the blood as a soluble salt, combines with the sulphur of the sulphuretted hydrogen set free by decomposition of food particles in the mouth.

Treatment.—The general treatment is to determine the cause of the plumbism, and administer iodide of potassium with the object of dissolving the lead deposited in the tissues. Quinine and iron are useful. Local treatment, such as the removal or stopping of carious teeth and the regular use of the toothbrush, is indicated.

Spongy gums.—The gums are liable to become swollen and engorged from a variety of causes, general as well as local. Among the more important general causes must be included scurvy, syphilis, and chronic mercurialism. Local causes are stomatitis and alveolar abscess.

Spongy gums are often very tender, mastication being a distressing process; they bleed freely and exhale a fœtid odour.

Treatment.—Remove the cause; improve the general health by tonics and a liberal diet; keep the mouth clean by the frequent use of astringent washes.

Alveolar abscess.—In the great majority of cases abscesses in connection with the alveolar borders of the jaws arise in the sockets of teeth, and are due to septic changes in the pulps of teeth, the roots of carious, or dead teeth.

The simplest form of alveolar abscess is that known as "gum-boil," which is a small collection of pus immediately beneath the gum, and is almost invariably associated with a carious or dead tooth.

The deep variety of alveolar abscess begins around the root of a tooth. The course of events in many cases is the following:—A portion of the crown or neck of a tooth is slowly destroyed by caries, or by accident, and the pulp is exposed: septic inflammation follows and pus forms in the pulp chamber. The septic changes extend along the root or roots and establish suppuration in the socket of the tooth, giving rise almost invariably to "toothache," which persists until the pus finds a way of escape. The pus may well up around the neck of the tooth, or so expand the alveolus in relation with it as to perforate the thin shell of bone, and burrow between the alveolus and muco-periosteum; it may then point in a variety of directions, according to the situation of the initial focus of suppuration.

In many cases it points at that portion of the gum immediately in relation with the tooth. Alveolar abscess in connection with a lateral incisor, second bicuspid, or first molar of the maxillary (upper) set, will point in the roof of the mouth, or may even extend backwards to the soft palate. An abscess connected with the central maxillary incisor will sometimes open on the floor of the nasal fossa. Pus connected with the second bicuspid, the first or second molar, may open into the antrum; when connected with any of the maxillary (upper) teeth it may open in the roof of the mouth, or any part of the cheek, and it has been known to point at the inner canthus or the orbit.

An alveolar abscess connected with the mandibular (lower) teeth usually points on the gum or mucous membrane between the cheek and gum. In a severe case, it will burrow beneath the mucous membrane and gain the connective tissue beneath the skin of the cheek and buccinator muscle. Pus will sometimes travel long distances and point on the cheek, in the submaxillary region, at the chin, or even as low as the clavicle. Alveolar abscess pointing at the chin is usually associated with the mandibular (lower) incisors.

The signs of an alveolar abscess are, in the early stage, toothache: as the suppuration becomes established, pain and swelling of the jaw, furred tongue, raised temperature and other febrile symptoms, and enlargement of the adjacent lymph glands, and, in exceptional instances, pyæmia and death.

Treatment.—Ascertain the cause, searching particularly for carious teeth, dead teeth, or hidden roots left behind during extraction.

If the tooth is hopelessly destroyed, or is reduced to its roots, it may be extracted. In very many cases a free incision into an alveolar abscess will relieve pain, the sinus will often close, and after the inflammation subsides a dental surgeon will treat, successfully stop, and thus preserve the tooth. When a troublesome sinus persists after the drainage of an alveolar abscess, the offending tooth should be extracted.

Many persistent sinuses are due to a small fragment of root left in the socket after extraction of the tooth, or to a small piece of necrosed bone.

When an abscess forms after a tooth has been "filled" it is necessary to extract it, unless the patient is willing to submit to drilling of the tooth. This can be carried out under gas, and may save the tooth. An abscess at the root of a dead tooth is occasionally treated in this manner.

Whenever possible, incisions into alveolar abscesses should be made in the mouth.

Abscess (empyema) of the antrum.—A collection of pus within the antrum is in nearly all cases secondary to suppuration connected with teeth, especially the second bicuspids, and the first and second molars of the maxillary set. Inflammation, and occasionally suppuration, of the antrum may be due to extension from adjoining cavities. An antral abscess in some cases follows injury to its walls.

The presence of pus within the antrum is indicated by local pain, which is sometimes very acute, but more often of a dull aching character. There is often tenderness, and the gum, as well as the mucous membrane of the nasal fossa of the affected side, is swollen, inflamed, and tender. Febrile symptoms attend the formation of pus, which sometimes declares its presence by trickling from the nostril when the head is bent forwards, or into the pharynx when the patient is lying on the back.

Occasionally, in acute cases, the overlying skin is inflamed, and epiphora, due to implication of the nasal duct, is by no means rare.

Pus may collect in the antrum, and give rise to very few symptoms, and these may be ambiguous. This condition is known as "latent empyema of the antrum."

Exceptionally, suppuration in the antrum may be due to a fungus —*e.g. aspergillus fumigatus.*

Diagnosis.—In a large proportion of the cases in which the symptoms indicate a collection of pus within the antrum as their most probable cause, it is by no means an easy matter to come to a certain diagnosis. This is due to the fact that the signs relied upon as indicative of an antral abscess are simulated by inflammation of adjacent cavities—for example, the nasal fossa, frontal sinus, and even the naso-pharynx. It is also important to remember that it is sometimes extremely difficult to decide between an antral abscess and a tumour growing in this cavity before it has begun to expand its walls.

There are two important signs of pus in the antrum. Of these the most certain is the periodical discharge of pus from the nose, in conjunction with the symptoms already detailed. The second sign will be found on examining the upper bicuspid and molar teeth, and if one of these be found with an exposed pulp, or recently filled with stopping, or dead, or reduced to a stump, it should be very carefully tested and extracted. In many cases thus treated pus will follow the withdrawal of the tooth.

Failing this, the surgeon may resort to what is known as transillumination of the antrum. A small but powerful electric lamp is introduced into the mouth of the patient (sitting in a dark room, or with his head covered with a dark cloth). The lips are then closed, and the current turned on. If the antrum is clear, a translucent curved band of light immediately appears beneath each lower eyelid. Should the cavity contain pus, this band of illumination is absent. There are many fallacies in this test. When the translucent bands appear, it is quite certain that no pus is present. Should the antrum remain opaque (and this happens with some healthy antra), the opacity may be due to a tumour, or to pus; and it is quite possible for an antrum to be occupied with fluid (not pus or blood), and yet remain translucent. Transillumination, though limited in its application, is nevertheless useful.

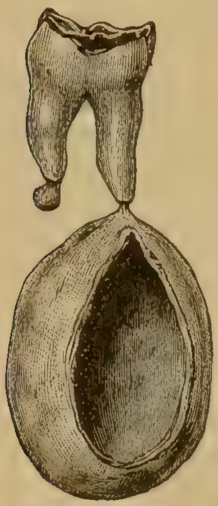

Fig. 360.—Pus Sac at Root of a lower Molar. Nat. size. (Trans. Odonto. Soc., 1894.)

Another method of determining the presence of pus is to cocainise the nasal mucous membrane over a limited area immediately under the anterior part of the inferior turbinal, and then tap the antrum with an exploring syringe. This method is useful in cases of latent empyema of the antrum.

Pus sacs.—In connection with alveolar and antral abscesses, it is necessary to describe the pus sacs sometimes met with at the roots of carious teeth.

It occasionally happens in extracting carious teeth, or stumps, to find at the apex of the root a small fibrous sac which may be scarcely larger than the head of a pin, or it may be as big as a bantam's egg; the average size and shape of the sac are those of an apple-pip. The cavity of the sac is usually filled with pus or fluid rich in cholesterine.

Pus sacs occur in connection with the roots of the mandibular and maxillary teeth; they sometimes attain a large size in the upper jaw when they invade the antrum, and it is probable that some of these cysts are of sufficient size to simulate an abscess or a cyst of that cavity. Pus sacs in connection with the mandible have been observed as large as a pigeon's egg (Fig. 360). These pus sacs are chronic abscesses, and the sac is formed of fibrous tissue lined with granulations such as usually beset the wall of an abscess.

Treatment.—This is the same as that used for abscesses in general—evacuation through the most dependent part. When the suppuration is due to a carious tooth, extraction of the tooth and the enlargement of its aperture of communication with the antrum will afford good drainage. It may be necessary from time to time, when the discharges are offensive and abundant, to irrigate the cavity with some mild antiseptic solution.

When the teeth are sound the cavity may be perforated with a bone-drill through the canine fossa, and drainage, assisted with free irrigation, resorted to. As soon as free drainage is established, many cases do well and rapidly close; in others, a sinus persists for many months. In such it is well to make a thorough search for dead bone, pieces of teeth, and foreign bodies within the antrum.

Diseases of the bone.—The jaws in common with other bones are liable to periostitis and necrosis.

Acute periosteal abscess.—The most frequent cause of this affection is injury, and it is common as an extension of alveolar abscesses.

Periosteal inflammation and abscess of the jaws are accompanied by the usual signs of periostitis, such as a local swelling attended with severe pain and swelling. General febrile symptoms are markedly manifest.

Treatment.—Immediate incision into the inflamed and swollen tissues, made, whenever practicable, through the mucous membrane, so as to avoid external scars; the incision should extend quite down to the bone, or there will be great risk of necrosis and a troublesome sinus; all these complications may often be avoided by prompt and free incision. When the case has been allowed to run its own course and destroy the underlying bone, the sinus must be maintained until the necrosed bone separates. The sinus is then dilated or slit up and the dead bone removed.

Necrosis.—This condition may follow injury, alveolar abscess, and syphilis, excessive use of mercury, irritation from the fumes of phosphorus, and tubercle. Extensive necrosis of the jaws occurs as a sequel to the acute exanthemata and typhoid fever. (*See* page 884.)

In cases where necrosis follows an alveolar abscess, the amount of bone which dies is usually very limited, and in most cases involves merely a superficial lamina of bone. Where it occurs as a sequel to scarlet or typhoid fever, the greater part of the maxilla or mandible may die. In such cases the suppuration is very profuse, and the necrosed jaw will be found quite loose, merely lodged in a bag of muco-periosteum. It is a curious fact that when the body, alveolar and palatine processes of the upper jaw necrose, although the periosteum remains, new bone is not produced. On the other hand, the whole of the body of the mandible and a large portion of its rami have been destroyed, and a new lower jaw has been formed by the periosteum, as the shaft of a long bone would be produced under similar conditions. It is said, however, that a mandible reproduced under such conditions is not permanent, but slowly re-absorbs. This is not true in all cases. In at least one instance a boy was able to crack a nut with a mandible of this kind ten years after its reproduction. (Warren Tay.)

Phosphorus necrosis.—This curious and now rare disease arises from the effects of the fumes of phosphorus on the exposed pulps of teeth, and then upon the bone of the jaws. The rarity of this disease is due to the compulsory (Factories Act) examination of operatives, leading to the rejection of those with dental caries, and the employment of red amorphous phosphorus in the place of the yellow, easily oxydisable form of this element. (*See* page 884.)

The three chief features of this disease are : its insidiousness, the extent to which it may involve the jaws, and, coincident with the destruction of the bone, the formation of a sheath or shell of porous osseous material. The soft tissues overlying the jaws are swollen, spongy, and bleed freely on slight provocation. As soon as the dead bone is removed, the investing sheath of porous bone is, as a rule, quickly absorbed. The greenish colour of the necrosed fragments is often a striking feature.

Although in the majority of cases phosphorus necrosis is met with in those employed in the manufacture of lucifer matches, it may occur in other circumstances ; thus, in one instance, a man suffered from this variety of necrosis in consequence of sucking habitually a piece of ginger which he carried in his waistcoat pocket with lucifer matches. In exceptional instances, fatal pyæmia and abscess of the brain have supervened on phosphorus necrosis.

Treatment.—Necrosis of the jaws is treated on the principles applicable to bones in general. As a rule, wait until the dead bone has separated before attempting its removal. During the waiting period means must be taken to allow of free escape of pus, and the patient strictly enjoined against swallowing it. The mouth is kept clean by the free use of lotions containing tincture of myrrh, or very weak solutions of Condy's fluid. The last preparation produces a superficial discoloration of the teeth, but it is easily removed by the use of a rotatory brush. Stumps of teeth in relation with the diseased segment of the jaw should be removed.

Affections of the jaws—such as alveolar abscess or caries, and many cases of necrosis—would be prevented if individuals would seek more timely aid from the dental surgeon. As soon as dental caries is detected its course, under proper treatment, will not only be arrested, but the pulp of the teeth may be saved exposure ; and even if exposed, the prompt devitalisation of a tooth, by destroying the pulp by means of arsenic, etc., would often prevent the formation of alveolar abscess.

Pyorrhœa alveolaris.—This disease consists of slow removal of the alveolus, and gradual loosening of the teeth, accompanied by a muco-purulent discharge from the free edges of the gums.

The disease begins with a very slight degree of inflammation of the gum and alveolar-dental periosteum, accompanied in the majority of cases by a deposit of tartar on the denuded surfaces of the teeth beneath the level of the gum. This tartar sometimes occurs in small nodules, or it may form large masses on the roots.

Pyorrhœa alveolaris may be limited to a few teeth, but in many instances it attacks the whole set, and renders a mouth edentulous.

There is reason to believe that this malady may be constitutional in origin. Other careful observers deny this and maintain that it is a local affection.

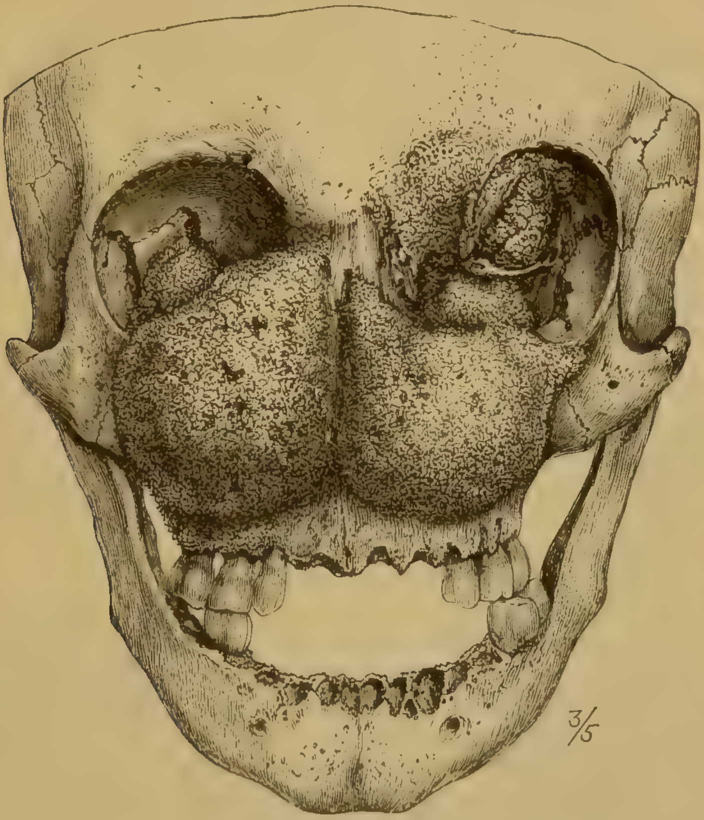

Fig. 361.—Leontiasis Ossea. (St. Thomas s Hospital Museum.)

Treatment.—This is very unsatisfactory. It consists in removing all tartar from the teeth. The swollen margins of the gums should be swabbed with a solution of perchloride of mercury (1 in 5,000). Some writers have reported benefit from the use of pure carbolic acid or powdered sulphate of copper, applied to the groove between the teeth and the gum by means of a pad of cotton-wool on the end of a probe. In severe cases the only treatment of any avail is to anticipate the effects of the disease by wholesale extraction of the teeth.

Leontiasis ossea.—This disease, remarkable for the hideous manner in which it deforms the skull, is fortunately rare. Its effects are confined almost entirely to the bones of the skull. It may attack the cranial bones, or be limited to a few bones—such as the maxillæ, the frontal, and ethmoid.

When the disease attacks the skull generally, the bones thicken, and encroach upon the cranial cavities; and in the case of the orbits, nasal fossæ, ethmoidal cells, sphenoidal sinuses, and the antra, obliterates them. The nerve foramina of the skull are narrowed, and the nerves traversing them compressed.

Of all the skull bones leontiasis ossea seems especially prone to affect the maxillæ, and the leading features of the disease are displayed in Figs. 361, 362. All parts of the bone are thick and dense, the antrum is obliterated, and the body of the bone, transformed into a solid nodular boss of dense bone, blocking up the nasal fossæ and encroaching on the orbits, displace the eyeballs. Sir Astley Cooper stated that this woman (Fig. 362) presented a hideous appearance, even for Billingsgate, where she obtained her living as a fishwoman.

The disease begins during childhood and, as a rule, progresses with extreme slowness, and rarely attracts attention until the protuberant bosses form in the maxillæ. Gradually the nerves of special sense are nipped and their functions destroyed. The complete history of six patients is known. In one (Forcade's) the disease was noticed at the age of twelve years, and the patient died at forty-five. The best known British case is that reported by Bickersteth. (*See* page 905.)

From observations on a recent case, and a study of the available records of known specimens, especially the famous Jadelot cranium and similar specimens from mammals (monkeys, bears, and lions), the disease appears to be a manifestation of rickets.

Treatment.—Nothing is of any avail in staying the course of the disease. When the bosses on the maxillæ are large enough to distort the cheeks, they may be easily removed with a chisel.

Acromegaly.—The jaws, especially the mandible, are conspicuously affected in this extraordinary disease, and produce the profound alteration in the physiognomy which gives such a marked character to this affection. (*See* page 904.)

Actinomycosis.—This disease may attack any part of the alimentary tract, and has been on several occasions observed in the jaws, to which it gains access by means of carious teeth. The fungus (actinomyces) has been detected in the cavities of carious teeth, which seem to offer favourable conditions for its development. There is reason to believe that the fungus may be present and cause no harm. Occasionally, however, it gives rise to inflammatory troubles, which in their beginning simulate an alveolar abscess or a sarcoma. The swelling rapidly increases, involves surrounding tissues, and implicates the skin. Sinuses form and discharge serous or purulent fluid containing small yellow grains, which contain cluster of actinomyces, and lead to the identification of the disease. The adjacent lymph glands enlarge. Secondary foci of the disease may develop in the lungs or intestine. (*See* pages 49 and 314.)

The disease is very grave, and may terminate in death within a few months. Occasionally it runs a more chronic course (one to two years).

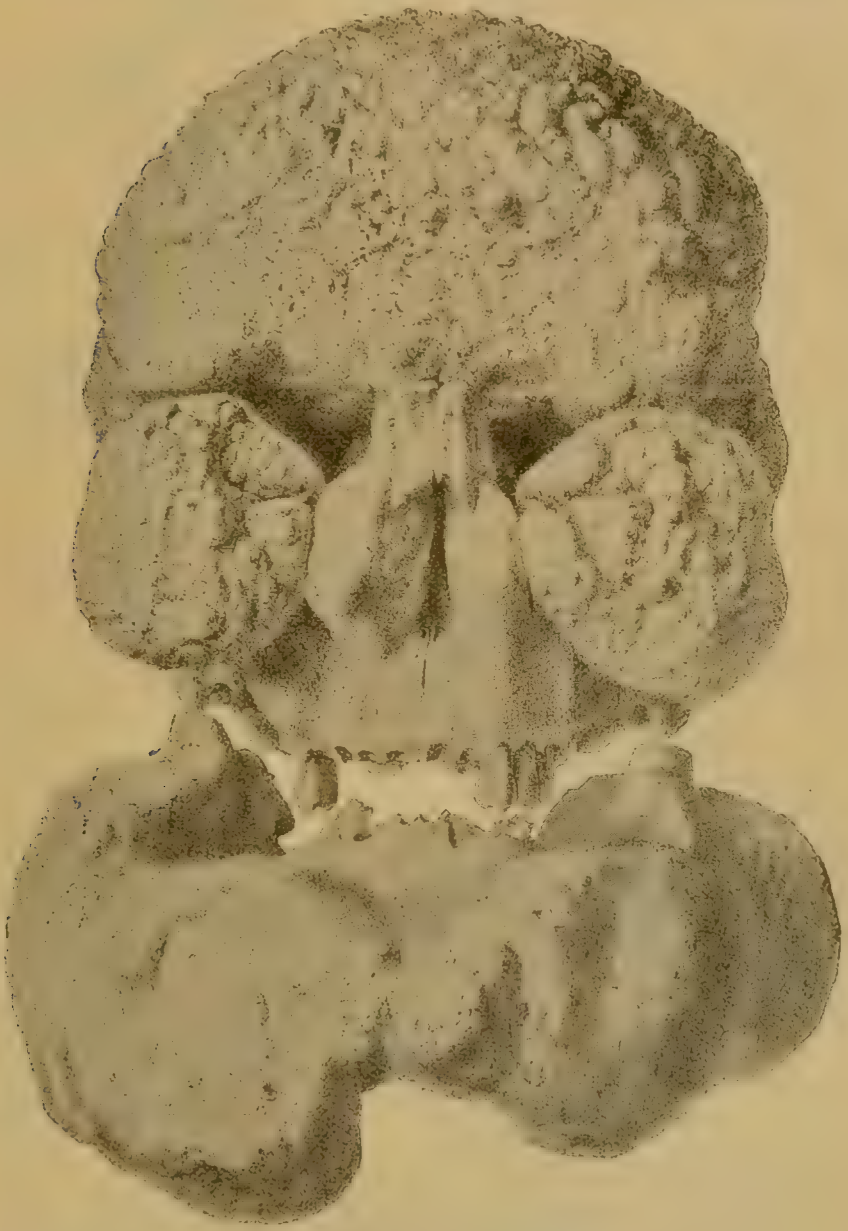

Fig. 362.—Leontiasis Ossea. (After Bickersteth.)

Treatment.—This to be effectual must be radical: prompt removal of the diseased tissues by scalpel and sharp spoon. To be of any real service, these measures must be adopted in the earliest stages of the disease, before there is wide infiltration of tissues, or secondary foci in internal organs.

TUMOURS OF THE JAWS.

These will be considered under four headings, according as they arise from (1) bone, (2) gums, (3) teeth, or occupy the (4) antrum.

1. Tumours arising in bone and periosteum of the jaws.

—These belong to the same genera as those common to bones in general, and include osteomata and sarcomata.

Osteomata.—These are somewhat rare tumours, more frequent on the mandible than the maxilla, and are usually sessile (Fig. 363).

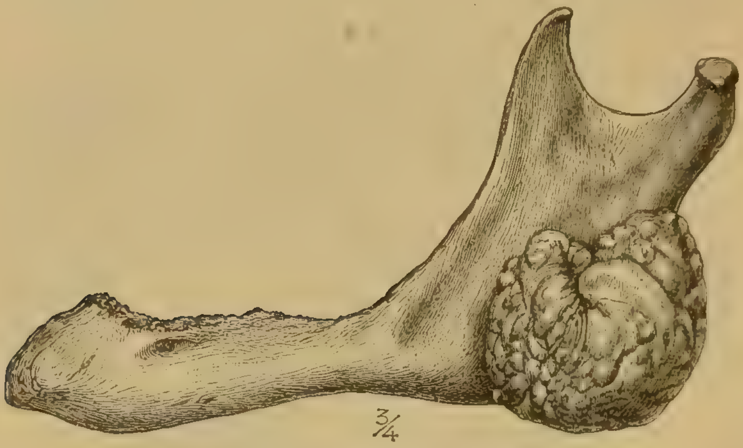

Fig. 363.– Osteoma of Mandible. (St. George's Hospital Museum.)

Many cases, formerly described as osteomata or exostoses of the jaws, were examples of leontiasis ossea, shells of bone due to ossification of periosteal sarcomata, and large odontomes. Localised outgrowths of bones, often described as exostoses, are not infrequent in connection with the nasal process of the maxilla. The affection is sometimes bilateral (Fig. 364) and its cause is obscure. Small irregular osseous prominences often occur along the alveolar borders of the maxilla and mandible. (*See* page 907.)

Sarcomata.—These occur at any age, even in infants a few months old. They usually spring from the periosteum or mucoperiosteum, and are equally common in both jaws. They belong to the round and spindle-celled species; and, as a rule, grow with great rapidity, recur quickly after removal, and attain to great proportions (Fig. 365).

The spindle-celled species, as is usual with these tumours in other situations, often contain tracts of hyaline cartilage; hence many tumours, erroneously described by previous surgical writers as enchondromata of the jaws, belong to this species of sarcomata.

Sarcomata springing from the gums usually project into the space between the cheek and the teeth; when large, they produce great displacement of the teeth and marked alterations in the conformation of the alveolar borders of the jaws. When very large they will protrude beyond the lips. These tumours were formerly called "malignant epulides." (*See also* page 914.)

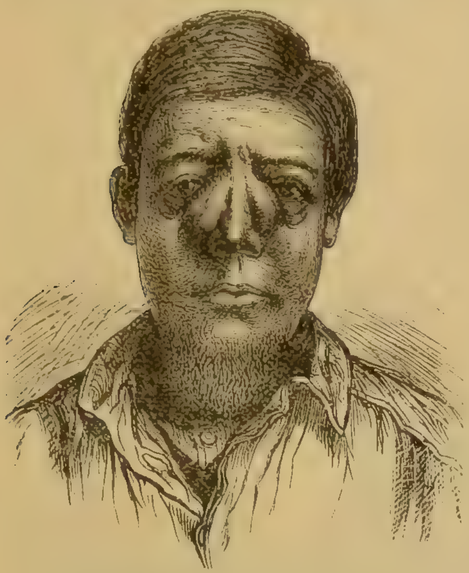
Fig. 364.—Bilateral Exostoses of the Nasal Process of the Maxilla. (After Hutchinson.)

Periosteal sarcomata of the jaws are rare before the age of fifteen, but they are occasionally found in very young children. (*See also* page 914.)

Myeloid sarcomata occur in the maxilla, and, as a rule, arise in connection with the nasal or alveolar processes, and occasionally in the pre-maxilla. In the mandible they spring from the body of the bone, and expand its inner and outer plates.

To judge from current descriptions, it would be imagined that myeloid sarcomata are fairly frequent in the alveolar borders of the jaws; this error is due to the circumstance that sufficient attention has not been devoted to sarcomata arising in connection with the developing teeth. When the specimens preserved in museums as examples of myeloid sarcomata are critically examined they will be found to fall in three categories: (1) fibrous odontomes; (2) sarcomata originating in the follicles of teeth; (3) myeloid sarcomata. The characters of myeloid sarcomata are described on pages 468 and 914.

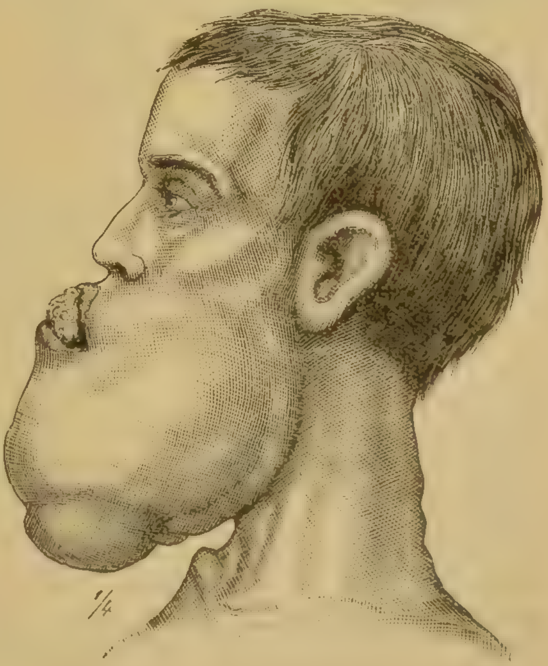

Fig. 365.—Large recurrent Sarcoma of Mandible.

2. Tumours of the gums.—These belong to four genera: fibromata, sarcomata, papillomata, and epithelioma.

All tumours of the

gum were formerly called epulides; but when the microscope was employed to assist in the classification of tumours, it was found that some epulides were sarcomatous, others fibrous, a few myxomatous, and so on. As a consequence the term came to have merely a topographical significance. It will be wise to restrict the term to tumours composed of fibrous tissue arising from the gums or from the peridental membrane.

Fibromata (epulides).—A fibroma arises either in connection with the root of a decayed tooth or from the retained root of a carious tooth hidden by the gums. An epulis of this character is made up of fibrous tissue covered externally with the gingival mucous membrane; it may be pedunculated or sessile, and occasionally two may be present. When freely excised and the stump or carious tooth (with which they are invariably associated) removed, the growth rarely returns. Although an epulis is seldom larger than a walnut, it may attain a size equal to the closed fist. Such a tumour will exercise great pressure upon the dental arches, distort the cheek, alter the shape of the maxilla and mandible, encroach upon the palate, and even protrude between the lips.

It is important to bear in mind that some of the most malignant sarcomata in their early stage very closely simulate the innocent fibromata. Prognosis in such cases should be guardedly given.

Sarcomata of the gums were referred to in the preceding section.

Papillomata are not common, and are unimportant. They present the usual characters of warts in other situations.

Epithelioma.—This is a frequent and very grave condition. It may begin in any part of the gum, but appears more frequently in the mucous membrane covering the lower, than in that of the upper alveolar processes. It occasionally starts at the edge of leucoplakic or ichthyotic patches, which are sometimes found on the gums as well as on the tongue and mucous membrane of the cheek. More frequently the disease starts near the stump of a carious tooth, and very quickly infiltrates the adjacent mucous membrane; thus, whilst it is eroding the bone, it is creeping along the mucous membrane towards the cheek on one side and the tongue on the other. The way in which epithelioma erodes such a firm and compact bone as the mandible is very astonishing. Similar effects may be observed when the disease attacks the gums in relation with the maxilla; as the alveolar process is destroyed the cavity of the antrum is exposed, and a foul ulcerating chasm formed. One of the facts connected with epithelioma of the mucous membrane of the mouth—and it matters little whether the disease begins on the tongue, cheek, hard or soft palate, or gums—is the extraordinary size which the infected lymph-glands in the neck sometimes attain, whilst the ulcer scarcely exceeds 1 cm. in diameter. This is worth bearing in mind, because an enlargement of the cervical lymph-glands in individuals past middle age should always induce the surgeon to examine the various recesses of the mouth and fauces for small, inconspicuous epitheliomatous ulcers, which are very liable to escape detection. Death is usually due to exhaustion, the result of pain, distress of mind, difficulty in taking food, and hæmorrhage. Many die from septic pneumonia.

3. Tumours of the teeth.—These belong to two genera: odontomes and sarcomata.

Odontomes.—These tumours demand careful consideration, as they are clinically innocent, and in treating them surgeons have performed many unnecessarily severe operations in ignorance of the nature of the tumour.

Odontomes are as frequent in the maxilla as in the mandible. The anatomical peculiarities of teeth tumours were considered on page 456, which should be re-read in connection with this section.

Epithelial odontomes.—These occur most frequently about the twentieth year. They are encapsuled; but when the overlying mucous membrane ulcerates, they somewhat resemble epithelioma, and are sometimes mistaken for myeloid sarcomata. When large, they erode and destroy the surrounding bone (Fig. 366).

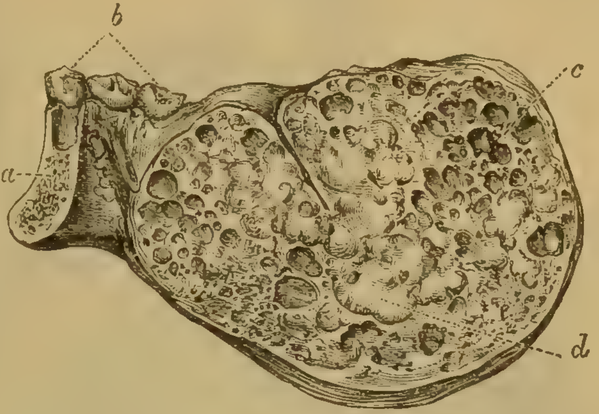

Fig. 366.—Epithelial Odontome of the Mandible. (After Pepper (St. Mary's Hospital Museum.)
a, Divided bone; *b*, teeth; *c*, cysts containing a glairy fluid; *d*, lobules of the growth.

Follicular odontomes (dentigerous cysts). — These are most common between the seventh and twenty-fifth years. The clinical signs vary with their size and situation. They may be bilateral, and as many as four may be present. When small and impacted between the plates of the mandible, diagnosis is sometimes difficult, but the key to the nature of the case is furnished by detecting the absence of a tooth, as follicular odontomes only arise in relation with unerupted teeth. In the early stage they are liable to be confounded with myeloid sarcomata. Later, when the tumour has emancipated itself from the jaw, its walls expand and form a globular fluid-containing tumour, which may give rise to fluctuation and egg-shell crackling.

When these tumours arise from the follicles of maxillary teeth, they quickly invade the antrum; when bilateral, they hideously distort the face. Several cases have been recorded in which tumours as large as the specimen represented in Fig. 367 have occupied both antra.

Fibrous odontomes.—This species occurs as tumours impacted in the mandibles or occupying the antra of children from the commencement of the second dentition to puberty. They are often bilateral, and when associated with the six-year-old molars, produce a curious and characteristic appearance of the face; such patients usually exhibit traces of rickets. These tumours may attain the size of bantam's eggs; as the child gets older they dwindle. Fibrous odontomes are often mistaken for myeloid sarcomata. They are absolutely painless.

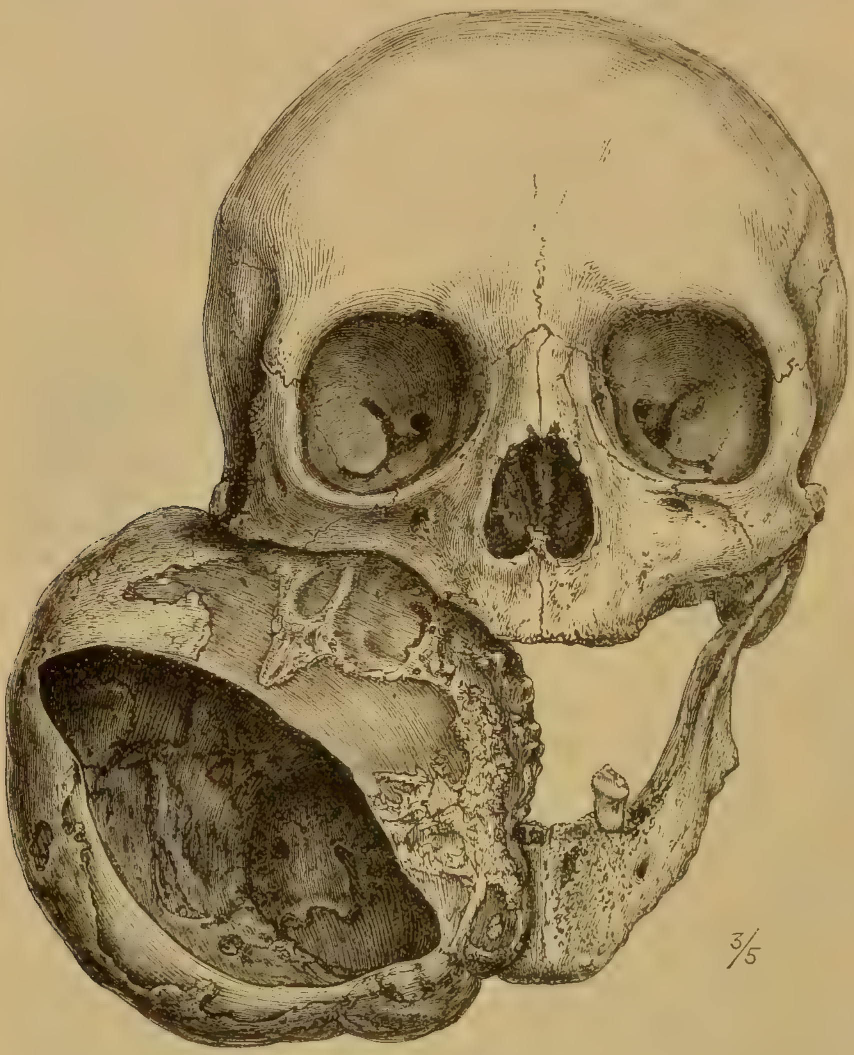

Fig. 367.—Large Follicular Odontome (Dentigerous Cyst) occupying the Mandible. (St. George's Hospital Museum.)

Radicular and composite odontomes.—As with other species of odontomes, these are equally common in the maxilla and mandible. Those growing in the maxilla are often far larger than those found in the lower jaw, as they are able to invade the antrum. Composite odontomes in the mandible rarely exceed a pigeon's egg in size (Fig. 368); those in the upper jaw are sometimes as big as a bantam's egg, and occasionally exceed this size. Some large odontomes from the antrum have been described as exostoses, but careful histological examination shows that they are composed of dentine, cementum, and enamel (Fig. 369).

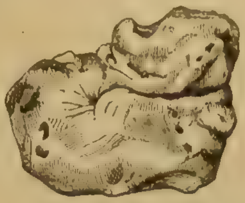
Fig. 368.—Radicular Odontome from the Mandible. Natural size. (Humphreys and Windle.)

To the present time upwards of thirty composite odontomes are known; of these two-thirds were situated in the mandible, and the remainder occupied the maxillæ. The largest specimens were from the upper jaws, and in some of them the clinical facts were very extraordinary.

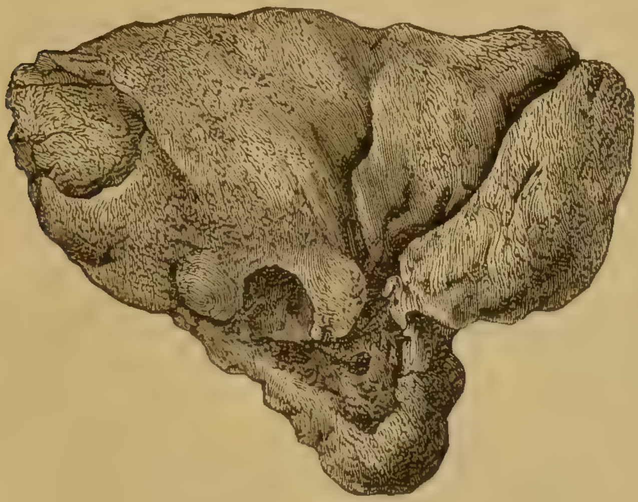
Fig. 369.—Large Composite Odontome from the Antrum. Natural size. (T. Duka.)

Composite odontomes resemble teeth, in that for a time during their development they remain hidden below the gum; in due course they enter on an eruptive stage, when the suppuration, and constitutional disturbance dependent thereon, draw attention to them (Fig. 370). In some cases the eruption of an odontome has placed life in jeopardy.

Most cases of odontomes, when they come under observation, are usually regarded as tumours of the jaws, or necrosed bone. When a young individual, with a tumour of the jaw, comes under observation, it is incumbent on the surgeon to satisfy himself that the tumour is not an odontome before proceeding to excise a portion of either mandible or maxilla.

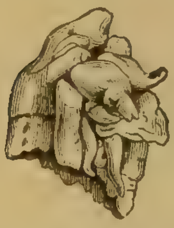
Fig. 370.—Composite Odontome from the upper Jaw. Natural size.

4. Tumours of the antrum.—These include the following genera:—Myxoma, sarcomata, and epithelioma. In addition, the antrum is extremely liable to be invaded by tumours, originating in the orbit, nasal fossa, nasopharynx, gums, and teeth.

Myxoma.—Tumours of this genus growing in the antrum are identical, and usually associated with the common mucous polypus of the nose. When very numerous, they expand the body of the bone and produce great disfigurement.

The second variety of myxoma springs from the infra-orbital nerve and invades the antrum. Its chief clinical feature is agonising pain. Such a tumour is scarcely likely to be diagnosed until it begins to expand the surrounding bone.

Sarcomata.—These are of the round or the spindle-celled species, and arise from the muco-periosteum of the antrum. Their growth leads to expansion of this cavity, and enlargement of the maxilla, causing it to encroach on the nasal fossa and obstruct respiration. Very often an antral tumour pushes up the orbital plate and displaces the eyeball (proptosis) and in a certain proportion of cases the alveolar border is depressed. The nasal duct is frequently implicated, and when completely obstructed epiphora is the consequence. Clinically, a sarcoma originating within the antrum behaves like a central tumour of a long bone, and by degrees processes of the tumour make their way through the thin walls and implicate the skin of the cheek, or, projecting into the nasal fossa, ulcerate, and give rise to frequently recurring hæmorrhage. When an outrunner of the tumour makes its way through the posterior wall of the antrum it will enter the zygomatic and spheno-maxillary fossæ, and creep thence into the temporal fossa, or make its way through the spheno-maxillary fissure and ramify in the orbit, or steal through the sphenoidal fissure or foramen rotundum into the middle fossa of the cranium.

Epithelioma.—In the majority of cases of epithelioma of the antrum the disease begins in the gingival mucous membrane, often in the socket of a tooth, and early invades the antrum. There is, however, a rare disease to which this cavity is liable, named by Reclus "epithelioma térébrant," usually rendered in English as "boring epithelioma." It is quite open to question whether the disease is epithelioma, but it is certainly an excessively malignant and extremely insidious disease.

It commonly attacks patients past middle life. They complain of pain in the jaw, for which no adequate cause can be assigned. Gradually a slight fulness is observed in the infra-orbital region, with, perhaps, œdema of the eyelid; the skin becomes brawny, and at last an epitheliomatous ulcer appears in the skin of the cheek, and the antrum is then found to be filled with a tumour. When such a case is submitted to operation, and the skin of the cheek reflected, the extensive inroads the disease has been silently making on the surrounding parts will be seen to be truly extraordinary. The greater part of the maxilla will be found destroyed, and outrunners from the growth will be found in the orbit and among the pterygoid muscles. The skin of the cheek is usually so infiltrated that it must be removed. The successful treatment of such cases demands much boldness on the part of the operator, as he will find it necessary to sacrifice the eye and the orbital contents, the palatine aspect of the maxilla, and a portion of the skin covering the cheek;

as a result, a large yawning chasm remains. Life is rarely prolonged, but by the removal of the tumour the patients are spared much pain and discomfort.

Diagnosis of tumours of the jaw.—This is a matter which in many cases is simplicity itself, and in others surrounded by the greatest difficulties.

In the case of the mandible, the chief point is to decide, if possible, whether the tumour arises in connection with the teeth or the periosteum. Tumours of the teeth in young patients, in the immense majority of cases, are innocent. In those who are over thirty the tumours most likely to occur in connection with teeth sockets are epithelioma and actinomycosis, and these are scarcely likely to be mistaken for innocent affections. Tumours growing from the bone are osteomata or sarcomata, and the hardness and slow increase of an osseous tumour are not likely to be confounded with the rapid growth, pain, and constitutional disturbance caused by a sarcoma.

It is in connection with the antrum that the great difficulty is met with. Rapid growth, bulging walls, great pain, and bleeding from the nose are ominous indications of sarcomata; nevertheless many sarcomata cause no pain or even discomfort until the late stages. Slow growth and painlessness may mean some species of odontome. Pain, fever, and purulent discharge may indicate abscess of the antrum or suppuration around a composite odontome.

Early signs of antral tumour are obstruction of the nasal fossa, bulging of the hard palate or cheek, or proptosis and epiphora. Enlarged pre-auricular and sub-maxillary lymph glands occur with epithelioma. Lastly, nasal, orbital, and naso-pharyngeal tumours invade the antrum, and very closely simulate primary tumours of that cavity. A sarcoma of the ramus of the mandible is apt to be confounded with a tumour of the parotid gland.

The most satisfactory manner of avoiding errors in diagnosis, and consequently grave blunders in treatment, is to study carefully on museum preparation the morbid anatomy of tumours of the jaw, especially those illustrating their early stages.

Treatment of tumours of the jaw.—One principle only underlies the treatment of tumours of the jaws: that is, removal at the earliest possible moment. Unless the whole of the tumour can be completely extirpated, no operation should be undertaken. The mode of removal varies with the tumour. Encapsuled tumours —such as odontomes of all species—merely require enucleation. Localised tumours—such as epulis and osteoma—should be excised with a piece of the underlying bone. Diffuse tumours—such as sarcomata and epithelioma—demand wide removal. In the case of the mandible, it is often necessary to excise the body of the bone from near the symphysis to the angle. Occasionally the symphysis is excised. This is rare. In the case of the maxilla, wide removal of the bone is nearly always demanded.

In doubtful tumours of the antrum the cautious surgeon explores it by means of a perforation through the canine fossa before resorting to extirpation. In all cases where there is doubt as to the nature of the tumour, *cut into it before proceeding to cut it out.*

Results.—The effects of treatment in the case of encapsuled tumours are very gratifying, and with care many tumours can be enucleated from within the mouth, and leave no external scar or disfigurement—a matter of especial importance in the case of females. In sarcomata and epithelioma the results of excision are not encouraging. Patients recover, as a rule, easily enough, even after operations of appalling magnitude; but the disease quickly recurs, and may demand repeated operations. Although operation in these cases offers no prospect of permanent cure, it nevertheless frees the patient from pain, relieves him of a foul and disgusting mass, and in many instances not only makes life fairly comfortable, but actually prolongs it.

In the case of myeloid sarcomata life may be prolonged many years by operation, and in many instances the benefit is permanent.

THE TEMPORO-MANDIBULAR JOINT

The diseases of this joint are: acute arthritis, chronic arthritis, enlargement of the condyle and neck of the mandible, and anchylosis.

1. **Acute arthritis.**—This is occasionally due to traumatism, but more frequently occurs as a sequel to the exanthemata, especially scarlet fever, and as a sequel of rheumatic fever. It is more common in children than in adults.

The symptoms are those common to acute inflammation of joints. They are local pain, swelling, and redness. The pain is increased by movement of the joint and by pressure. Febrile symptoms are present.

Should the changes proceed to suppuration, the pus may point immediately over the joint, or in children find its way into the external auditory meatus through the foramen in the tympanic plate, and simulate otorrhœa. Exceptionally, the condyle dies, and the necrosis may involve the neck and outer surface of the ramus of the mandible.

Treatment.—In the early stage rest for the joint by interdicting speech, and supporting the chin by a soft bandage tied around the head. The patient should be kept on liquid diet. Should the condyle necrose, it will require excision.

2. **Chronic arthritis.**—This is a somewhat unusual disease, and, like osteo-arthritis of other joints, is incidental to advanced life. The disease may be unilateral or bilateral. The inter-articular disc and the articular cartilage gradually disappear, the condyle, and less frequently the glenoid fossa undergo erosion, and the synovial membrane becomes villous. Occasionally the margins of the fossa become lipped, and processes of bone form around the borders of the condyle.

In the majority of cases the patients complain of aching in the joint, a creaking sensation on movement, and are occasionally annoyed by partial dislocation when the mouth is widely opened. This condition is by no means uncommon in thin delicate women after middle life.

Treatment.—Mild counter-irritants to the joints are sometimes beneficial. Internal remedies are of no avail.

3. Enlargement of the condyle and neck of the mandible. —This is a somewhat rare condition, but it produces unmistakable distortion of the face. From the few examples which have been examined—*post-mortem*, or after removal by operation—the changes in the condyle are those characteristic of chronic (osteo-) arthritis.

In severe cases it has been found necessary to excise the condyle and its neck.

4. Closure of the jaws. — This condition presents two varieties: (1) Spasmodic and (2) organic.

Spasmodic closure of the jaws is sometimes induced in consequence of irritation of the mandibular division of the fifth cranial nerve, leading to reflex contraction of the muscles of mastication. This condition is sometimes called trismus, and is occasionally due to the impaction of a wisdom tooth. It is important to bear this in mind, as the discovery and removal of the tooth at once brings about a cessation of the symptoms.

Trismus is not infrequently simulated in hysterical women.

Organic closure of the jaws is brought about in the following ways: anchylosis of the temporo-mandibular joint, contractions due to cicatrices, and defective development of the mandible.

Anchylosis.—This may be due to adhesions the result of acute arthritis. Anchylosis has followed fracture of the base of the skull, the presumption being that the fracture implicated the glenoid fossa. The condition has not been demonstrated *post-mortem.*

Cicatricial contraction.—This may follow sloughing of the buccal mucous membrane due to cancrum oris, or to scars on the cutaneous surface of the cheek, the results of extensive burns, or upon the neck and front of the thorax, in very severe cases of lupus, inflammatory infiltrations of the masseter, and operation on the face for epithelioma, rodent ulcer, gunshot injuries, and the like.

DEFECTIVE DEVELOPMENT OF THE MANDIBLE.

Individuals occasionally come under observation, complaining of inability to separate the jaws. On examining them the jaws will be found so firmly closed, that in many cases food can only be introduced into the mouth in a state of fine subdivision, and in severe cases through a gap due to loss of two or more incisor teeth.

On examination no disease of the mandible or temporo-mandibular joint can be detected, but what strikes the surgeon most is the stunted character of the mandible. This produces a peculiar appearance, owing to the fact that the face above the level of the mouth corresponds in development to the age and stature of the individual, but the mandible, chin, and associated structures are in degree of development similar to those of a child of six or seven years of age. These characters lead to ready recognition of the condition (Fig. 371). Of the cause of this affection nothing is known.

Treatment.—Spasmodic closure of the jaw due to trouble connected with the wisdom tooth usually responds to appropriate measures. In fact, many cases of trismus terminate with the eruption of the wisdom tooth. In some cases, when there is doubt as to the position of his tooth, the second molar has been extracted; this has afforded space, relieved the nerve from pressure, and the tooth has subsequently erupted. At the time of extracting the second molar, a careful search should be made for the errant third molar.

When the closure is due to anchylosis, whether it be fibrous or osseous, the best treatment is excision of the condyles; in the most successful cases it has been necessary to excise both. Some good results have followed division of the neck of the condyle.

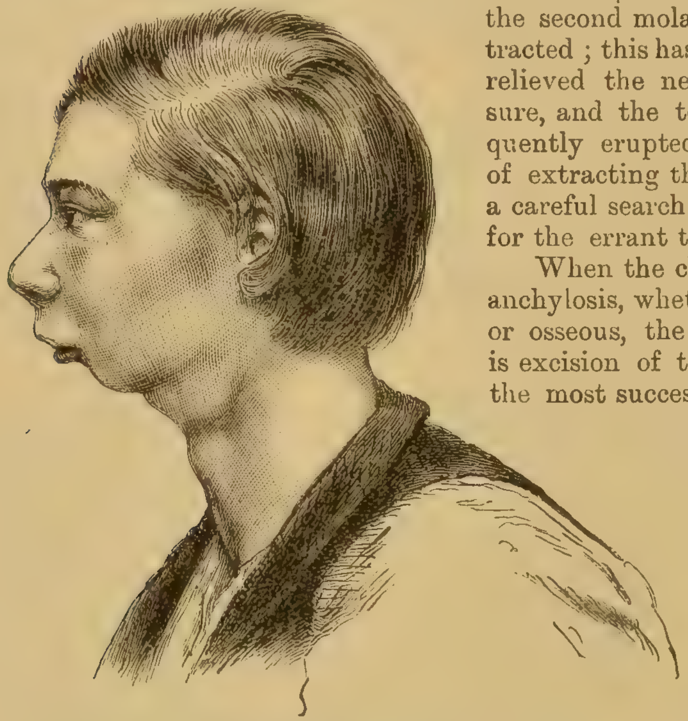

Fig. 371.—Closure of the Jaw due to defective Development of the Mandible. (Humphry.)

When the closure results from the contraction of a cicatrix, the surgeon has to face one of the most trying cases in surgery. Attempts are at first made by the use of lever gags and spring gags to limit the amount of contraction, usually with little avail. Then efforts are made to establish a false joint after the method introduced by Esmarch. This consists in removing a wedge-shaped piece from the mandible at a spot anterior to the cicatrix, the apex of the wedge being at the alveolar border.

In less severe cases it may not be necessary to remove a wedge-shaped piece of bone; then Rizzoli's operation may be practised. This consists in dividing the jaw from within the mouth by means of cutting forceps.

Esmarch's procedure, however, is the operation usually adopted, and it certainly gives the best results.

In some cases it may be necessary to perform Esmarch's operation on both sides. In cases where excision of the condyles has failed, Esmarch's operation has subsequently been practised with success.

In closure of the mouth from defective development of the mandible, excision of the condyles, and even division of the necks of the condyles has been followed with very satisfactory results.

EXTRACTION OF TEETH.

Although the treatment of the teeth constitutes a very special branch of the Art of Surgery, it is nevertheless the duty of every medical practitioner not merely to be capable of extracting a tooth with skill, but to understand how to save a damaged tooth. All village surgeons and practitioners in remote stations, surgeons in the army or the navy, and even operating surgeons in large towns, should know how to be able to extract a tooth properly.

Teeth require extraction for the following conditions: (1) When they are the cause of an alveolar abscess. (2) When the crowns are so destroyed by caries that they cannot be prepared to hold a stopping, or utilised for an artificial crown. (3) It is occasionally necessary to remove a tooth when the dental arch is very crowded and irregular in consequence. (4) Impacted wisdom teeth. (5) When the crown of a tooth has sharp edges and irritates an epithelioma of the tongue or cheek. (6) Canine teeth lodged transversely. (7) All stumps should be promptly extracted. (8) A carious tooth associated with an epulis. (9) In operations for the removal of a portion of the mandible or maxilla. (10) When an abscess or great pain ensues on an immediate root-filling.

It is a very pernicious practice to extract teeth simply because they ache. In many instances the pain is due to an exposed pulp. When this is the case, the tooth may often be saved by proper treatment. This consists in destroying the pulp by arsenical paste; it is then extirpated, the pulp cavity and root or root's canal are then rendered aseptic, and subsequently stopped.

When teeth are fractured by blows or falls the pulps should be destroyed—in some cases the pulps may be extracted immediately under gas—and the roots serve as admirable foundations for artificial crowns.

The method of extraction.—Two instruments are now used for the extraction of teeth: forceps and the elevator. Forceps are of various patterns, and so adapted to the different kinds of teeth, that they may be grasped and extracted without crushing the crowns.

It is the duty of the student to make himself acquainted with the peculiarity of each tooth, including the number, shape, and disposition of its roots. He should master the characters of the forceps designed for each tooth, and watch the mode of using them in the extracting-room under the supervision of the dental surgeon. He will then learn that teeth are extracted not by the exhibition of great force following the indiscriminate selection and application of the forceps, but by the skilful application of force.

There are many dangers incidental to the extraction of teeth, such as hæmorrhage; fracture of the jaw; extraction of the wrong tooth, extraction of two teeth simultaneously; the passage of a tooth or stump, or a piece of the forceps or mouth prop, into the larynx and trachea; fracture of the tuberosity of the maxilla; forcing the stump into the antrum or nasal fossa; injury to the mandibular (inferior dental) nerve or dislocation of the mandible.

Hæmorrhage after extraction.—It is rare that the loss of blood after tooth extraction is a source of anxiety; nevertheless hæmorrhage from a tooth-socket is occasionally severe enough to imperil life. In many cases it is sufficient to plug the socket firmly with a strip of lint, or small pieces of sponge dusted with tannic acid, or persulphate of iron. In exceptional cases, Paquelin's cautery at a dull heat may be employed. Fifteen grains of gallic acid given internally has a useful styptic effect.

When a healthy tooth is extracted by mistake, the socket should be irrigated with warm water to clear it of blood; the roots of the tooth should be similarly cleansed and the tooth replaced and firmly pressed home. Many teeth replaced thus have retained their position for years.

The passage of a tooth or other body into the larynx during extraction is a very grave accident. Should the patient be under the influence of an anæsthetic, and the foreign body remain in the larynx, it will induce spasm and rapid asphyxia unless promptly removed with the fingers, or by tracheotomy, or laryngotomy.

Should it slip between the vocal cords, it may lodge in the trachea or fall into a bronchus and set up septic pneumonia, abscess of the lung, etc. The treatment of foreign body in the trachea is considered in Article XLIII., on INJURIES AND DISEASES OF THE NECK, Vol. II.

Occasionally when a tooth is extracted it slips from the forceps, and falling upon the tongue is sometimes carried backwards into the pharynx and swallowed. Such an event causes momentary uneasiness to the operator.

In extracting teeth under nitrous oxide it is necessary to remove any artificial plate worn by the patient, and in placing the mouth-prop in position to take care that it does not rest on loose teeth; when two or more are to be extracted, it is necessary that the operator should be expeditious, but he should take care that the tooth or stump first extracted has been released from the blades before he re-introduces the forceps into the mouth. It is a fact that this is one of the means by which teeth get into the larynx and trachea.

In extracting a molar-tooth from the mandible, the beginner is very apt to pull hard upon it; suddenly the tooth yields, and the forceps comes in contact with the crown of an upper molar or bicuspid: sometimes the concussion is so great as to knock off a part or the whole of the crown. Knowledge of the liability should be sufficient to prevent such an accident. Lastly, should a tooth or stump fall into the patient's mouth, do not proceed with the extracting until it is recovered and removed.

XXXII. INJURIES OF THE JOINTS AND DISLOCATIONS.

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Contusions of joints. **Pathology.**—The local conditions associated with contusions of a joint will vary with the nature of the injury, and the size of the articulation involved. Heavy blows and crushing violence, as from a spent shot or railway accident, may inflict much damage on a joint without rupturing the skin. Thus, the articular ends of the bones may be splintered, or blood extensively extravasated into the cancellous tissue. The joint may be distended with blood, and portions of cartilage, bone, ligamentous tissue, or synovial membrane torn or entirely detached. The main vessels of the limb may also be ruptured. In slight contusions all these severe lesions are absent, and the only changes found are slight periarticular bruising, and, perhaps, some blood extravasation in the cancellous bone, with serous exudation into the joint causing heat and swelling. The term "synovitis" was formerly applied to these cases. It is doubtful if extensive inflammation, in the absence of organisms, exists, and the articular effusion may be looked upon as merely the result of irritation.

Consequences.—In severe crushes of a joint implicating the main vessels, gangrene may ensue, or secondary sloughing of the distended skin, the injury becoming compound. Then diffuse suppuration and various septic disorders are very prone to arise. Necrosis of the bone ends may follow, with separation of sequestra and acute abscess. The most severe constitutional pyæmic symptoms may follow a moderate contusion of a joint, especially if the epiphysial region be bruised in a young person, even though the skin remains intact. The pathological explanation is not easy. It may be rationally supposed that the organisms (streptococci or staphylococci) so abundantly found in these cases gain access to the circulation from some unsuspected source, and arriving at a part where the normal resistance and vitality of the tissues are lowered by the injury, are thus able to grow and multiply, forming a source of potent general systemic infection.

Bruising or effusion of blood at the epiphysial line may lead to subsequent arrest of growth, and this is important from a prognostic point of view. Effused blood is usually absorbed, but fibrinous masses may long remain in a large joint, as the knee. Portions of bone or articular cartilage may become permanently detached after a severe blow, but this is comparatively exceptional, the loose bodies found after these accidents being usually pre-existing inflammatory formations broken off by the violence. Anchylosis may follow any severe crush of a joint. This is usually fibrous, as the cartilages are not entirely removed. Should the injury be compound, and followed by suppuration and destruction of cartilage, osseous anchylosis (synostosis) may eventuate.

Slight contusions may also be followed by tuberculous disease of the bone in predisposed delicate individuals, by abscess and central necrosis of the articular ends, and lastly, by the rapid growth of a soft sarcomatous tumour. The latter affection is rare, but it may be readily confounded with abscess, unless an exploratory puncture be employed. Contusions and severe sprains of the larger joints in elderly persons may originate a kind of chronic arthritis, associated with roughening of the articular surfaces, calcareous deposits, "lipping" of the articular margins of the bones, in fact, many of the changes commonly attributed to chronic rheumatoid arthritis. In the hip, absorption of the head of the bone may occur, with consequent shortening. After a severe contusion of this articulation, gradual shortening with grating and creaking on motion, and lameness with loss of power over the articulation, are familiar to surgeons. Some of these cases are, doubtless, overlooked fractures of the neck of the femur. The majority can only be explained as the consequences of chronic arthritis originated by the injury.

Treatment.—Crushes of a joint implicating the bone ends, and rupturing the main vessels, demand amputation. As a rule, very extensive bruises are well recovered from, if only the skin remains intact. All the efforts of the surgeon should be directed towards subduing the subsequent inevitable severe inflammation. The ice-bag or Leiter's tubes should be used with caution, if the skin be much bruised or likely to perish. The limb should be well elevated and carefully fixed in splints; the patient, if robust, kept on low diet, with occasional purgation. In strong young adults, either in sprains or contusions, when there is intense inflammatory reaction, free application of leeches may be practised with the best results. Should suppuration set in, or necrosis ensue, the complications must be treated on general principles. Continuous immersion in warm baths, or continuous irrigation will often give excellent results in compound crushes of joints. The stream of water in irrigation must be free, and flow from a large receptacle raised above the bed. By this means compound crushes of the larger joints may now be saved, which formerly would have been condemned to amputation. I strongly advocate this treatment.

Sprains of joints.—A sprain may be defined as a wrench or twist of a joint stretching or rupturing the ligaments, and associated with intra-articular hæmorrhage, and much exudation of serous fluid about the joint and into its cavity. The tendon sheaths may be filled with blood or exudation, or the fibrous part of the sheath ruptured, and the tendon displaced from its natural groove.

Pathology.—The amount of damage done to a joint in a severe sprain varies. The ligaments being exceedingly strong, scales of bone may be torn away rather than the ligaments themselves be ruptured. Synovial folds may be stretched or torn. Any inflammatory outgrowth of cartilage may be detached, forming a subsequent "loose body" in the joint. Surrounding tendons may be displaced from their grooves, or muscular fibres ruptured; and in the knee, a severe sprain may be followed by a displacement or detachment of an articular cartilage.

Diagnosis.—The diagnosis of sprain being mainly negative, too great care cannot be exercised in giving an opinion as to the actual nature of the injury. If much swelling has supervened, additional caution is needful; for in such cases the best observer may overlook a fracture or dislocation. The presence or absence of bone lesion is a matter of the first importance. In children, separations of the epiphysis closely resemble sprains; and should the surgeon suspect this injury, he may warn the relatives of the possibility of arrest of growth. Great care should be exercised in examining for displaced tendon.

It is often difficult or impossible to know in what direction a limb has been twisted in a sprain. A useful hint will soon be given to the surgeon by the appearance of effused blood, which will extend along that side of the limb corresponding to the torn ligaments. The joint may then be placed in the most advantageous position for repair. Thus the foot may be everted in extravasation along the outer side of the leg.

Treatment.—As in contusions of joints, rest, the application of splints, elevation, and the judicious use of cold by Leiter's tubes or the ice-bag are efficacious in the early stages. Moderate compression by a Martin's bandage or Gamgee tissue is highly recommended, but, in my experience, is seldom tolerated. The skin being sound, free leeching will be found to give great relief, when the inevitable heat, swelling, and congestion supervene. Should the effusion of blood and exudation into the joint be very excessive, the limb must be well elevated, and aspiration with a fine and perfectly clean needle and syringe may be adopted. This must not be looked upon, however, as a routine treatment.

In the young and plethoric the diet should be low, and the bowels kept freely open with mercurials. In all sprains, so soon as the heat and excessive swelling have disappeared and the ligaments united, massage and movement, combined with the hot douche, should be sedulously carried out, to prevent the formation of adhesions. Galvanism should be employed for the disabled peri-articular muscles, especially in the case of the shoulder.

In some instances effusion long persists, with painfulness of the joint, and imperfect restoration of its functions (chronic sprain). In such cases, one or more painful spots are usually to be detected on pressure about the joint, these being probably due to the entanglement of nerve filaments in the healed tissue. In such cases, the administration of nitrous oxide gas and the carrying of the limb through all its normal movements will result in the audible rupture of adhesions and the setting free of the nerve twigs, the symptoms rapidly improving. The surgeon will find valuable indications as to the time to commence movement in a sprained joint from the local temperature. So long as a joint is hot, caution must be exercised. When it is cool to the touch, movements may be freely adopted.

After a bad sprain a large joint is painful and insecure for many months, owing to the weakness of the ligaments from rupture, and the impairment of movement and functions of the tendons in their sheaths. Nothing is so beneficial for these cases as prolonged massage, with tonics and sea air, and carefully-made leather supports.

Injuries of the tendon sheaths in sprains.—Hitherto enough attention has not been drawn to the condition of these important structures in severe sprains or blows upon joints. Blood is extravasated into the synovial sheaths, with serous exudation. The subsequent organisation impedes the free play of the tendon, and this explains the extraordinary stiffness and weakness of a joint which may persist for months after a bad sprain. Again, the fibrous vincula which retain the tendon in a groove may give way, allowing actual displacement. This is well known to occur in the case of the peroneal tendons, and it may reasonably be assumed that it occurs elsewhere. The most cautious prognosis must be given in a bad sprain, especially in a large joint of the lower extremity and in those who are obese, aged, rheumatic, or gouty. The majority of these cases may be looked upon as more serious than fractures.

Wounds of joints.—These are peculiarly dangerous injuries. The gravity of the case will be in proportion to the size and synovial complexity of the articulation involved, the nature of the injury, and the cleanliness or otherwise of the instrument with which it is inflicted. Serious examples would be a bullet wound of the knee splintering the bone, or the opening of this articulation with a dirty butcher's knife. An example of a favourable kind would be the clean-cut, aseptic wound into the knee made by a surgeon in removing a loose body, which usually heals with no local trouble or constitutional disturbance.

Symptoms.—It is in punctured wounds that the main difficulty arises in deciding whether the articulation is opened, and especially is this the case when the external orifice of the wound is situate at a distance from the joint. Two symptoms of the gravest significance are, escape of synovia, and rapid swelling of the joint from effused blood.

Synovial fluid may escape from a wounded bursa or tendon sheath; but this symptom should always lead to great care in treatment, and injudicious probing should be avoided. Immediate swelling of the joint is sufficiently conclusive, in cases of punctured wound, to lead the surgeon to suspect that the articulation has been opened.

In favourable cases, beyond a little transient effusion, nothing of note happens; the wound heals, and the articulation is soon restored to its ordinary state. In neglected punctures, and especially in dirty wounds, the phenomena of acute intra-articular suppuration ensue, and tetanus may possibly supervene if earth or street soil has gained access.

Acute septic arthritis, abscess of joint, intra-articular abscess, suppurative synovitis. *Symptoms.*—The joint rapidly swells, becomes red and dusky, very hot, and intensely painful, the integuments pitting on pressure. It is usually flexed, and any attempts at movement are dreaded and resisted, the patient shrieking out with pain and apprehension. Occasionally, the muscles spasmodically contract, rubbing the inflamed articular surfaces upon each other. When the cartilages are eroded and the carious bone ends are thus rubbed together, agonising "jumping" pains arise, so familiar in these cases. This symptom is marked at night when the patient is "dropping to sleep," and the control of the will is temporarily removed from the irritated nerves and muscles. It quite prevents sleep or rest. The patient is in high fever, with a dry tongue, and in bad cases may be delirious. Rigors, sweating, and "secondary" abscesses may indicate pyæmia. As the case progresses, the whole limb swells enormously, and abscesses form, burrowing among the muscular planes. These, when opened, leave discharging sinuses, communicating with the now carious bone ends. The joint may become displaced and totally disorganised; or if supported in proper position, anchylosis may ultimately ensue, should the patient survive. The anchylosis will be bony if the cartilages are entirely destroyed and removed. These cases may prove fatal from pyæmia, septicæmia, tetanus, or exhaustion from hectic and prolonged suppuration. They may be regarded as some of the most grave accidents of surgery.

Treatment of wounds into joints.—In extensive injuries with comminution of the bone ends, or when compound fractures extend into large joints, as when a bullet strikes the femur in its lower third and fissures extend into the knee, amputation is often requisite. In some cases, and especially in the hip and joints of the upper extremity, primary re-section may be advisable. The value of continuous irrigation in these cases is very great. In punctured wounds the parts should be carefully cleansed, and the wound well washed out with hot (1 in 2,000) perchloride of mercury solution. To cleanse the skin, carbolic soap and hot water may first be employed, and this should be followed by ether, and subsequently by a lotion (1 in 1,000) of corrosive sublimate, with rectified spirit and hot water. The wound, if clean cut, may be accurately united at once. Should the edges of the wound be ragged and bruised, immediate union had better not be attempted. In cases where the surgeon has reason to suspect that some septic material has gained entrance, the strictest watch should be kept upon the temperature and local symptoms, and any accumulation of serum or pus in the track of the wound should be afforded speedy exit. In more extensive injuries, when it is determined to save the joint, every recess of the cavity should be flushed clear of blood and dirt by hot perchloride lotion (1 in 4,000), and large drainage-tubes should be inserted in convenient dependent situations.

After healing of a punctured wound, should swelling and fever supervene, with the signs of acute suppuration, the joint should be aspirated, and if sero-pus is found in it, the articulation must be opened and freely drained, and especially irrigated. This is too often insufficiently done. In the knee, where there are several complex pouches, a tube may reach into one, and not the others. In this articulation abscess may readily burrow up the front of the femur beneath the quadriceps muscle, and even point near Scarpa's triangle. The deep bursa beneath the quadriceps is also very apt to become filled with pus. A knowledge of this will point to the importance of inserting large tubes in more than one situation.

Secondary amputation for suppuration following wound of a joint is seldom, if ever, needful in the upper extremity, and in the case of the knee or ankle, such an operation should never be performed during high fever, when the limb is swollen and infiltrated with products of inflammation. When abscesses are opened, and the temperature has become hectic in type, the chances of recovery after amputation are far greater. In a child a movable articulation may be obtained, even after suppuration, if the joint be promptly drained. In adults, firm fibrous anchylosis usually—but not universally—follows. Bony anchylosis will only occur if the whole of the cartilage be destroyed, and the bone ends kept in accurate apposition. It is most important to maintain the limb in such a position that its future usefulness may be ensured, even if stiff. Anæsthetics—especially chloroform—which I believe to be the best agent to administer to patients who are ill and in great suffering, are here very useful, to enable the surgeon to straighten a flexed and painful joint.

Dislocations of joints.

Nomenclature and comparative frequency.—Dislocations are well divided, for purposes of description, into the traumatic, spontaneous or pathological, and the congenital. Besides these, dislocations are spoken of as complete or incomplete, and partial, simple or compound, and complicated, when associated with fracture.

An idea of the frequency of different dislocations may be obtained from the records of the Middlesex Hospital for ten years, quoted by H. Morris in Holmes' "System of Surgery." Out of 283 cases of dislocations of all sorts, there were: Of the shoulder, 133; hip, 8; clavicle, 8; patella, 10; jaw, 12; knee (partial), 1; knee (semilunar cartilages), 5; foot (including ankle, astragalus, and sub-astragaloid displacements) 9; radius and ulna at elbow 20; upper end of ulna, 9; upper end of radius, 6; wrist, 6; thumb, 39; fingers, 17.

The frequency of shoulder dislocations is accounted for by the joint being mainly dependent for its security upon muscles, and also for other reasons, which will be hereafter stated. The power of leverage of a long bone in producing dislocation is well shown in the hip. It is difficult to understand how displacement of this articulation could occur, were it not for the effects of indirect violence acting at great mechanical advantage.

Predisposing causes.—Accidents which generally need considerable violence to produce them are, of necessity, more common in the male sex, in active adult life, than in females or young children. Occupations, as mining, machinery-work, and the like, predispose to these injuries. Congenital laxity of the ligaments is an important cause; and some professed acrobats, as the celebrated Warren, have the power of spontaneously dislocating certain joints. Many individuals are able partially to displace the thumb at the metacarpo-phalangeal joint, owing to laxity of the capsule and lateral ligaments.

The weakness and laxity of ligaments following injuries of joints with effusion, as a bad sprain, is an undoubted predisposing cause of dislocation; and exposed joints, as the shoulder, are more liable to suffer than others. Once a joint has been dislocated, it may readily be again put out. Previous disease, with excessive synovial distension, may predispose to dislocation. Knock-knee is said to render the patella prone to become displaced. Paralysis of muscles round a joint also renders the articulation insecure.

Exciting causes.—In some cases this is violence applied directly to the displaced bone, as when a heavy blow on the thumb in fighting dislocates the first phalanx. Far more commonly the force is indirect, as when the head of the femur is dislocated from a heavy fall on the foot of an abducted limb. Some "ball and socket" joints, as the shoulder, have been dislocated by muscular action alone, as in the spasms of epilepsy, the act of swimming, or striking out at and missing an adversary. The lower jaw and patella are known to be displaced by muscular action alone. When once a bone is dislocated, the action of the muscles in drawing it into secondary positions is very important and marked. Sometimes muscular action persistently reproduces a dislocation (recurrent dislocation). Then there is undue laxity of the ligaments, or want of union in them after the primary injury, or concomitant fracture of an articulating surface at one part.

General pathology of recent traumatic dislocations.—The severity of the local injuries found in recent dislocations will vary greatly with the distance the bone is thrown from its proper connections and the nature of the violence which produced it, the wrenching or twisting movements inflicted on a limb entangled in powerful machinery being peculiarly disastrous. The capsule and ligaments are always more or less lacerated, some of the short muscles torn or stretched ; the neighbouring vessels and nerves may be pressed upon, contused or actually torn. Gangrene or diffuse aneurysm may even supervene. Much blood is extravasated about the articulation and in the substance of the contused and torn muscles. Fractures may co-exist, and when the ligaments are very strong—as the ligamentum teres—a portion of bone is often torn away rather than that the ligaments themselves should yield. Grave cerebral, thoracic or abdominal injuries are sometimes found associated with dislocations. An evident displacement of the hip, for instance, may readily draw away the surgeon's attention from graver or more fatal co-existent lesions in other parts.

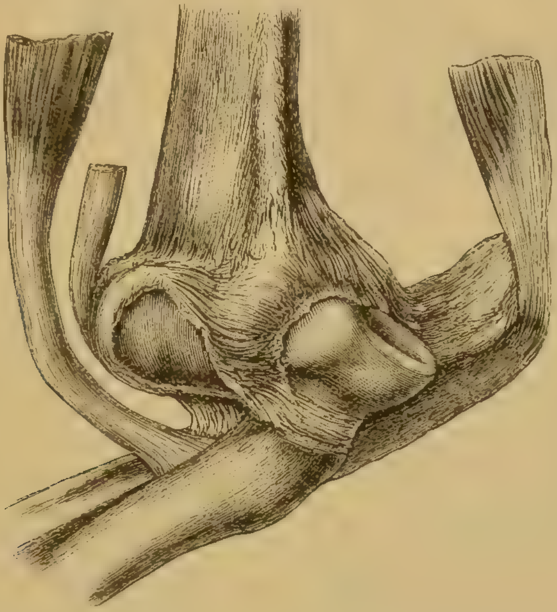

Fig. 372.—Old Dislocation of the Elbow backwards. The head of the radius has worked for itself a new socket, and the ulna is welded to the humerus by firm fibrous adhesions. (St. George's Hospital Museum.)

General pathology of unreduced traumatic dislocations.—When a dislocation remains unreduced, the inflammation of the muscles and peri-articular structures speedily terminates in exudation and organisation, so that the muscles become structurally shortened, and incapable of relaxation after anæsthesia. The new adhesions fix the bone to its fresh surroundings, enclosing it in a kind of spurious capsule, and the old articular cavity becomes filled with fibrous deposit, and subsequently is rendered shallow and well-nigh obliterated. The displaced head of the bone, by pressure absorption, soon works for itself a cavity in the bone upon which it rests and moves, and becomes itself altered in shape, and smooth and porcellanous on the surface (Fig. 372). The main vessels and nerves may be glued to the displaced bone or fragments of capsule by firm fibrous adhesions. This is a matter of vital importance in the instance of old dislocations of the shoulder.

The time taken for these changes to occur can hardly be approximately stated. Dislocations have been reduced after almost incredible periods. Whether it is best to attempt this treatment is another matter. As a general rule, if a dislocation of a large joint has remained unreduced as long as it would take a fracture of the corresponding long bone firmly to unite, great, and often insuperable, difficulties will be found in reduction. I believe this statement will be found reliable in practice.

General pathology of congenital dislocation.—The various pathological factors which, separately or combined, are found in congenital dislocations are well summed up by Hamilton as follows :—

(1) Physiological causes, arrest of development, or an original defect in the germ.

(2) Pathological causes—as nerve lesions, contraction and paralysis of muscles, laxity of ligaments, hydrarthrosis, or other articular disease.

(3) Mechanical causes—as intra-uterine violence applied to the fœtus or by injuries during delivery.

Considering the latter cases, it may be remarked that some of them are really true traumatic displacements that may be rectified. Others are separations of the epiphyses, followed by arrest of growth.

Some of the more pronounced forms of congenital dislocations are common in idiots; and such congenital deformities as hare-lip, webbed digits, or extroversion of the viscera may also co-exist. The bone ends are usually ill-shaped or stunted, and the normal articular surfaces altered in shape, shallow, or actually absent.

General pathology of spontaneous or pathological displacements.—It has been already pointed out, that in cases where ligaments have been severely stretched or ruptured by sprains or previous dislocation, displacements may take place from the slightest muscular exertion, and that some contortionists can produce certain dislocations apparently at will. Verneuil well sums up the causes of pathological displacements under the terms, dislocation by *distension*, by *destruction*, or by *deformation*. The majority of spontaneous displacements met with in practice are really examples of *destruction* from disease of joints, with implication of the bone ends and ligamentous attachments. Good clinical examples of dislocation from destruction are seen in tuberculous affections, when, the ligaments being gradually disintegrated, the bone surfaces become displaced. This is occasionally experienced in the knee. In certain cases of pyæmia, and especially as a sequel to bad cases of the specific fevers —typhoid, scarlet fever, and the so-called "puerperal state"— *distension* of the joint may occur, the ligaments are softened, and dislocations take place most unexpectedly, and often without obvious suppuration. In actual practice, dislocation from distension is generally seen in the hip, and in the joint affections associated with the acute fevers (such as typhoid). The head of the femur is generally displaced backwards.

In these cases the patient is usually too ill to allow of rigid replacement of the bone, and the efforts of the surgeon must be directed towards maintaining the limbs in a good position for subsequent firm anchylosis. Should the patient recover, excision or osteotomy may be afterwards performed with good result.

Dislocation from *deformation* is more usually partial than complete. The distortions of the joints of the fingers, in chronic rheumatoid arthritis, are familiar examples.

Deformation displacements are principally of interest, from the possibility of confounding them with traumatic dislocations, when the sufferers from these conditions of the joints are exposed to falls or other violence.

Partial or incomplete dislocations.—When large flat surfaces of bone articulate, as in the knee or astragalo-calcanean joint, partial displacements are the rule rather than the exception. In the "ball-and-socket" joints, as the hip and shoulder, much discussion has arisen as to their possibility. Many of the related cases have doubtless been mistaken for disease, as for rheumatoid arthritis; others may be attributed to speculative diagnosis in injuries associated with much swelling. When fracture co-exists, a portion of the capsule and articulating surface may be carried away with the head of the bone. This can hardly be looked upon as true partial dislocation. In "partial dislocations" of the shoulder the capsule is said not always to be ruptured; and although it is possible for a dislocation of the head of the humerus to occur without rupture of the capsule, probably this is complete—*i.e.* the articular surfaces are quite separated. Many cases diagnosed as partial dislocations of the shoulder, are really displacements of the long biceps tendon.

The cases of South and Le Gros Clark point strongly to the conclusion that partial dislocation of the shoulder is possible, but not probable. This subject will be again alluded to from time to time in speaking of the symptoms of dislocation of the larger joints.

General symptoms of dislocation.—These may thus be summarised: a person is subjected to some force acting upon a previously healthy and well-acting articulation. He may be conscious of a "snap" or sensation of tearing violence, and forthwith the movements of the joint are more or less lost, and passive motion becomes difficult, impeded, and painful. Severe "numb" pain is experienced in the course of the main nerves implicated. The appearance of the part to the eye is by far the most important sign of dislocation. The sound joint should always be compared with the injured, when alterations in contour, and of the distances and relations of salient bony points to each other, will be apparent to sight, measurement, and palpation.

No crepitus is elicited if the parts are examined directly after the injury. Very shortly the sheaths of the tendons and joint surfaces become roughened by exudation, whereupon a crepitus, hard to distinguish from that of fracture, may be appreciated in a joint merely dislocated.

On reduction, the parts are restored to their normal shape, with a sudden snap, audible frequently to the surgeon. The bones usually remain permanently in apposition, the deformity not returning. Finally, the head of a long bone may be felt moving out of its socket on rotating its shaft, and the axis of the limb will be altered in a corresponding manner to the position of the displaced head. The detection of the head of the bone in an abnormal position is, of course, a quite indubitable sign of dislocation. Anæsthetics are frequently indispensable in making a diagnosis, and examination should always be carried out before swelling has occurred. If the case is not seen for some days, great swelling and extravasation of blood, especially in patients with large muscles or much subcutaneous fat, may render the diagnosis impossible, even by the most experienced surgeons. In these perplexing cases no definite opinion should be given, unless the surgeon be absolutely certain of his grounds.

It is of the utmost importance in all dubious cases, that both limbs be examined and compared in various postures of the trunk. For instance, in suspected dislocation of the hip the patient should be examined standing up, as well as lying down. In examining for dislocation of the shoulder, the patient should stand up, sit down, and be inspected from both before and behind.

General treatment of dislocations.—If a patient be seen immediately on the occurrence of a dislocation, while still faint and collapsed, the surgeon is frequently able to restore the bone to its place with ease. When some hours or days have elapsed in the case of a muscular adult, full anæsthesia is always advisable. I believe ether to be the best and safest anæsthetic for the complete muscular relaxation requisite. Care should be taken to study each case; and the principles of manipulation have for their foundation, relaxation of muscles and ligaments by flexion or rotation, and the making the head of the bone retrace its steps through the torn tissues. In no case is extension with great force to be employed, unless with attention to this principle. For instance: it would be wrong to pull strongly upon a dislocated hip in extension, but considerable force may be exercised upon the femur when the thigh is flexed upon the belly. In old dislocations it must be remembered that considerable risks attend violent efforts at reduction, whereas these cases, even if left alone, recover with useful limbs in time.

The open method, that of cutting down upon old dislocations, dividing opposing structures, and returning the head of the bone to its place, or excising the head of the bone altogether, has been very successful; and if carefully performed, is devoid of the risks attending upon violent efforts at reduction, which are associated with further laceration, perhaps of important structures. These vexed questions of treatment have especially to be considered with reference to the hip and shoulder, in which connection they will again be dealt with.

Importance of considering constitutional peculiarities in joint injuries.—It is a practical rule of utility to study and consider *constitutional diatheses* in the prognosis and treatment of joint injuries. This is perhaps more important in the slighter cases. A delicate young person, the offspring of tuberculous parents, will readily suffer from tuberculous disease of an articulation after a slight blow or sprain implicating the hip, spine, or ankle. So in the rheumatic or gouty, a slight joint injury may be associated with such severe pain and intense local reaction as to lead a superficial observer to imagine that serious mischief was taking place. In such instances appropriate remedies, especially the salicylates, will speedily remove the symptoms. In the syphilitic, periostitis is readily induced by a slight blow, and is very obstinate unless the iodides be administered. The most inveterate chronic joint affections, associated with a tendency to fibrosis and calcification, are found after injury in those who suffer from chronic gout or rheumatism; and prognosis in such should always be guarded.

The treatment of compound dislocation of the larger joints.—Injuries of the joints of the upper extremity are less serious than like injuries to joints of the lower extremity. The age of the patient and his condition of general health are powerful considerations in determining the question of amputation. A healthy country boy, or young adult, will recover from conditions which would be hopeless in the aged or intemperate, or the subjects of visceral disease. Primary amputation for compound injuries at the hip, should always be avoided, if possible. Lesions which make the surgeon's mind lean towards amputation are extensive splintering of the bones, ruptures of the main vessels and nerves, extensive lacerations of the soft parts, or these conditions more or less combined. Simple reduction of a compound dislocation should never be attempted unless the wound in the skin is clean-cut, and there is no extensive laceration.

Compound dislocations of the elbow generally do well, but the joint frequently anchyloses. If primary resection is had recourse to, plenty of bone should be removed in joints like the elbow, where subsequent movement is required. In resections the parts should be very freely exposed, and no separated fragments of bone should be left to necrose and keep up irritation. This is especially important in compound dislocations of the astragalus. Very free drainage should be carried out by larger tubes than those usually employed, and the parts should be repeatedly flushed with hot antiseptic lotions, and frequently dressed afterwards so long as serous oozing persists. The continuous irrigation treatment is here very applicable. No fixed rule can be laid down for the treatment of these injuries. The local conditions and the constitutional peculiarities, the surroundings and occupation of the patient, will all be carefully weighed by a judicious surgeon.

SPECIAL DISLOCATIONS.

Dislocation of the lower jaw. **Causation.**—When the mouth is widely open the condyle of the jaw and the inter-articular cartilage advance as far as the summit of the eminentia articularis. The posterior fibres of the lateral and capsular ligaments check further progress. Sudden muscular action, especially of the external pterygoid, may now dislocate the jaw; and this accident occurs in the acts of yawning, vomiting, or bawling with the mouth widely open. It is also occasioned by violence, as a blow from a fist on the chin when the mouth is open, the action of a gag in mouth operations, and the like.

Morbid anatomy.—As a rule, there is no great laceration of ligaments. The displaced condyle and cartilage are drawn upwards by the masseter and pterygoid muscles, and lodged under the zygoma in front of the eminentia (Fig. 373). It has been stated that the coronoid process becomes hitched against the back of the malar bone; but it is questionable if the process is sufficiently long to become thus fixed, unless the muscles should be much lacerated. The inter-articular cartilage is not invariably displaced with the bone. This accident is most common in women about the middle period of life, and is nearly always bilateral. The displacement forwards is the only known dislocation; but if fracture occur, displacements outwards and also backwards have been related, and the head of the bone has even been driven into the auditory canal.

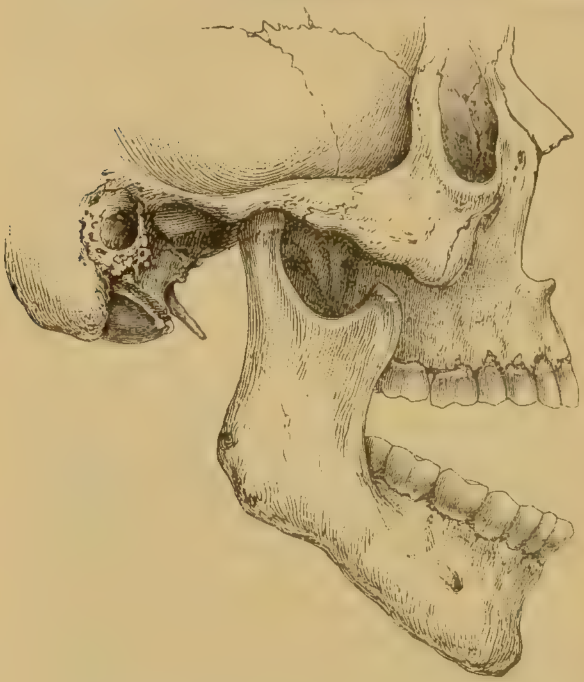

Fig. 373.—Position of Bones in Dislocation of Lower Jaw.

Symptoms.—In the *forward dislocation* the mouth is widely opened, the chin and lower incisor teeth are advanced, the sides of the face are elongated, and there is a marked hollow in the natural position of the condyle, which can be felt anterior to it. The displaced coronoid may be felt by examining through the mouth. The chin is fixed, and cannot be elevated. The masseters and temporals are spasmodically contracted, and their contour is evident. There is much pain from pressure on the auriculo-temporal nerve, articulation and deglutition are well nigh impossible, and the saliva drips copiously from the open mouth (Fig. 374).

In *unilateral dislocation* the symptoms are less marked, and the accident may be overlooked. The mouth is partially opened and less

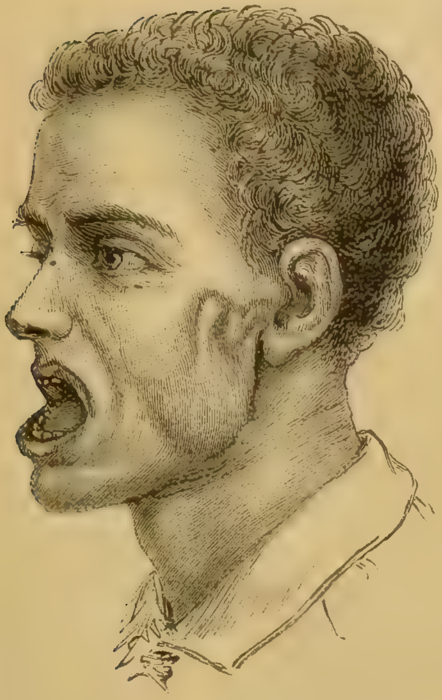

Fig. 374.—Appearance of a Patient with Dislocation of the Jaw. (After Anger.)

fixed. The two guiding symptoms are the hollow over the position of the condyle on the injured side, and the prominence of the chin towards the opposite side to the dislocation.

If these dislocations remain *unreduced*, the parts soon get welded by tough and abundant fibrous tissue, and interstitial shortening of the lacerated muscles affords a powerful obstacle to reduction. Movement is gradually and partially restored, so that a patient can swallow and articulate, but only imperfectly.

Treatment.—In recent cases reduction is simple. The patient being seated in a chair, the surgeon presses with well-guarded thumbs upon the lower molar teeth downwards and backwards, thus disengaging the coronoid process and condyle from beneath the zygoma; at the same time, with his fingers and palms he lifts the chin upwards (Fig. 375). The elevator muscles, should reduction be effected, will close the teeth with a violent snap.

Should the case still prove obstinate, large wedges of cork should be introduced between the molar teeth as far back as possible, and the chin lifted up with the hands, or by means of the strap of a tourniquet passed round the head, or a band-

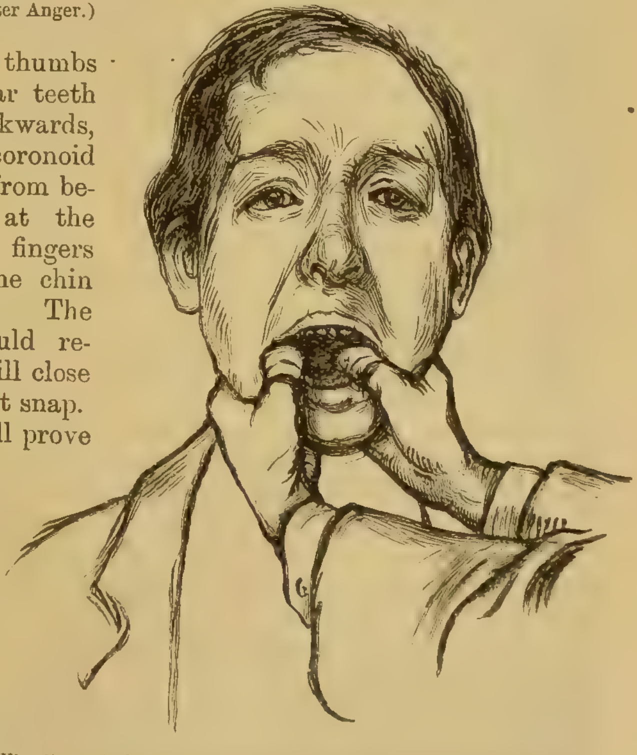

Fig. 375.—Reduction of Dislocation of lower Jaw by depressing the Ramus and lifting the Chin.

age. These are powerful methods. Long wooden levers have been used, and also screw-gags, but it is the depression of the jaw which is the first and most important movement; and unless this can be carried out, elevation is of little use. In ancient cases, reduction may be tried up to twelve months, for if efforts to reduce fail, the breaking of adhesions will improve mobility, and one side may be tried at a time. In old unreduced dislocations, should great distress be experienced, free excision of the condyle should be practised, or division of the ramus of the jaw below the fixed parts.

After reduction of a dislocation of the jaw, the ordinary bandage should be carefully applied, and used for at least a month; and after this the patient should be cautioned against any incautious movement of the jaw, as yawning, which may reproduce displacement, unless the chin be well supported. Feeding is carried out by a tube introduced behind the last molar tooth.

This dislocation is apt to be obstinately recurrent, and great care must be exercised in opening the mouth widely for some months.

Sub-luxation of the lower jaw.—This is a peculiar condition of the temporo-maxillary joint, observed in young people with lax ligaments. On movements, such as opening the mouth, there is a sensation of displacement, with a slight "click," and one side of the face becomes fixed and painful. The parts usually restore themselves on raising the chin, but the condition may so often recur as to be very troublesome. The pathology of this displacement is not founded on dissection, but it has been affirmed that the condyle slips in front of the inter-articular cartilage, or that the extreme laxity of the ligaments allows of an actual dislocation to take place.

Avoidance of exciting causes, improvement of the general health by tonics, with blisters over the articulation, have been generally practised. Time is the great remedy for this affection; and as a patient grows older the tendency to it spontaneously ceases.

Dislocations of the clavicle.

1. Dislocations of the sternal end of the clavicle.

Causation and classification.—These accidents are usually caused by violence applied to the acromial end of the bone. Thus, a severe blow or heavy fall on the front of the shoulder, by forcing the acromial end of the clavicle backwards, will displace the sternal end forwards. Conversely, if a patient be caught between the buffers of railway carriages, and the shoulder be violently forced inwards and forwards, the head of the clavicle may be driven backwards. Direct violence, as the passage of a wheel over the parts, may also drive the sternal end of the clavicle backwards. The rare dislocation upwards can only be produced by violence acting on the outer end of the clavicle from above, and forcing the shoulder downwards and inwards. I have seen spontaneous displacement of the sternal end of the clavicle forwards, from muscular exertion in a weakly lad with lax ligaments. Congenital displacements are also on record. Pathological dislocations forward, from pyæmic abscess or quiet tuberculous processes are comparatively common. Sir A. Cooper notices displacement of the head of the clavicle backwards associated with extreme curvature of the spine, and relates a case where dysphagia and emaciation were thus produced, the head of the bone being ultimately removed with the saw.

Dislocations of the sternal end of the clavicle in their order of frequency occur (*a*) *forwards*, (*b*) *backwards*, and (*c*) *upwards*.

The rhomboid is the strongest ligament of the sterno-clavicular articulation, and its fibres pass upwards, backwards, and outwards. The attachments of this structure permit displacement forwards. The anterior sterno-clavicular ligament is comparatively weak. In abscess of the joint, for instance, displacement forwards readily occurs. The weakness of the anterior ligament explains the frequency of traumatic displacement forwards. The posterior ligament is stronger; hence, displacement posteriorly is more infrequent. The head of the clavicle cannot pass far backwards without rupturing the rhomboid ligament, which needs great violence. In the dislocation upwards the rhomboid ligament must, of course, be torn; hence, this displacement is very exceptional. Some anatomists believe that the inter-articular cartilage forms the strongest bond of union between the clavicle, the cartilage of the rib and the sternum; but a careful examination will lead one to the conclusion that the rhomboid ligament is the more important structure in this connection.

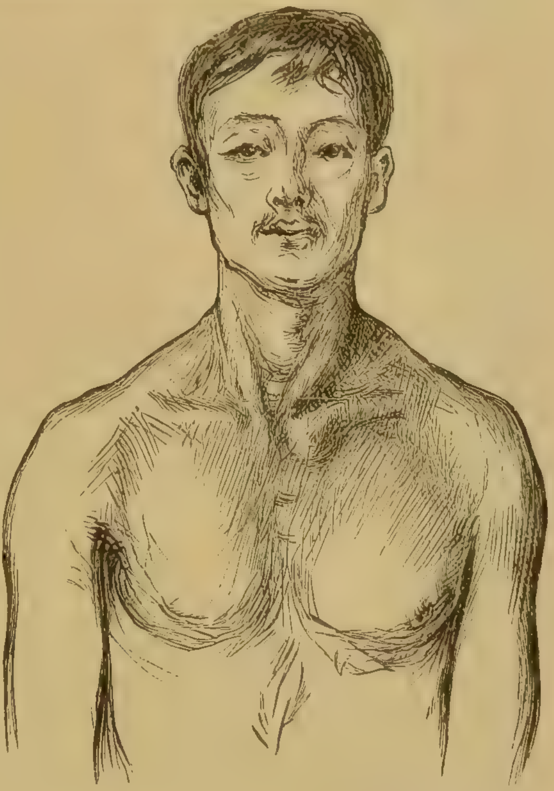

Fig. 376.—Dislocation of the Sternal End of the Clavicle forwards.

(*a*) **The dislocation forwards.** *Morbid anatomy.*—This displacement may be incomplete, the capsule being ruptured anteriorly, and the front of the head of the bone making an undue prominence. When complete, the head of the clavicle is thrown on the anterior surface of the sternum. The ligaments, especially the anterior, must be more or less torn, according to the completeness of the displacement. The inter-articular cartilage is either torn away from the clavicle or the sternum. Unless great violence and marked displacement occur, the rhomboid ligament does not materially suffer.

Symptoms.—The marked projection anteriorly, having the contour by feeling and sight of the articular end of the clavicle, is characteristic of this injury, the diagnosis of which is only difficult in the very obese. On placing a pad in the axilla, and throwing the shoulder outwards, the prominence tends to disappear. On raising the elbow, it is depressed; on depressing the shoulder from above, it ascends. It is devoid of the sharp irregular outline of a fracture close to the sternal end of the bone, which it much resembles. There is considerable pain. The patient's head is inclined towards the affected side, and the distance between the root of the neck and the point of the shoulder is diminished. The clavicular tendon of the sterno-mastoid is prominent (Fig. 376).

Treatment.—Dislocation of the sternal end of the clavicle forwards is reduced by drawing the shoulders backwards, to disengage the bone from the front of the sternum, and especially by throwing the shoulder on the affected side outwards by means of a large axillary pad. To keep the head of the bone in position for a month or five weeks, until the lacerated ligaments heal, is a matter of no small difficulty. If the patient will rest, reclining against a soft cushion with the arm in a sling, and direct pressure be made on the part by a pad—and nothing is better for this purpose than a soft Turkey sponge—the displacement will be to a great extent reduced. Reduction should always be aimed at, in order to give the ligaments a chance of re-uniting. A complete displacement occurring in a muscular and restless individual will probably not remain completely reduced, even with the aid of the most skilled mechanicians. The best plan in such a case is to draw out the shoulder with a large axillary pad, and bandage the arm to the side, advancing the elbow towards the middle line. Direct pressure may be made upon the displaced head by a sponge pad. Nelaton's method of making pressure on the dislocated joint with a spring truss is very difficult to apply in practice. The surgeon should warn the patient of the probability of persistence of more or less displacement; he may also encourage him by the prospect that the limb will be useful. Indeed, little harm accrues if the joint be left permanently displaced.

(*b*) **The dislocation backwards.** *Anatomy.*—Here the head of the bone is situated behind its normal level, either above the sternum or behind it, the head of the bone then lying posterior to the origins of the sterno-hyoid and sterno-thyroid muscles. The trachea or œsophagus may be pressed upon or displaced, and so may the innominate vein or great arteries. The rhomboid ligament and the posterior ligament are more or less torn.

Symptoms.—There is a marked depression at the root of the neck, and the head of the displaced bone may be detected above the sternum by tracing the shaft inwards. There may be great dysphagia or dyspnœa, arrest of the pulse at the wrist, and congestion, from venous obstruction of the side of the head and neck. The distance of the shoulder from the root of the neck is diminished.

Treatment.—This dislocation is reduced by placing a large cushion between the shoulders, and bandaging them backwards over it by a broad figure-of-8 bandage. Reclining against a large soft cushion during the process of repair is very useful. Complete reduction and restoration are seldom to be hoped for. In extreme cases resection of the head of the bone may be called for, or the shaft of the bone may be cautiously divided near its sternal end with a fine saw (Edmund Andrews).

As good a plan as any of treating this dislocation is by bandaging the shoulders to a broad padded splint applied across the back, or to a cushion fixed between the shoulders posteriorly.

(*c*) **The dislocation upwards.**—In this exceptional displacement the head of the bone rests upon the top of the sternum, and passes inwards beyond the median line, in front of the trachea and sterno-hyoid muscles (Fig. 377). The rhomboid ligament, with the anterior and posterior ligaments, must be extensively torn. The sternal portion of the sterno-mastoid will be tense and prominent, the clavicular portion relaxed. The distance of the shoulder from the root of the neck is shortened, and the head of the clavicle can be felt in its new position, following the movements of the shaft, on pushing the shoulder forwards or backwards.

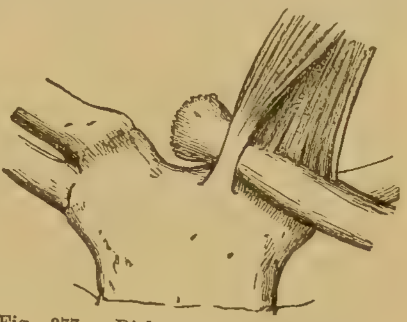

Fig. 377.—Dislocation upwards of the Sternal End of the Clavicle.

Treatment.—By drawing the shoulder outwards with the aid of the axillary pad, and making pressure upon the shaft of the clavicle from above, reduction may be effected. The usual difficulty is experienced in keeping the bone in position. To depress the shaft of the clavicle, a padded belt may pass across the bone, and be secured in front and behind to a waist-band. A band passes from the clavicular part of the belt round the root of the neck, to prevent it from slipping outwards. The elbow and shoulder should be at the same time well raised, to heave up the acromial end of the clavicle, and depress the sternal end.

General remarks on the treatment of dislocations of the sternal end of the clavicle.—The drawing of the shoulder outwards by means of a large axillary pad, together with the supine position, have given, in my experience, better results than any more complicated treatment. The pressure of apparatus on the displaced head is not long tolerated, and the skin soon becomes torn and inflamed. A soft Turkey sponge, fixed in position by well-padded straps and buckles, has seemed a decided aid. So good are the final results of these cases, if the dislocation remains unreduced, that such operations as resection of the head of the bone, or restoration to its place, and fixing it there by a wire suture, can scarcely ever be justifiable.

I have obtained favourable results also by moulding a large piece of guttapercha round the elbow, shoulder, and root of the neck, over a soft flannel bandage and flat sponge pad on the displaced articulation. When the guttapercha has become hard it is pierced by a bradawl, to allow of transudation of perspiration, and is supported externally by a sling and bandages.

2. **Dislocations of the acromial end of the clavicle from the scapula.**—It is right to state that many authors consider this dislocation as a displacement of the scapula from the clavicle, the former being the more movable bone.

Causation and classification.—Dislocations of the outer end of the clavicle are stated to be more common than dislocations of the inner end. The main obstacles to displacement are the strong conoid and trapezoid ligaments. The shape of the articular surfaces would lead one to expect that dislocation *upwards* would be the more frequent; and this is the case. Dislocation *downwards*, the clavicle being driven below the acromion, is also met with, though it is exceedingly rare. The violence which produces this accident almost always acts upon the scapula, and usually from behind. Thus, a man may be pitched from a horse, and falling heavily on the back of his shoulder produces this dislocation. Sub-acromial dislocations can only be produced by very heavy blows on the shaft of the clavicle, driving the acromial end of the bone downwards. I have never seen an instance of this dislocation.

Louie ,

Morbid anatomy.—The capsular ligament is ruptured, and the clavicle is displaced on the acromion for about half an inch. The trapezius is very tense, and tends to reproduce the displacement by its constant traction. The conoid and trapezoid ligaments remain intact in the incomplete displacements; but when the clavicle is thrown for an inch or more on the acromion, the trapezoid ligament, at all events, must be torn. In the displacement downwards the conoid and trapezoid ligaments have been found torn, and the coraco-acromial ligament also (Hamilton). The head of the clavicle lies on the capsule of the shoulder. It has been stated that the acromial end of the clavicle may even be thrown under the coracoid process; but the reputed cases of this strange accident are not universally accepted as genuine by authors.

Symptoms of dislocation of the acromial end of the clavicle upwards.—The arm appears lengthened and hangs by the side, while the shoulder is depressed and nearer the median line than on the opposite side. The prominence formed by the displaced outer end of the acromial end of the clavicle is apparent, and the clavicular portion of the trapezius is exceedingly prominent. The patient is unable to raise his arm over his head.

Treatment of dislocation of the acromial end of the clavicle upwards.—This dislocation is readily reduced by drawing the shoulder outwards, and making digital pressure upon the displaced bone. It is next to impossible to keep the clavicle in permanent position. The extreme upward movements of the arm will be subsequently impeded; otherwise, a very useful joint will result, should the dislocation remain unreduced. The main indications are to keep the scapula raised and the clavicle depressed. Several kinds of apparatus have been advised (Fig. 378). I have seen good results in this dislocation by setting the shoulder in a firm plaster-of-Paris spica, a sponge-pad being fixed over the outer third of the clavicle. Should great inconvenience arise from an unreduced dislocation of this nature, there could be no serious objection to refreshing the joint surfaces and wiring them together. Division of the trapezius, which pulls the bone upwards, is a severe measure; and bandaging the head towards the affected side to relax this muscle cannot long be tolerated.

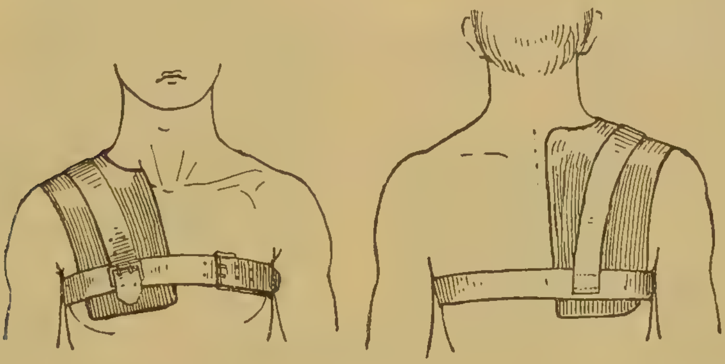

Fig. 378.—Tracy's Apparatus for displaced Acromial End of Clavicle.

Dislocation of the acromial end of the clavicle downwards.—The acromion process projects markedly, and on tracing the clavicle outwards, a marked hollow will be detected where its acromial end normally lies. The patient cannot raise the arm, but the surgeon can move it backwards and forwards. The displacement is reduced by drawing the shoulders backwards, and then bandaging the arm and scapula to the side.

Simultaneous dislocation of the clavicle at both ends.—This accident is one of the curiosities of this part of surgical literature, and is generally produced by extreme violence. The sternal end is thrown forwards, the acromial backwards. Hulke treated a case of this nature by drawing the shoulders well backwards, and moulding a guttapercha splint on the parts, to prevent re-displacement.

Displacement of the scapula.—In weakly individuals the lower angle and vertebral border of the scapula not infrequently project unduly. At the same time, the movements of the arm are well performed, and there is no sign of muscular paralysis. In the special displacement we are considering, the lower angle of the scapula projects in a very unsightly manner from the back of the thorax (scapula alata). This was formerly attributed to the bone slipping from under the latissimus dorsi muscle, but is now known to be generally due to paralysis of the serratus magnus or rhomboids, from "infantile paralysis" or nerve injuries. When the serratus contracts it draws forwards the vertebral border of the scapula; and as the inferior digitations are far the stronger, the lower angle of the scapula is especially rotated forwards, thus raising the superior angle and aiding the functions of the levator anguli scapulæ and trapezius. The most important action of the serratus is to bind the scapula firmly to the ribs, and thus to fix that bone while the deltoid raises the arm. When the serratus is paralysed, the arm cannot be elevated. Efforts on the part of the patient to do so, only cause increased projection of the vertebral border and inferior angle of the scapula, which assumes a very peculiar and characteristic appearance.

No treatment beyond a padded belt, fitting over the scapula round the thorax, and supported by shoulder-straps, is of any great use. The nerve conditions should, of course, receive consideration and treatment.

Dislocations of the shoulder. **Causation.**—The exposed position of the shoulder-joint, the shallowness of its articular surface, the laxity of the capsule, and the powerful leverage exerted through the long humerus, cause displacements of this articulation to be more common than those of any other joint in the body. The majority of these dislocations are caused by indirect violence, as falls on the elbow or hand, when the limb is abducted, and the position of the long axis of the humerus at the moment of displacement largely determines the course and position of the head of the bone.

Direct violence, as heavy falls or blows on the shoulder, is asserted to cause dislocation, though it is difficult to understand how this can occur; and muscular action, as striking out at and missing an adversary, swimming, the convulsive efforts of epilepsy, or even such slight efforts as the act of sneezing, have been known to produce displacement. Exceptional causes of dislocation are dragging upon the limb by the pulling of a runaway horse, or the twisting of an entangled upper limb by revolving machinery. Congenital displacements are sometimes met with, and pathological displacements also, in chronic rheumatism or pyæmic disease.

Classification.—Considerable difference of opinion exists as to the comparative frequency of these displacements. The majority of surgeons in England will endorse the following classification, which is similar to that founded on an extensive inspection of museum specimens by Holmes and Hulke:—

(1) Dislocation forwards and a little downwards—*subcoracoid.*
(2) Dislocation downwards and a little forwards—*subglenoid.*
(3) Dislocation forwards and inwards—*subclavicular.*
(4) Backwards—*subspinous.*
(5) Upwards and forwards—*supracoracoid.*

As in the case of the hip, sub-varieties of these displacements have been described by anatomists, which rather tend to confuse the student than to serve any useful purpose. There is no doubt that the causes which produce these injuries being diverse, and the nature, extent, and application of the forces at work composite, slight differences in position of the head of the bone will be found in different cases. For the same reasons, the amount of laceration of the soft parts in each displacement is not constant, and the obstacles to reduction may slightly vary. It follows that differences may be appreciated in the symptoms of any one given dislocation of the humerus, and that methods of reduction applicable to one case of a given dislocation, are not necessarily applicable to another.

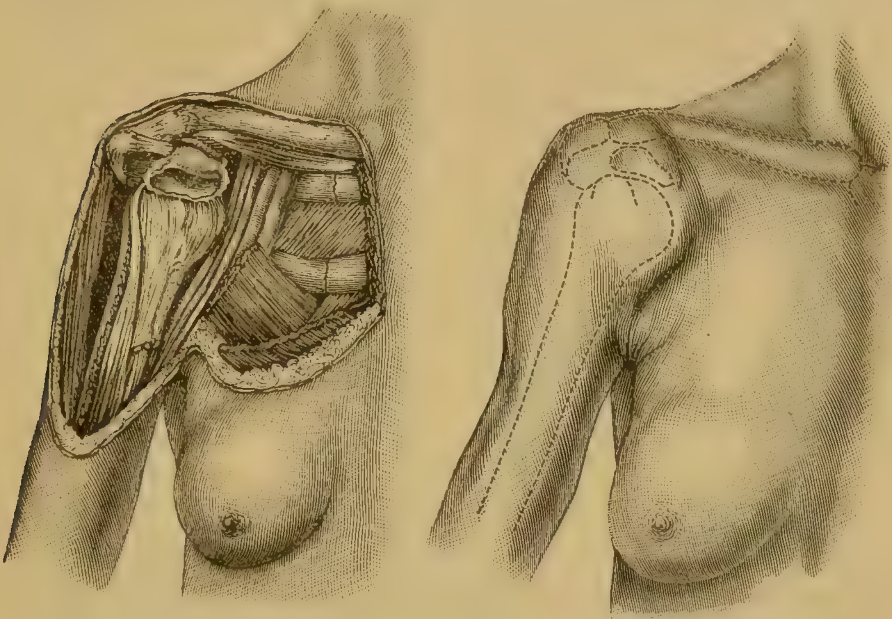
Fig. 379.—Dislocation of the Humerus beneath the Coracoid Process. (After Anger.)

1. **Subcoracoid dislocation of the humerus.** *Causation.*—Direct violence applied to the shoulder, as a heavy fall or blow; a fall upon the elbow or hand, when the limb is abducted from the body; muscular action.

Morbid anatomy.—The head of the humerus is thrown forwards and very slightly downwards, resting upon the anterior surface of the neck of the scapula, while the anatomical neck impinges upon the anterior edge of the glenoid fossa (Fig. 379). So long as the head of the humerus lies beneath the coracoid process in any part, the term subcoracoid may be applied to the displacement. The subscapularis muscle may be torn, or stretched across the joint or the head of the humerus, which, however, is usually markedly above it. The supraspinatus, infraspinatus, and teres minor muscles are stretched or ruptured, or torn away from the bone; the great tuberosity may be detached from the shaft. The pressure of the head of the bone renders tight the biceps and coraco-brachialis, and presses upon, or even ruptures, the circumflex nerve, the cords of the brachial plexus, or in very severe cases the main vessels. The capsule is torn below, the rent running anteriorly, or the lower part of the capsule may be torn away from its glenoid attachment. The long tendon of the biceps usually escapes rupture.

Symptoms.—It will be convenient here to detail the symptoms which are common to all dislocations of the shoulder. They are: (1) A depression immediately beneath the acromion, with an undue prominence of that process; (2) pain about the part, and more or less immobility, though this latter symptom is varied and inconstant in practice; (3) alteration in the axis of the limb, and the detection of the head of the bone rolling in an abnormal position; (4) the sign of Dugas. If the fingers of the injured limb be placed and maintained upon the sound shoulder, the elbow, in dislocations, cannot be brought against the side. The sign of Dugas depends upon the principle that a straight line cannot touch a circle at more than one point at a time. When the humerus is dislocated it is laid along the barrel-shaped thorax, and must obey the rule of the straight line and circle. In practice, this sign is extremely fallacious, for the patient usually drops his body towards the painful and affected side. It is most important in this, as in all diagnostic matters connected with the shoulder, that the patient should sit upright against a hard-backed chair, and be well stripped. Obesity, a prominent abdomen, or great mammary development, makes the test of Dugas dubious and difficult; and as it is in such cases that its additional testimony is needed, it is difficult

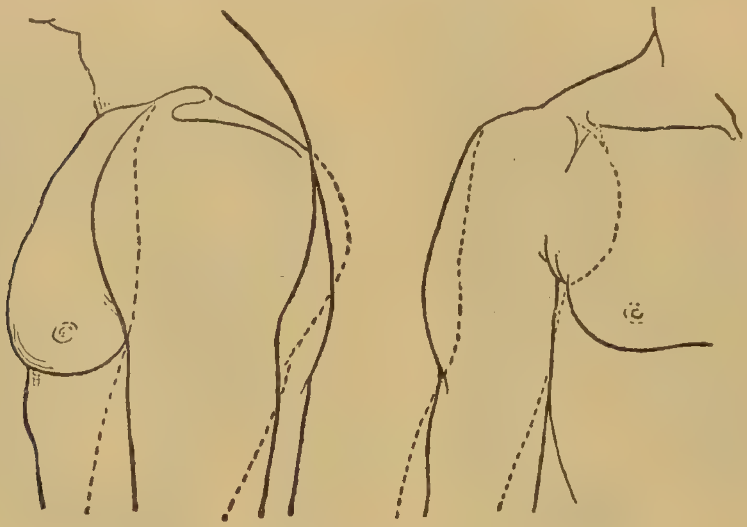

Fig. 380.—Subspinous and Subcoracoid Displacements. The dotted line shows the altered outlines of the limb.

conscientiously to assign it a high practical value. (5) An increase in the circumferential measurement of the shoulder taken vertically; (6) if a rule be placed along the outer side of the arm it will impinge against the point of the acromion. (*See* Fig. 380.)

The special symptoms of subcoracoid displacement, in addition to the above, are as follows: The elbow is carried backwards, and is directed away from the side. The limb appears lengthened, but on measurement from the tip of the acromion to the external condyle, this will be difficult to verify. On deep palpation in the axilla, the upper part of the shaft will be felt, and on raising the elbow the head of the humerus will be detected anteriorly and internally; while at the same time the hollow under the acromion is more evident to sight and feeling. There is a prominence beneath the coracoid, and severe "numbing" pain down the fore-arm and in the fingers is often experienced in this dislocation.

2. **Subglenoid dislocation of the humerus.** *Causation.*—A fall on the abducted limb and outstretched hand, or a heavy blow upon the upper and outer end of the humerus (Hamilton).

Morbid anatomy.—This dislocation was long thought to be far more common than is really the case. It may be looked upon as a variety of the former dislocation, in which the head of the bone rends the capsule inferiorly, but the muscles fail to raise it beneath the coracoid, on account of the capsule not being torn anteriorly. The head of the bone lies beneath the glenoid fossa on the triangular surface, and origin of the long head of the triceps (Fig. 381). The tendon of the subscapularis is now above the head of the bone, the triceps is behind it, and the teres major below.

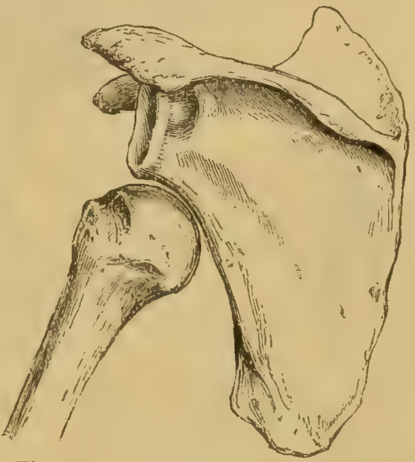
Fig. 381.—Subglenoid Dislocation of the Humerus.

The structures just named may be lacerated or stretched. The short muscles attached to the tuberosity may be torn, or even that process wrenched off. The vessels and nerves are invariably pressed upon, and the circumflex nerve is very likely to be seriously damaged.

Special symptoms.—The arm is really lengthened, and the elbow is markedly thrown away from the side, to which it cannot be approximated without much difficulty. The hollow beneath the acromion is very marked, and the head of the bone is felt with ease in the axilla. The fingers can be passed beneath the coracoid, above the head of the bone. The anterior axillary fold is so markedly lowered as to furnish the most characteristic inspection symptom.

Luxatio erecta.—This is a most rare variety of subglenoid dislocation. The arm is abducted and raised, the axis of the humerus being directed upwards and outwards. This accident seems

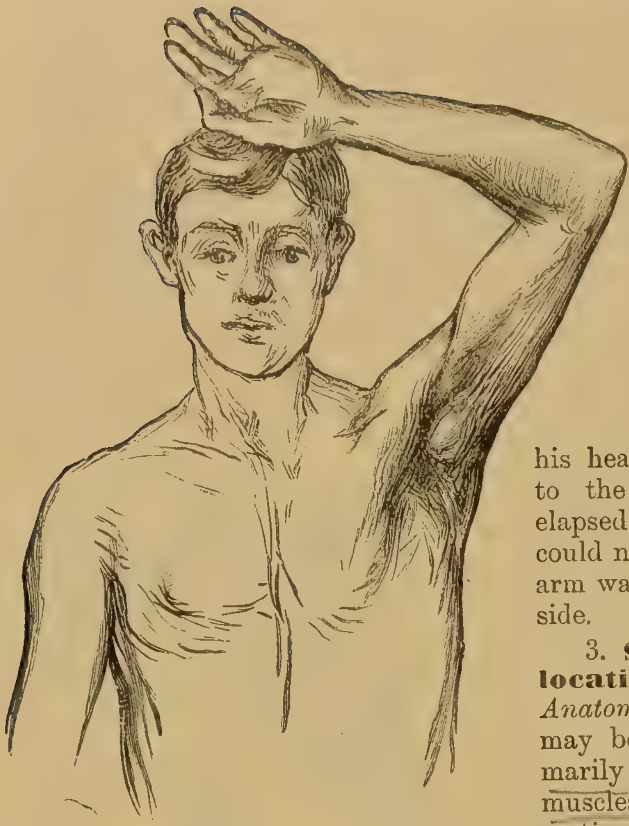

Fig. 382.—Luxatio Erecta

to be produced by a fall, the patient clutching something in his descent. In one of Hulke's interesting cases of this dislocation, a baker fell through a trap-door, and seized a ladder to save himself. He was constrained after the dislocation to keep his hand near the top of his head (Fig. 382). Owing to the interval which had elapsed complete reduction could not be effected, but the arm was brought down to the side.

3. **Subclavicular dislocation of the humerus.** *Anatomy.* — This dislocation may be looked upon as primarily subcoracoid, but the muscles, and perhaps the direction of the force, cause the head of the bone to pass under the clavicle to the inner side of the coracoid process (Fig. 384). Here it lies on the upper ribs beneath the pectoral muscle, forming a manifest prominence. The capsule is torn inferiorly and anteriorly; and the muscles attached to the great tuberosity must be torn, or that process separated from the shaft.

Special symptoms. — The arm is shortened, and the elbow thrown backwards and (usually) markedly outwards. The head of the bone can be felt and seen below the clavicle; and on deep axillary palpation the shaft of the bone can be felt crossing the cavity upwards and inwards to the displaced head (Fig. 383).

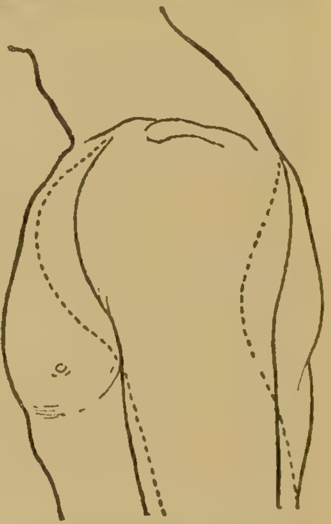

Fig. 383.—Subclavicular Dislocation. The dotted line shows the altered outline of the limb.

4. **Subspinous dislocation of the humerus.**—In this exceptional dislocation the violence must be applied

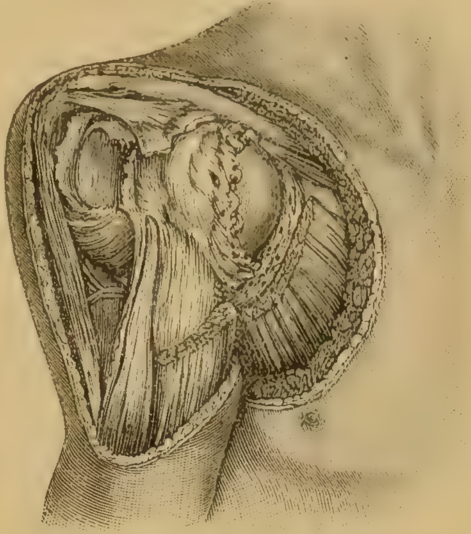

Fig. 384.—Subclavicular Dislocation. (After Anger.)

to the arm when thrown forwards, and away from the side; or it may be produced by a violent blow upon the front of the shoulder. Sir A. Cooper relates a case from the muscular spasms of epilepsy. The direction of the force would seem more potent in producing the dislocation than muscular contraction. The head of the bone may rest anywhere between the posterior edge of the glenoid cavity and the infraspinous region (Fig. 385). The capsule is torn below, and the subscapularis is usually completely ruptured.

Special symptoms.—The elbow is advanced, and the arm (usually) rotated inwards; the arm is close to the side also. There may be slight lengthening. There is a marked hollow beneath the coracoid process, and a prominence under the spine of the scapula, formed by the large globular head of the humerus, which, being readily seen and felt, affords a characteristic sign of the displacement (Fig. 386).

5. **Supracoracoid dislocation of the humerus.**—This very rare displacement may be shortly dismissed. It is caused by violence in an upward direction applied to the shoulder or elbow. The acromion or coracoid processes are usually fractured, or a longitudinal fracture separating the great tuberosity has also been disclosed on dissection. "The possibility of this form of dislocation without fracture is now definitely established by dissection" (Hamilton).

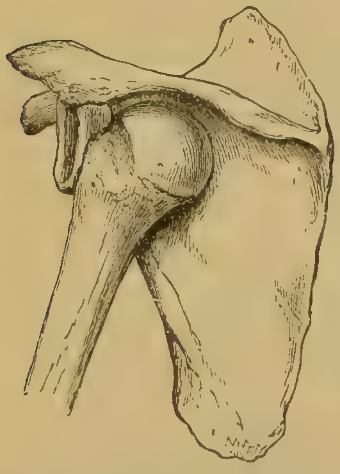

Fig. 385.—Subspinous Dislocation of the Humerus.

Partial dislocations of humerus.—The remarks on page 955 especially apply here, and while the possibility of this accident is not to be positively denied, yet the majority of the recorded cases are obviously severe sprains associated with rupture or displacement of the biceps tendon, or of one or more of the short muscles, or of paralysis of muscles from injury to the circumflex nerve or central cord lesions.

Difficulties in the diagnosis of shoulder dislocations.—In thin or muscular subjects, when seen immediately after the accident, the diagnosis of dislocation can readily be made at a glance. In the very fat, whose muscles are large and flabby, a subcoracoid dislocation is overlooked with ease, and professional reputations are wrecked upon this shoal with unhappy frequency. I would make it a rule to examine the shoulders of any aged and obese person who has had a fall upon the hand or elbow, or point of the shoulder; for such individuals, especially when alcoholic, are often quite unaware of dislocation having occurred. When these cases are treated as bruises or sprains, the swelling subsides in some weeks, the muscles atrophy, and the familiar outlines of an unreduced dislocation are revealed with painful distinctness. I would draw forcible attention to an anatomical point of extreme value, to which my notice was first directed by Sir George Humphry. The great tuberosity of the humerus is prominent beneath the acromion, and projects somewhat externally to it, and is there readily to be seen and felt on raising the arm from the side. If the great tuberosity be in its proper position, and this can be detected even in fat shoulders, the head of the humerus must be in the glenoid cavity. Exceptions must be made when the great tuberosity is separated from the dislocated head, and the converse does not necessarily hold true that when the tuberosity has sunk inwards dislocation must have taken place; for in fracture of the anatomical neck of the humerus, with displacement, this may occur, the head still remaining in the glenoid fossa. The latter condition is so rare, however, as hardly to need mention, and I do not hesitate to look upon the position of the tuberosity of the humerus with regard to the acromion, as the key to the question of dislocation of the shoulder.

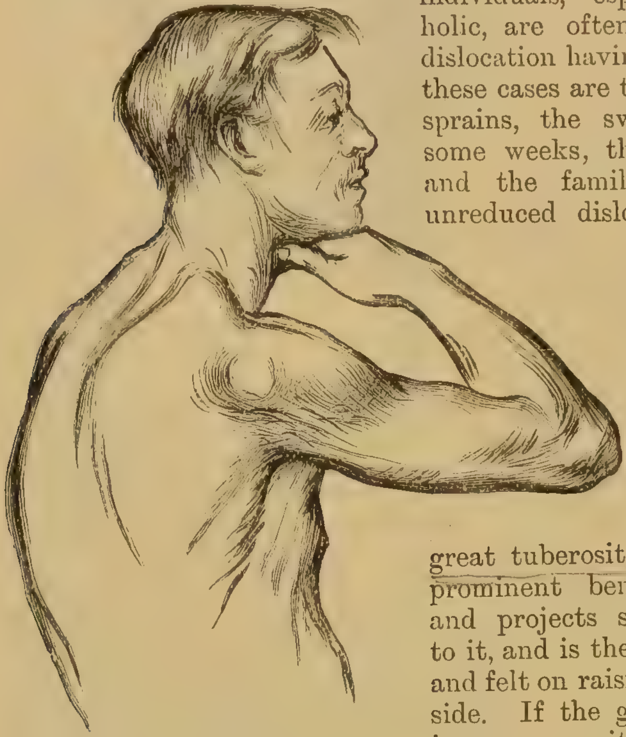

Fig. 386.—Attitude of the Arm in Subspinous Dislocation.

The conditions which most closely simulate dislocations are as follows:—(1) Atrophy of the deltoid muscle from injuries to the circumflex nerve, or too long fixation after accident, or in disease of the articulation; (2) separation of the upper epiphysis of the humerus; (3) fracture of the surgical neck of the humerus; (4) fracture of the anatomical neck of the humerus; (5) fracture of the neck of the scapula; and (6) fracture of the great tuberosity.

Confusion frequently arises regarding atrophy of the deltoid, especially as this condition may follow a properly reduced dislocation. The marked flattening of the shoulder closely simulates displacement, but the great tuberosity of the humerus will readily be felt beneath the acromion projecting in its normal position, and passive motion may be free.

Separation of the epiphysis should be suspected if an apparent dislocation of the shoulder be brought to the surgeon in a patient under twenty years of age. The line of separation will be below the great tuberosity, which is part of the upper epiphysis, and maintains its normal position. There is an abrupt projection in front, below the coracoid process, and on examination and reduction under anæsthetics a sort of crepitus is experienced, and the displacement is readily reproduced. Such an accident in a young person may be followed by arrest of growth, so the diagnostic distinctions between it and dislocation are of obvious import.

In fracture of the anatomical neck of the humerus there is crepitus high up in the joint, and if impacted the head will move in the glenoid cavity. In fracture of the neck of the scapula the coracoid process will move with the fragment of scapula. All the symptoms of dislocation will be exactly simulated; but on reduction crepitus will occur, and the coracoid will be observed to move as the scapular fragment is rotated with the humerus. In separation of the greater tuberosity there is an extraordinary appearance of widening anteriorly, which is very characteristic. A deep sulcus exists between the fragments, and crepitus can always be elicited on rubbing the fragments together. The depression below the acromion in fracture of the surgical neck of the humerus at first sight simulates dislocation, but crepitus is evident, and the depression is not immediately beneath the acromion, but some inches lower. The tuberosity occupies its proper position. Careful examination under ether is absolutely essential in all dubious cases, and this should be done as soon as possible after the accident. (*See also* page 814.)

Treatment of recent dislocation of the shoulder.—If a patient be seen within an hour of the accident, while still, perhaps, pale and collapsed from pain and shock, the surgeon may be able to replace the bone by extension with the heel in the axilla, without anæsthetics. In cases of longer standing and in muscular individuals, deep ether anæsthesia had better be induced. The patient should recline upon a couch with a raised back, or be well propped up with pillows, or placed in an arm-chair. As the subjects of these dislocations are often muscular and alcoholic individuals, with their stomachs full of food, great care should be exercised in the administration of an anæsthetic in the sitting posture, and in my judgment ether should always be selected. The stomach may, with advantage, be previously cleared by an emetic. The muscles should be rendered completely flaccid. Care should be taken to ascertain that the displacement is really a recent one. I have seen and read of unavailing attempts at reduction being made upon the ancient dislocation of a person rendered stupid from alcohol or head-injury.

Every practical surgeon knows that when the muscles are completely flaccid, a dislocation of the shoulder will frequently "slip in," on such slight manipulations as flexing the fore-arm, and rotating the arm outwards or inwards. This must be taken into account in estimating the vaunted infallibility of any one method. The chief modes of effecting reduction will now be described.

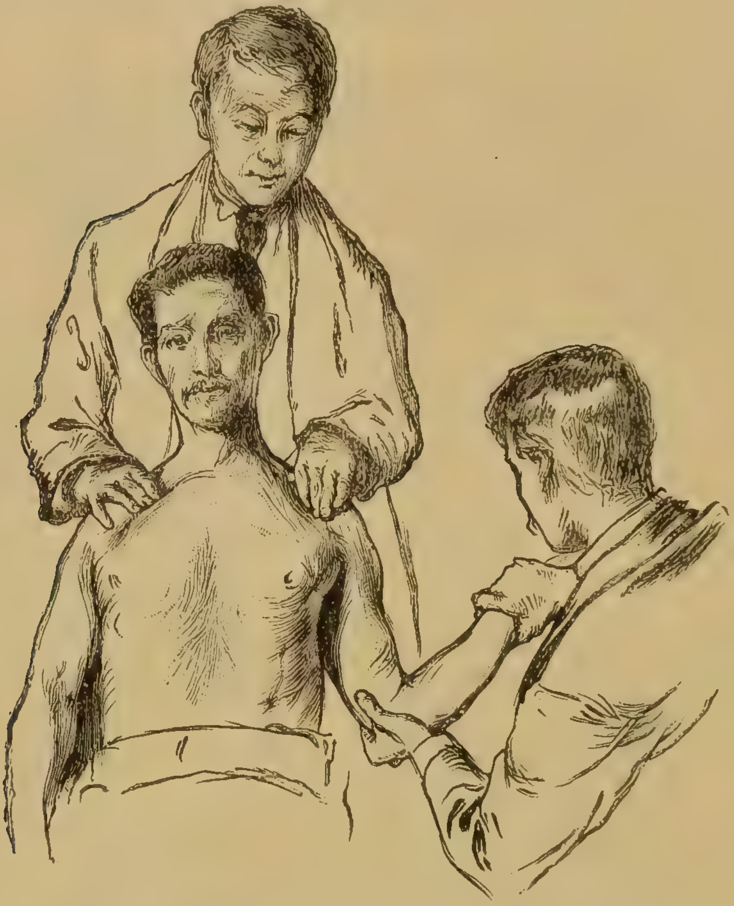

Fig. 387.—Method of Kocher. Abduction and external rotation.

1. **Method of Kocher.**—This is of peculiar value, and is commonly now adopted in the hospitals of England. It will be observed that it combines external and internal rotation, which manipulations are especially valuable in the reduction of dislocations. In the first part of this manipulation the fore-arm is bent, and the surgeon, grasping the wrist and elbow, abducts the limb and rotates it outwards until marked resistance is encountered and a distinct swelling

appears beneath the deltoid. Secondly, the elbow is brought forwards, upwards, and inwards until it is opposite the median line, but still maintaining external rotation and abduction of the wrist. Thirdly, the arm is rotated inwards, the hand being carried towards the opposite shoulder. These movements should be executed in a continuous sweep, and not with "jerky intervals" (Figs. 387, 388, 389).

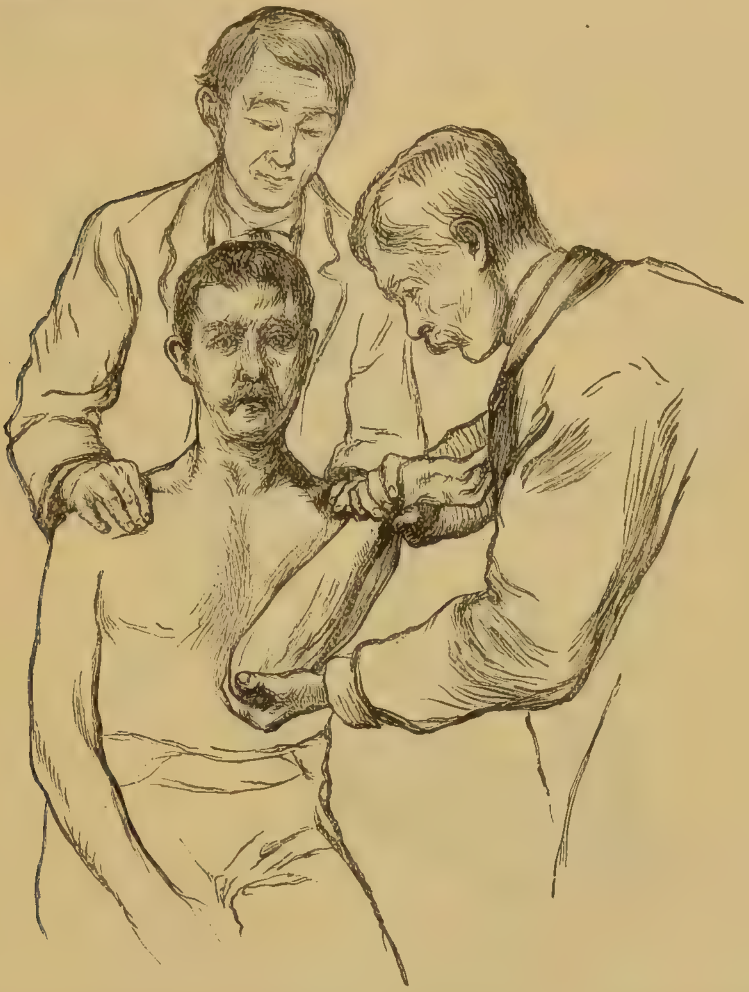

Fig. 388.—Method of Kocher. Advancement of the elbow forwards, upwards, and inwards; still maintaining external rotation.

Explanation of Kocher's method.—When the fore-arm and arm are rotated outwards, the upper part of the capsule and the coraco-humeral ligament are also twisted outwards, and the posterior and untorn part of the capsule is removed from the glenoid fossa, while the gap inferiorly gapes wider and wider as the external rotation is proceeded with. When the arm is raised and carried towards the median line, the head of the bone passes from the edge of the glenoid fossa through the gap. For this latter manipulation relaxes the upper part of the capsule, but renders tense the untorn fibres of the lower part, so that the head of the bone cannot move forwards. Rotation inwards completes the position of the head of the bone.

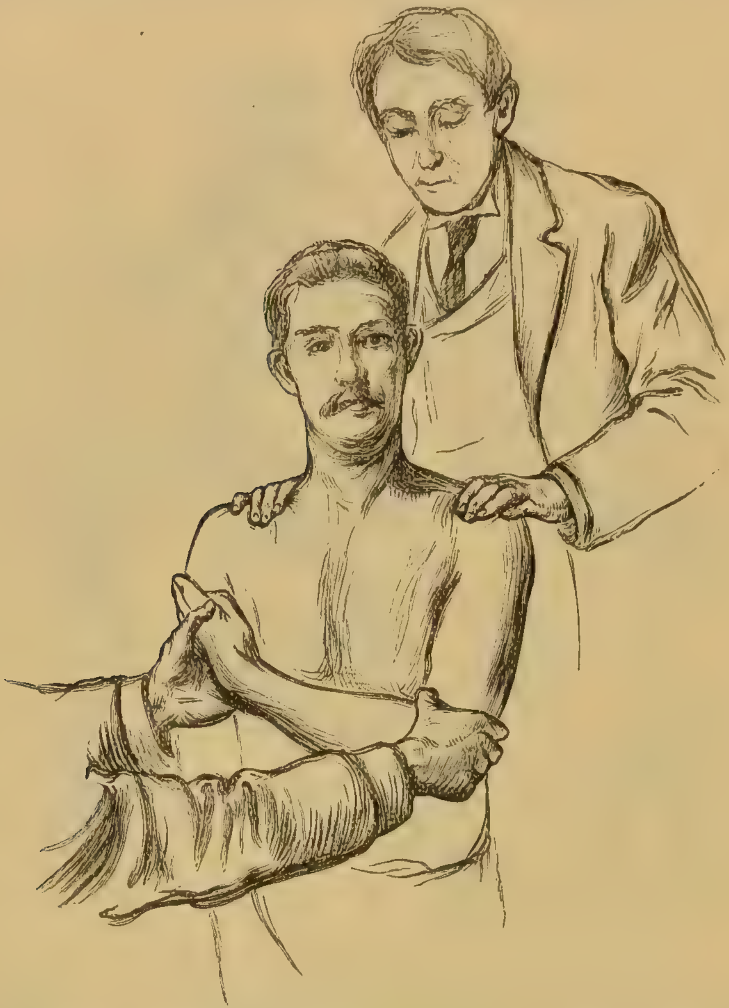

Fig. 389.—Method of Kocher. Rotation inwards, the hand being carried towards the opposite shoulder.

2. **Traction with the knee in the axilla.**—This is a powerful and efficacious method. The patient being secured by broad bandages in the sitting or reclining position, the surgeon brings his knee well into the axilla against the head of the bone. Assistants now make extension outwards from the fore-arm or arm with or without the aid of a strong towel. At a signal from the surgeon the direction of the force is lowered, he, at the same time, bending down the humerus over his knee (Fig. 390).

3. **Extension with the heel in the axilla.**—This simple and valuable method is often bungled in practice; but if properly carried out, I would venture to look upon it as the simplest and most reliable method of reducing dislocations of the humerus. The anæsthetised patient must lie perfectly flat on a mattress, on the floor, and the surgeon, seated by his side, presses his unbooted heel, not his foot, against the head of the humerus, the arm being well carried from the side to allow of his doing so. It is of importance that he presses up no folds of skin in front of his heel, or serious laceration may readily occur. Traction is now made downwards and outwards, and at the same time the surgeon swings the humerus inwards, using his heel as a fulcrum. An additional assistant may easily draw upon the fore-arm, should this be required; and a strong towel may be fixed round the arm and hitched over the surgeon's shoulders. Few, if any, recent dislocations will resist these endeavours.

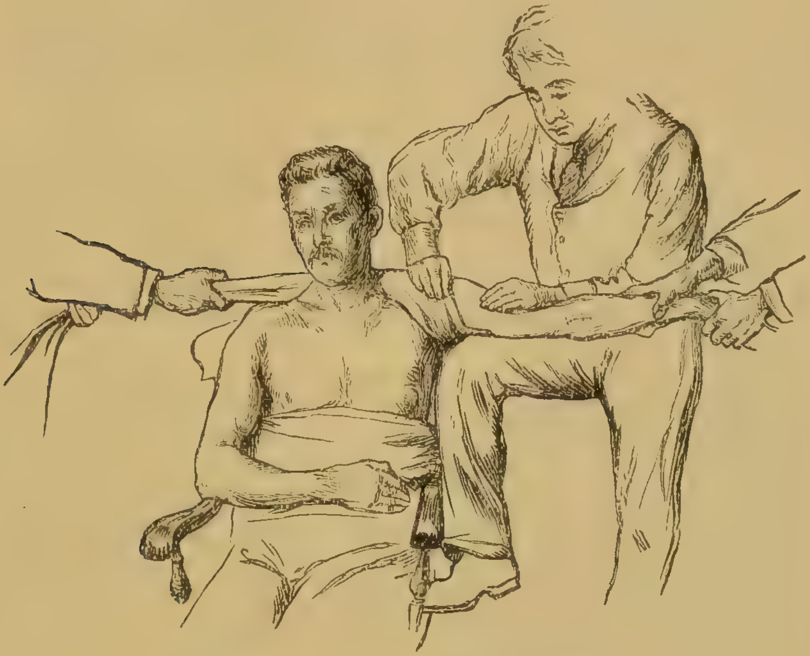

Fig. 390.—Traction with the Knee in the Axilla.

4. **Other methods of reduction.**—These are very numerous, but in this section we will only mention the following:—

Extension of the limb, the scapula being pressed downwards by the hands of an assistant or the foot of the operator. This is an efficacious method, but throws a serious strain upon the axillary vessels, especially in dislocations of long standing. For this reason it should be adopted with caution. The patient being placed perfectly supine upon a mattress laid on the table, the surgeon makes steady traction upon the arm and fore-arm for about ten minutes, the limb being nearly at right angles to the trunk. As the traction proceeds, the arm is raised nearer and nearer towards the head, but is, of course, carefully maintained in the same horizontal plane as the body. The scapula must be well pushed downwards, and fixed as much as possible by an assistant, who takes up his position on the opposite side to the surgeon, and facing the patient. He keeps hold of the patient's trunk, and introduces thereby a counter-extending force. In this manipulation the limb is kept horizontal throughout. The muscles usually cease to resist from the long-continued traction, and the limb being suddenly brought down against the side, reduction is effected. With some surgeons it is a very favourite method, and the principle is to tire the muscles by long-continued traction.

Simple flexion of the fore-arm, with external or internal rotation, is employed by many surgeons. Kelly, of Dublin, advises that the patient lie upon the edge of a low bed; the surgeon insinuates his hip into the axilla, and draws the affected limb across the front of his own pelvis, and round to the opposite hip. He now slowly rotates his body outwards, and makes traction upon the wrist of the dislocated limb at the same time, thus prising the head of the bone outwards to its socket.

Methods of treatment of subspinous and subclavicular dislocations.—These displacements are far more difficult to reduce than those into the axilla; and in a case of subclavicular dislocation seen by the writer, when five days had elapsed before treatment was commenced, all efforts at restoration of the parts were quite unavailing. The first principle of treatment of these dislocations seems to be to make extension with rotary movements in the axis of the dislocation, until the head of the bone is brought close to the glenoid cavity, and presumably below it, when flexion of the fore-arm, and external or internal rotation, or extension with the heel in the axilla, will probably replace it.

Treatment after reduction.—After the reduction of a dislocation of the humerus the limb may long remain feeble, with greatly impaired movement at the shoulder, especially in the upward direction. This is due to the weakness of the deltoid and the cicatricial contraction of the lower part of the capsule. If the circumflex nerve has been stretched or ruptured the deltoid will waste, and in some dislocations one or more of the axillary nerve-cords may be injured, with permanent damage to groups of muscles of the upper extremity. When the great nerve trunks are much bruised, muscular wasting will persist for many months, and yet under treatment recovery may ensue. In some cases traumatic neuritis sets in, and this is marked by atrophy of the hand and fore-arm, with contractions of the joints, "glossy" skin, ulcerations about the terminations of the digits, modifications of local temperature, and severe burning pains.

After reduction of a dislocation the arm should be bandaged to the side, over a large, soft axillary pad, and the fore-arm supported in a large sling, while spirit lotion is applied over the joint. When a week has elapsed, massage and movement should be cautiously commenced, and continued daily with gradually increasing boldness. After a fortnight the fore-arm may be confined in a sling merely, and in a month the patient should be encouraged to use the limb cautiously. The prudent surgeon will see to all this himself. Should atrophy of muscles ensue, he will probably be unjustly blamed, and he should be exceedingly cautious in prognosis accordingly.

Treatment of dislocation of the humerus, associated with fracture of the shaft of the bone.—This accident is, fortunately, as rare as it is embarrassing. The surgeon is sometimes enabled to replace the head of the humerus by manipulation of the upper fragment, with direct digital pressure in the axilla, on the displaced head.

Should this fail, the case must be treated as a fracture. When the bone has united—if the surgeon resort to the treatment usually advised of endeavouring to reduce the dislocation—he will run the inevitable risk of refracture, and of failure to return the head of the bone to its place. Excision of the fragment has also been performed, and a false joint has been fashioned by moving one fragment upon another.

Recently, McBurney, of New York, has treated one of these cases most successfully, by exposing the upper fragment, drilling it, and inserting a strong hook. By traction upon the hook, reduction was readily accomplished. This method seems likely to prove very useful for these troublesome cases.

Treatment of old dislocations of the shoulder.—The pathology of these cases has been sufficiently indicated on page 953. A new cavity is soon formed by pressure-absorption, and interstitial shortening of the muscles, with fibrous thickening forming a spurious capsule, is an early phenomenon. The adhesion of the vessels and nerves to the lacerated capsule is a matter of the utmost surgical importance. Many unreduced dislocations of the shoulder, if energetically treated by galvanism, daily massage, and movements, become surprisingly useful; but such treatment must be persevered in for many months. No hard-and-fast line can be laid down for the time when it is, or is not, justifiable to attempt reduction. The age and vigour of the patient, and especially the condition of his vessels as to atheromatous disease, must be carefully taken into account. As a rough rule, when a dislocation has been left unreduced as long as it would take a fracture of the humerus to firmly unite (about six weeks), great trouble will be experienced in reducing it, and every week that elapses after this time increases the difficulty markedly. The necessary force that is used in some of these cases, may result in the following lamentable complications:—

(1) Rupture of the vessels, diffuse axillary aneurysm.

(2) Extensive effusion of blood from laceration of the tissues, with subsequent diffuse suppuration.

(3) Laceration of the skin, with erysipelatous inflammation.

(4) Injury of the nerves.

(5) Fractures of the shaft of the humerus, or of the ribs.

(6) In old and feeble persons, death from shock, or cerebral symptoms, suggestive of thrombosis or embolism.

Lastly, after prolonged and exhaustive efforts, the surgeon may quite fail in his endeavour to restore the head of the bone to its place, though he has exposed his patient to one or more of the above dangers, and this, in my experience, is the common result of attempts to reduce old dislocations of the shoulder.

The resources of modern aseptic surgery have rendered open arthrotomy very advantageous in some of these cases. The short muscles and all resistant structures are freely divided, the vessels and nerves peeled from the capsule with a rugine, and the head of the bone restored to its place. The results of this proceeding have been excellent. The operation may be most difficult, and it is astonishing how the head of the humerus remains fixed, even when the ligaments and short muscles are extensively divided. No surgeon should lightly undertake it. Excision of the head of the bone is easier, attended with less operative interference, and gives admirable results. Great care should be taken not to divide the circumflex artery too close to the main trunk, or it may be impossible to arrest the bleeding without ligature of the axillary vessel or amputation. The special indication for open operations on old dislocations of the shoulder is nerve pressure from the head of the bone, with atrophy and weakness of the limb. Here great care must be exercised to discriminate between the symptoms of nerve pressure and nerve rupture.

Recurrent dislocations of the humerus.—It sometimes happens, especially after the reduction of dislocations of old standing, that the displacement persistently recurs on the least muscular exertion, so that the patient's life becomes a miserable one. The explanation of these cases is, that there remains a large unhealed rent in the capsule, or great laxity of it, or that a portion of the edge of the glenoid cavity is broken away, or a fracture of the neck of the scapula may complicate the dislocation. Joesoel, of Strasburg, affirms that by dissection he has been able to establish the fact that these dislocations are due to permanent separation of the tendons from the tuberosities. In these cases, a mechanician may adopt a suitable apparatus to support the shoulder. Should this fail, various methods of treatment are advised, many of which are fanciful and some positively harmful. These call for no mention here.

Excision has been performed by Volkmann and others, and a remarkably successful case has been related by Southam. This operation is likely to give definite and permanent good results, and I do not hesitate to advise it in obstinate cases of recurrent dislocation.

Compound dislocations of the humerus.—These are rare accidents, and the great violence that produces them would seldom leave the soft parts in such a condition that a surgeon would be satisfied with simple reduction. Primary resection of the head of the bone, with free flushing and drainage, would be likely to give good results.

Congenital dislocations of the humerus.—Care must be taken not to confound these cases with traumatic luxations or separations of the epiphysis, which may occur during the manipulations of obstetricians in complicated labours. The dislocations are commonly symmetrical, and may occur in any direction, but the subcoracoid seems the most frequent. The muscles round the joint are usually paralysed, the capsule weak and elongated, the head of the humerus altered in shape and size, and the bone much stunted in growth. The glenoid cavity is absent or ill-formed, and another cavity is found continuous with, or contiguous to it, where the deformed head of the humerus imperfectly articulates. Hitherto all treatment of these cases has been unsatisfactory.

Dislocations of the elbow joint. *Classification.*—It is possible to imagine a considerable number of dislocations at this joint, owing to the different directions in which the bones may be thrown, and the fact that two bones may be separately or conjointly implicated. Only the more common and important dislocations will here receive notice.

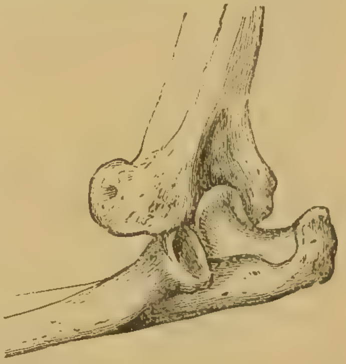
Fig. 391.—Dislocation of both Bones of the Fore-arm backwards.

The radius and ulna may be dislocated together, backwards, inwards, outwards, or forwards. They may be displaced in different directions, or either bone may be dislocated separately. All these displacements vary in degree of completeness.

The various dislocations of elbow may be arranged in order of frequency as follows :—(1) Dislocation of the radius and ulna backwards, (2) outwards, (3) inwards, (4) or forwards, (5) displacement of the ulna alone, (6) displacement of the radius alone, (7) displacement of both bones in different directions.

1. **Dislocation of the radius and ulna backwards.** *Causation and morbid anatomy.*—This accident is commonly caused by falls upon the palm of the outstretched hand. It is frequent in early life, and this is probably due to the comparatively small size of the cartilaginous coronoid process, which is an outgrowth from the shaft of the ulna. The weakness of the anterior and posterior ligaments, the absence of muscular aid, and the want of mutual support between the bones of the fore-arm and humerus, in the flexed or extended position, are all reasons for the frequency of anterior-posterior displacements (Fig. 391).

Pathology.—The anterior ligament is completely torn, and the lateral ligaments generally lacerated. The biceps tendon is tense, the brachialis anticus excessively tense or torn. The triceps tendon, carried backwards, is prominent, and always draws the olecranon upwards. The median and ulnar nerves may be stretched or contused. In some cases the coronoid process is fractured, and then the dislocation tends to reproduce itself. The head of the radius maintains its connection with the ulna. Dislocation backwards may be complete or incomplete.

Symptoms.—The fore-arm is fixed, slightly flexed, shortened; and any movement of it is exceedingly painful. The characteristic sign is the marked projection posteriorly of the olecranon and triceps tendon (Fig. 392). The head of the radius, too, may be felt projecting behind the external condyle. There is a marked broad prominence anteriorly, caused by the lower end of the humerus. The relations between the condyles and olecranon are altered, and the condyles do not move with the displaced olecranon. This test is of vital importance as distinguishing dislocation backwards from separation of the epiphysis in the young, or transverse fracture in the adult.

The hand and fore-arm are generally held midway between pronation and supination. On reduction, the parts regain their normal form with a "snap," and remain there, unless fracture of the coronoid co-exists. In a transverse fracture with displacement backwards crepitus is detected, and the deformity reproduces itself on relaxing extension. Measurement from the external condyle to the olecranon shows no alteration in transverse fracture of the lower end of the humerus. In dislocation the distance, as compared with the sound side, is altered. Separation of the lower epiphysis of the humerus in young children simulates this dislocation very closely, and the mobility or otherwise of the condyles must be most carefully ascertained by examination under an anæsthetic.

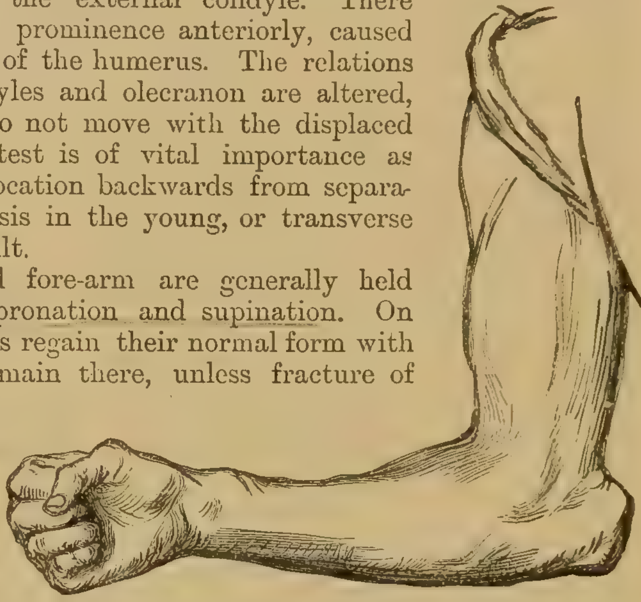

Fig. 392.—Appearance in Dislocation of the Elbow backwards.

2 and 3. **Lateral dislocations of the radius and ulna from the humerus.** *Causation and morbid anatomy.*—These accidents are produced by falls upon the outstretched hand, or upon the elbow; by blows, or twisting movements of machinery. They are practically always incomplete, and the dislocation outwards is by far the more common (Fig. 393). The sigmoid cavity of the ulna grasps the radial articulating surface of the external condyle, and the head of the radius is still more external, and quite clear of the humerus. In the incomplete dislocation inwards the sigmoid cavity impinges upon the internal condyle, above it or below it, while the

head of the pronated radius rolls in the coronoid fossa in front of the humerus. The lateral ligaments are much lacerated, but the orbicular ligament retains the connection between the radius and ulna, and the posterior ligament may remain unhurt. The ulnar nerve may be damaged in the inward displacement, and these accidents may be complicated with fracture, or separation of the epicondyle, a matter of importance to remember and ascertain. Dislocation backwards and outwards or inwards were also formerly described. Here the backward displacement of the olecranon is a more important symptom than the lateral deviation.

Symptoms.—In the dislocation outwards the fore-arm is flexed and pronated, and movements are restricted and painful. The limb appears widened opposite the joint. On the outer side will be felt the prominent displaced head of the radius. Internal to this is another projection; this is the displaced olecranon. Proceeding still more internally, we plainly feel the projecting internal condyle, and a gap beneath it, owing to the ulna having been thrown outwards.

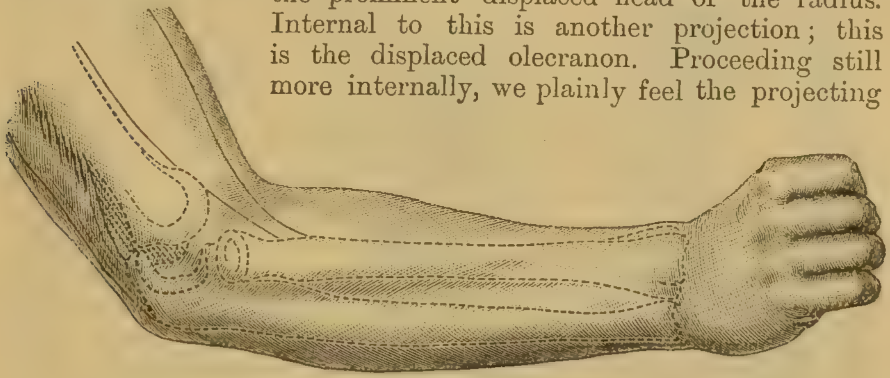

Fig. 393.—Partial Dislocation of the Elbow outwards. (After Tillmann.)

In the dislocation inwards the reverse occurs; the fore-arm is still flexed and held midway between pronation and supination, with the appearance of widening at the flexure of the joint; but there is a marked projection internally obscuring the internal condyle, and this, by manipulation, may be ascertained to be the upper end of the ulna. The external condyle is markedly prominent, and there is a gap below it, owing to the radius having been thrown inwards.

Method of examining for supposed dislocation of elbow.—The patient should have both upper limbs stripped, and should face the surgeon, placing the dorsum of his hands on the surgeon's shoulders. The surgeon, having carefully inspected the injured joint, places his thumb and little finger on the condyles and his middle finger on the olecranon of both elbow joints of the patient. Comparative alterations will be thus readily detected. He will decide—

(1) If the condyles move upon each other.

(2) If they have their normal relation to the olecranon by measurement.

(3) If crepitus exists; and if in any displacement the condyles move with the bones of the fore-arm.

(4) If the head of the radius rolls in its normal position to the external condyle.

These questions can usually only be satisfactorily answered with the aid of anæsthetics.

Treatment of dislocations of the elbow, backwards and laterally.—In recent cases of backward or lateral dislocation, extension of the fore-arm, with counter-extension of the humerus will, especially under anæsthesia, easily bring about reduction. In the lateral dislocations, the direction of the extension must be appropriately modified, and direct pressure made upon the projecting olecranon or radius.

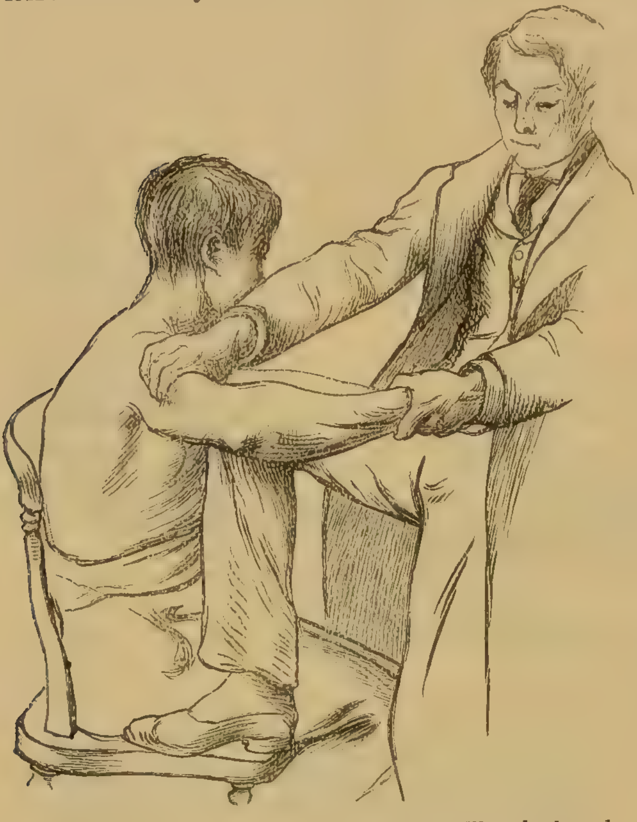

Fig. 394.—Reduction of Dislocation of the Elbow backwards.

Perhaps there is no better plan of reducing dislocations of the elbow backwards than by the knee method. The patient being seated, or reclining if anæsthetised, the surgeon places his foot on the edge of the chair, and presses with his knee against the humerus, drawing upon the wrist at the same time with one hand, so as to flex the joint round his knee, and pushing the humerus backwards with the other (Fig. 394).

If these dislocations have been overlooked for five weeks, reduction will probably be found impossible, the interlocked prominences of bone being soon welded firmly together by fibrous adhesions. Violent efforts, as with pulleys, are strongly to be condemned, and the too free "breaking-up" of adhesions may be followed by very serious inflammatory consequences, or associated with fracture of the bones. Subcutaneous division of the triceps and of adhesions has been had recourse to in these cases; but supposing the joint to be fixed in a faulty position, and the patient to be young, and a movable joint to be of vast importance to him, a well-performed excision of the elbow would probably give the best result, and be devoid of the risks of unscientific violence. The bones must be freely removed if mobility is to be arrived at, and the operation will be one of great difficulty.

4. **Dislocation of the radius and ulna forwards** (Fig. 395).—This exceptional injury is produced by direct violence to the back of the elbow when the fore-arm is acutely flexed, the bones of the fore-arm being thus driven forwards. In Canton's well-known case the radius maintained its relation with the ulna, and the olecranon lay in front of the humerus. The anterior, posterior, and lateral ligaments were torn, and the triceps muscle and ulnar nerve also. In this injury the fore-arm is lengthened and flexed. There is a projection posteriorly of the lower end of the humerus, and a depression in front of it, where the articular ends of the radius and ulna have passed forwards. Should the surgeon be called to treat this displacement, he would attempt to reduce it by flexing the joint acutely, and then thrusting the bones of the fore-arm backwards with one hand, while the humerus is drawn forwards by the other.

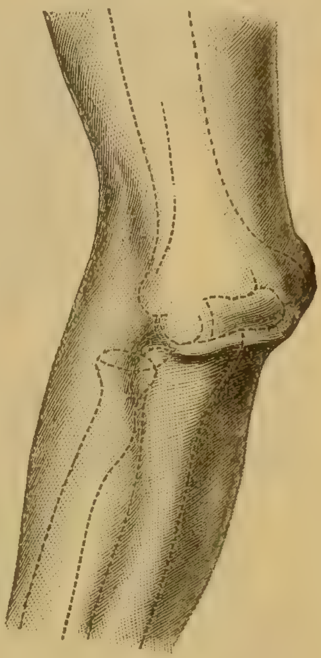

Fig. 395. — Dislocation of the Elbow forwards.

5. **Dislocation of the upper end of the ulna alone backwards.**—This is a rare accident. The orbicular ligament yields, and the radius maintains its position with regard to the external condyle, while the ulna is displaced backwards. The dislocation is generally not quite complete. The fore-arm is flexed at an obtuse angle, and held midway between pronation and supination, and the projection of the olecranon and triceps tendon can be made out posteriorly.

The treatment by the knee method or extension would here be applicable.

6. **Dislocation of the upper end of the radius alone** (Fig. 396). *Causation and classification.*—This accident is generally caused by falls on the hand, the direction of the force determining as to whether the head of the bone shall be thrown forwards or backwards. It may also be produced by blows on the head of the bone, and in children and those of weak muscular development and lax ligaments, by dragging upon the fore-arm. Dislocation *forwards* is said to be most common, yet differences of opinion on this point are evident. The head of the bone may also be thrown backwards, and more rarely outwards.

Displacement of the head of the radius downwards will receive separate mention.

Morbid anatomy.—In the dislocation forwards the head of the bone rests upon the front of the external condyle, or on the humerus. In the dislocation backwards the head of the radius impinges upon the back of the external condyle. The articular and external lateral ligaments are generally lacerated, and the oblique ligament may be torn in the displacement backwards (Fig. 397).

Symptoms of the dislocation forwards.—The fore-arm is pronated and slightly flexed: there is an undue fulness on the outer side of the fore-arm, which appears shortened. The head of the radius can be felt rolling in its new situation. Complete supination is usually impossible, from relaxation of the biceps; and on attempting flexion, the movement is suddenly arrested by the head of the displaced radius being brought up against the front of the humerus. The biceps tendon may be felt at the same time to be unduly relaxed. The external, anterior, and annular ligaments are torn.

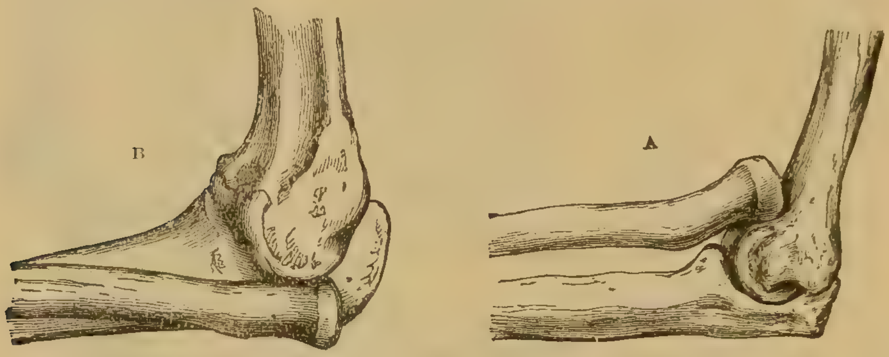

Fig. 396.—Dislocation of the Radius, (A) forwards and (B) backwards.

Treatment of the dislocation forwards.—The fore-arm should be flexed, and extension be made in this direction from the wrist, while the humerus is pressed backwards. The thumb of the hand, which presses back the lower end of the humerus, should also firmly force backwards the head of the radius by direct pressure. After reduction, the fore-arm should be kept flexed at an acute angle for about three weeks, to allow of the union of the ligaments and prevent the biceps tendon from pulling the head of the radius forwards. This is best effected by moulding a leather or gutta-percha splint to the back of the arm and fore-arm. Difficulty may be experienced in reducing this dislocation, from the interposition of the torn orbicular ligament between the head of the radius and the condyle.

Symptoms of the dislocation backwards.—Here the fore-arm is slightly flexed and held midway between supination and pronation, the action of the biceps being interfered with. The head of the bone can be felt rolling behind the external condyle. Extension is arrested by the radius being brought up against the back of the humerus.

Treatment of the dislocation backwards.—Difference of opinion exists regarding the manipulations necessary to reduce this injury. Some authorities advise extension and counter-extension, with direct pressure upon the head of the displaced bone, and this would seem to be generally efficacious. Afterwards, the limb should be put up in the flexed position, or it has been advised that the fore-arm be kept extended, to allow the tense biceps to pull the radius forwards. Should fracture of the external condyle complicate these injuries, the parts must be carefully padded, and great care and gentleness exerted in the earlier trials of passive movement.

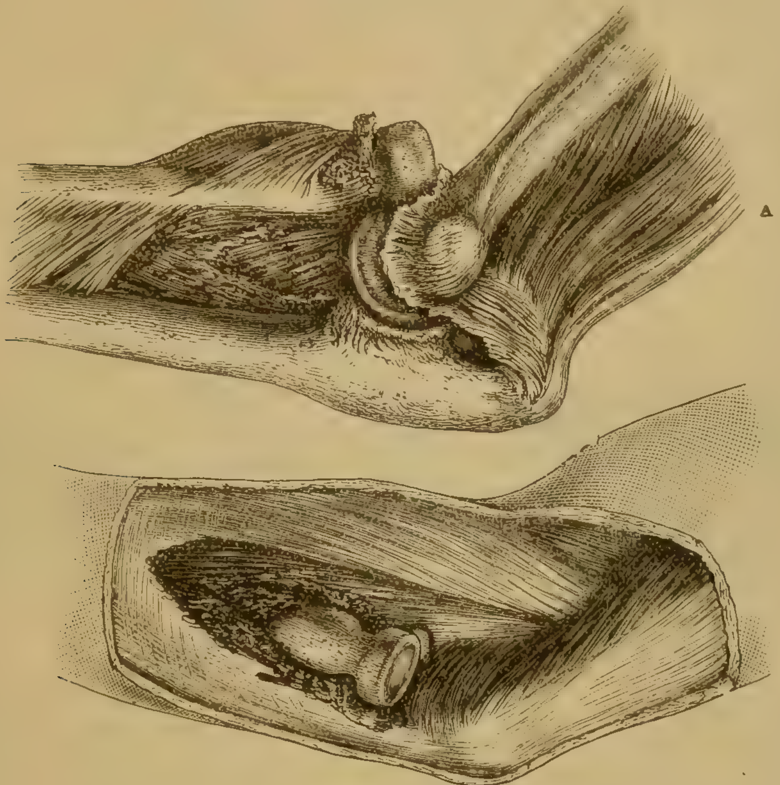

Fig. 397.—A, Dislocation of the Head of the Radius forwards; B, Dislocation of the Head of the Radius backwards.

Dislocation of the head of the radius outwards.—It is well to look upon these displacements as really modifications of the forwards or backwards dislocations. Thus the head of the radius may be thrown outwards and forwards, or outwards and backwards. The diagnosis and treatment require no special remarks.

Displacement of the head of the radius downwards, subluxation of the head of the radius.—This may be described as a peculiar condition of the elbow found in young children, produced, almost always, by lifting them up by the hand or arm, or dragging them along by the hand. Afterwards, it will be found that the child keeps its elbow slightly flexed and the fore-arm held midway between pronation and supination. The patient also complains of acute pain in the joint, and a slight projection is felt posteriorly in the situation of the head of the radius. If the hand be grasped, the elbow flexed, and the opposite movement made to the position the bones of the fore-arm have assumed, supination in pronation, pronation in supination, the parts will usually be restored to usefulness and movement. Anæsthetics are very useful in these cases, as movement may be unbearable. If a slight projection of the head of the radius can be made out, pressure by the thumbs should be made upon this at the same time.

Numerous pathological explanations have been given of this little accident. It has been attributed to a hitching of the bicipital tuberosity against the edge of the ulna, to the interposition of fibres of the supinator brevis, to separation of the epiphysis, while other authors have referred the mischief to the inferior radio-ulnar joint. The more probable explanation seems to be, that the laxity of the ligaments permits the head of the radius to be drawn downwards, and the orbicular ligament slipping upwards, a fold of it becomes interposed between the head of the radius and the condyle (Perigarde, J. Hutchinson, junior).

Compound dislocations of the elbow, laterally or backwards.—Amputation is rarely called for, unless the soft parts are exceedingly damaged and fractures co-exist. Should the wounds in the skin be clean cut, simple reduction should be effected, otherwise excision must be performed, and then the bone must be freely removed if the surgeon wishes to obtain a subsequently movable fore-arm. The prognosis of these injuries as regards danger to life is favourable, though fibrous anchylosis is apt to ensue.

Congenital dislocations of the elbow.—These are not unknown, especially in cases of "monstrosities." I have seen congenital displacement of the head of the radius in both limbs of the same child, on the one side forwards, on the other backwards.

Dislocation of the inferior radio-ulnar articulation.—Unless combined with fracture of the radius, this dislocation is so rare as hardly to merit notice. I have seen several cases, however, of pathological displacement from rheumatoid arthritis. The accident is caused by violent twisting movements of the hand. The internal lateral ligament is ruptured, the triangular inter-articular fibro-cartilage torn, and the head of the ulna being thrown backwards or forwards, makes an obvious prominence, posteriorly or anteriorly. Reduction seems to have been generally effected by extension and counter-extension. Well-padded splints or plaster of Paris dressing must be afterwards applied, or the displacement will return.

Dislocations of the wrist; of the carpus from the radius. *Causation and classification.*—Dislocation at the radio-carpal joint is generally caused by heavy falls on the palms of the hands. It seems a question whether a Colles' fracture should be produced or a dislocation, but the bone usually gives way. The bond between the radius and ulna, by the powerful triangular fibro-cartilage, is very strong. The numerous tendons which surround the wrist, as well as the ligaments, render the articulation very secure, and not likely to be displaced. Heavy falls on the back of the hand may drive the carpus forwards. The displacement backwards is the more common, the dislocation forwards exceptional, either displacement being rare. The writer has seen cases of pathological displacement and of congenital dislocation, and is inclined to think that accidents diagnosed as traumatic dislocations of the wrist, are really instances of fracture of the radius low down, or separation of the epiphysis. Both of these injuries closely simulate dislocation when the lower fragment is much displaced. Compound dislocations of the wrist from machinery accidents are not so uncommon.

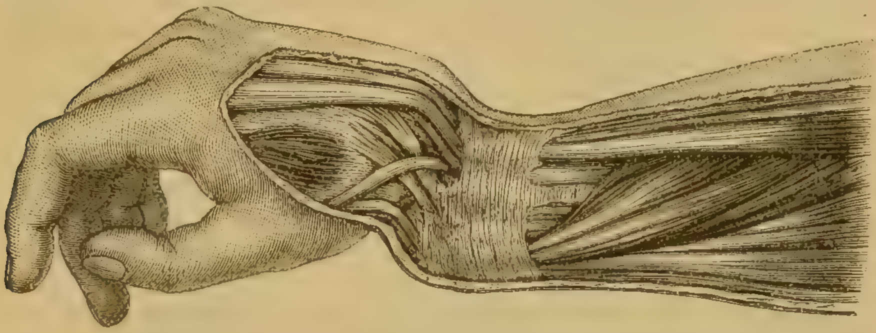

Fig. 398.—Dislocation of the Carpus backwards. (After Anger.)

Morbid anatomy.—The anterior, posterior, and lateral ligaments are more or less torn, the tendons displaced, and "ploughed up" from their grooves; the vessels and nerves may also be injured. Fractures of the lower ends of the radius and ulna may be discovered, and where the lesions of the soft parts are very extensive, the injury is most frequently compound.

Symptoms.—In dislocation of the carpus backwards, the deformity is much like that of Colles' fracture (Fig. 398). There is a dorsal prominence, and anteriorly on a lower level is another prominence with the flexor tendons stretched over it. The whole hand is thrown backwards, and the fingers are flexed. On close examination it will be found that the styloid process of the radius is in its normal position, and does not move with the dorsal protuberance on extension and rotation of the hand. The symptoms of the very rare forwards displacement are the reverse of the above. When there is not great swelling, the smooth, rounded contour formed by the upper row of the carpal bones can be plainly felt, and also the concave surfaces of the lower ends of the radius and ulna. When the injury is compound the diagnosis is easy.

Treatment.—Reduction is effected by extension and counter-extension, and the limb splinted carefully. The subsequent swelling is excessive, and great care must be taken that the splints are not unduly tight. In fourteen days the plaster of Paris dressing can be applied, and the fingers moved daily. Movements of the wrist may be commenced in about five weeks. In compound dislocations resection of the protruding bones may be needful, but amputation should never be performed unless the bones and soft parts are most extensively crushed.

Dislocations of the carpal bones from each other.—Although the carpal bones are so strongly articulated, some of them have been found from time to time separately displaced, and usually in the dorsal direction. Hamilton has found no single dislocation of the carpal bones, except in the case of the os magnum, pisiform and semi-lunar. These injuries may be compound, as seen in machinery accidents and gunshot wounds. The diagnosis is to be made by observing an abnormal dorsal projection in the situation of one of the bones, and care must be taken not to confound this with a thick-walled, deeply-seated ganglion. Reduction may be effected by direct pressure, and in compound injuries removal of the displaced bones is usually the wisest course to pursue.

Dislocations of the metacarpal bones from the carpus.—The metacarpal bone of the thumb may be thrown backwards or forwards from the trapezium, and more rarely one or more of the metacarpal bones may be displaced backwards or forwards, completely or incompletely. Such accidents are the result of falls, blows, twists, and especially machinery injuries. These dislocations are apt to be overlooked, especially where swelling has supervened. They are to be diagnosed by the undue prominence, posteriorly or anteriorly, of the base of the metacarpal bone, and in the case of the thumb by the fixity and loss of mobility of the metacarpal bone. When seen early they can be reduced by direct extension. When compound, the ends of the bones may be resected.

Dislocation of the phalanges from the metacarpal bones, and from each other.—Any of the phalanges may be displaced from the metacarpal bones, but the injury is most common and important in the thumb, index and little fingers. These dislocations are usually backwards, the phalanx being thrown upon the dorsum of the metacarpal bone. Dorsal, palmar, or lateral dislocations of the second or last phalanges are also found. The last phalanx is most commonly implicated, especially the last phalanx of the thumb, and the principal cause of this accident is a fall or blow on the end of the digit, such as is inflicted by a cricket-ball. The diagnosis of these injuries is obvious, and they are to be treated by direct extension under anæsthetics. If inseparable difficulties be found in their reduction, an antiseptic incision should be made, resisting structures divided, and the bones replaced. If compound, the articular ends may need resection. Dislocation of the first phalanx from the metacarpal bone of the thumb requires special mention, on account of the difficulty described as met with in reduction. I have found as much difficulty in reduction of dislocations of the first phalanx in some of the other digits, as in the case of the thumb.

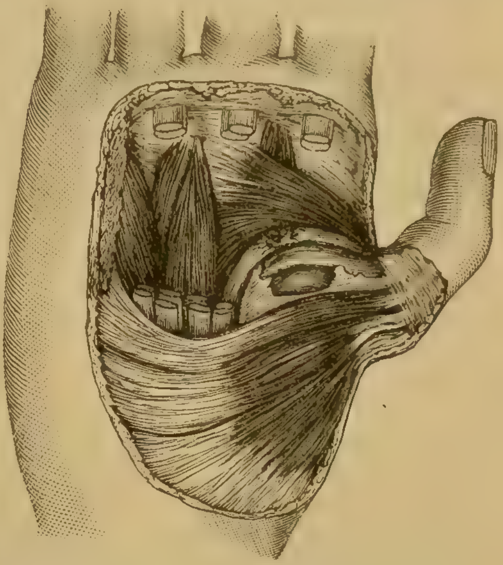

Fig. 399.—Dislocation of the first Phalanx of the Thumb backwards. (After Anger.)

Dislocations of the metacarpo - phalangeal joint of the thumb.—In the large majority of cases, the phalanx is displaced backwards, and the head of the metacarpal bone bursts through the glenoid ligament, between the two heads of the flexor brevis pollicis, containing the sesamoid bones which embrace its neck (Figs. 399, 400). The prominence of the head of the metacarpal bone may be felt anteriorly, and the base of the displaced phalanx dorsally. The phalanx is flexed dorsally, forming an obtuse angle with the metacarpal bone The obstacles to reduction are said to be the constriction of the neck of the metacarpal bone by the short flexor tendons; but I am inclined to believe that torn and twisted portions of the glenoid ligament intervening, and being wrapped round the neck of the bone, are potent causes of the undoubted difficulty found in the treatment of this displacement.

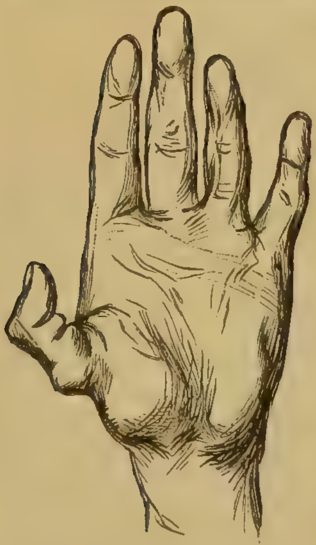

Fig. 400.—Appearance in Dorsal Dislocation of the first Phalanx of the Thumb.

To relax the ligaments and the short flexor tendons as much as possible, the metacarpal bone should be strongly adducted, and the base of the phalanx pushed forwards with the thumb of the hand that grasps the metacarpal region. The displaced phalanx should be strongly drawn at first in the direction of the displacement—*i.e.* in dorsal flexion—and as the surfaces disengage, it should be brought towards the palm, still keeping up extension. By adduction of the metacarpal bone, and traction in dorsal flexion the opening between the heads of the short flexor and in the aponeurotic structures gapes, and on extending, the metacarpal bone slips backwards and the base of the phalanx forwards, thus effecting reduction. The common error made in this manipulation is to exercise direct extension, before adducting and making traction on the phalanx in dorsal flexion. Should reduction prove impossible, open arthrotomy should be at once performed by a free incision on the palmar aspect. Resisting structures, as portions of ligaments or the short flexor tendons, may be divided or pulled aside with a fine hook, and thus reduction be effected. In old cases the base of the phalanx may be excised. I would advise this treatment in preference to subcutaneous tenotomy; for I have seen failure to reduce this dislocation on more than one occasion, even when the operator had divided the structures round the joint with the tenotome so freely and repeatedly that there appeared little left. Should much traction be required in dislocations of the phalanges, the "American forceps" (Fig. 401), the "Indian puzzle," or a clove hitch made with a soft bandage, are useful adjuncts. The "puzzle" is an elongated cone terminating in a cord, and hollow in the centre. "When applied to the finger," says Hamilton, "it is slipped on tightly, forming a cap to the extremity, and to half the length of the finger; but on traction being made from the opposite end, it fastens

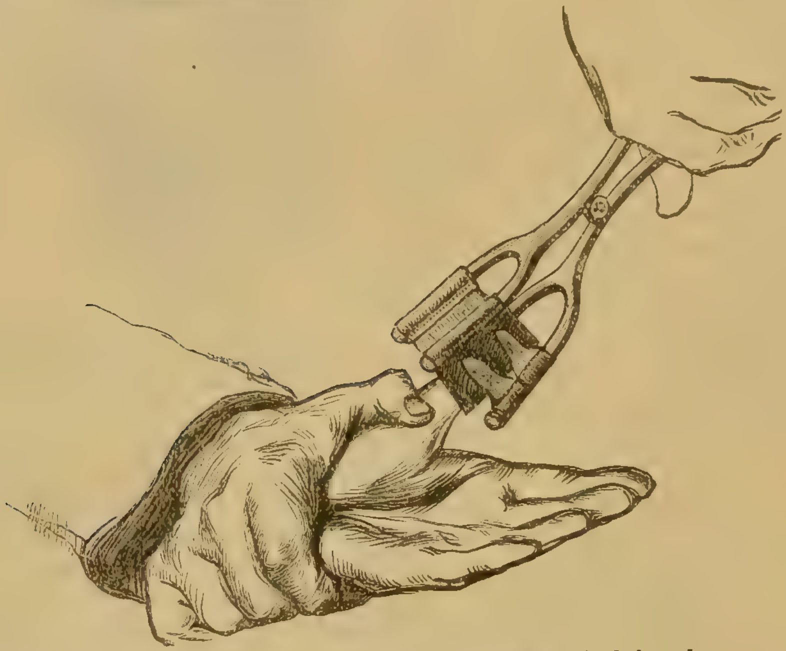

Fig. 401.—Dislocation of the Thumb. Traction on the displaced phalanx by means of the American forceps.

itself to the limb with uncompromising grasp." The advantage of this apparatus is the equable pressure it exerts, and it readily drops off on releasing extension.

Dislocations of the bones of the pelvis. *General remarks.*—In consequence of the great strength and breadth of the pelvic articulations, displacements are rare. Many cases cited as dislocations are really fractures, portions of bone being torn away by the powerful ligaments. These accidents are produced by violent crushes. Dislocations at the pubic symphysis have been caused by forcible separation of the thighs, and also by prolonged and difficult labours, especially in contracted pelvis. The bone is generally torn away from the inter-articular cartilage. All these injuries may be complicated by lacerations of the urethra or pelvic viscera, or great veins. The treatment is practically that of fractured pelvis. The parts should be manipulated into position by extension and pressure, and a broad pelvic belt should be worn, with rest upon a hard, flat mattress for six weeks to two months. Dislocations, usually partial, of the coccyx from the sacrum are found; but this injury will receive notice in the article on INJURIES OF THE SPINE (Art. XXXVIII., Vol. II.).

Dislocations of the hip. **Causation and classification.**—Dislocations of the hip are usually produced by great violence, and are more common in males during active adult life, between thirty and sixty. The accident may occur, however, in infancy or extreme old age. On account of the depth of the acetabulum, the strength of the ligaments, and the protected position and deep situation of the articulation, clothed round with muscles, dislocations of the hip are far more rare than those of the shoulder. In exceptional instances these displacements are produced by slight causes in those who have lax ligaments. Some contortionists possess the power of dislocating the joint at will. Congenital dislocations of the hip-joint are comparatively common, and so are displacements from softening of the capsule and ligaments in inflammatory or pyæmic affections. The displacements of the hip in such conditions as acute ataxia and rheumatoid arthritis are associated with considerable osseous changes, and should hardly be classified as true dislocations. The literature of dislocations of the hip shows that a considerable number of traumatic displacements may occur. There is good reason to say with Bryant that the head of the femur may be displaced anywhere in the immediate vicinity of the acetabulum. I may also point out that the greatest variety must occur in the lesion or extent of lesion found in the muscles, tendons, and ligamentous structures in these cases. The directions and nature of the great forces producing dislocation may also modify the described portions of the limb; and so especially may concomitant fractures of the pelvis. Such considerations may well account for some of the discrepancies found in the description of these accidents by authors, and of the appropriate methods of treating them by manipulation. Every surgeon of experience has met with cases of dislocation of the hip which differed notably in one or more symptoms from the ordinarily received descriptions; and the reader would do well to look upon the explanations of the four more common dislocations of the hip as being typical, and guides to the subject in that sense only. The belief that certain positions and symptoms must inevitably occur in these accidents, may lead to woeful mistakes in practice.

Arranged in order of frequency, the dislocations of the hip may be thus classified: (1) Dislocation backwards and upwards on to the dorsum ilii; (2) Dislocation backwards into the vicinity of the sciatic notch; (3) Dislocation downwards and forwards into the obturator foramen; (4) Dislocation forwards and inwards on to the pubes.

Mechanism of dislocations of the hip.—The capsule of the hip-joint is strongest anteriorly and superiorly. Anteriorly a fan-shaped band of dense and extremely strong fibres passes from the anterior inferior iliac spine to the front of the lesser trochanter and the anterior inter-trochanteric line. This part of the capsule has received the name of the Y-ligament (Bigelow). All the capsule connected with the front of the femur is exceedingly strong, while that connected with its back is weak. The capsule attached to the lower and inner aspect of the acetabulum is thin and weak. The strongest and deepest part of the acetabulum is above and posteriorly, just where the capsule is strongest. The shallowest and weakest part of the acetabulum is below, near the transverse notch, where the capsule is also thin and weak.

If the femur be adducted, the head of the bone passes into the deepest and most secure part of the cavity. If the femur be abducted, and especially if it be rotated inwards at the same time, it presses upon the weakest part of the capsule, and tends to slip over the incomplete rim of the here shallow acetabulum. The round ligament, which varies much in consistence and strength in different individuals, in excessive adduction is tense, in abduction slack, and any sudden violence may rupture it or tear it away from its connections. From anatomical considerations, therefore, we shall expect the head of the bone primarily to leave the capsule *inferiorly* in all dislocations of the hip.

The examination of museum specimens, and the evidence of dissections, support the above anatomical considerations. The rent in the capsule being, in the vast majority of cases, extensive, and starting inferiorly near the transverse notch of the acetabulum, spreads towards the back of the neck of the femur.

Experiments on the cadaver show that in forcible abduction the head of the bone passes out inferiorly, and is lodged in the obturator foramen, which may be looked upon as the first position of the femoral head in dislocations of the hip. Experiments upon the cadaver must, however, be accepted with caution, since no one can tell the various forces that may be at work in any given case of accident; and the secondary contractions of the muscles, so important in dislocations, is of course absent. The head of the bone having ruptured the capsule inferiorly, and passed into the obturator foramen, what is to determine its further course? The answer to this question will be found in the position of the limb, the direction and continuation of the force, aided by the contraction of the muscles. If the abducted femur be flexed and rotated inwards, the head of the bone, which it may be remembered is always opposite the internal condyle, will pass from the obturator foramen backwards, and if the force be continued in flexion of the thigh, will also pass upwards. Thus are produced the dislocations into the vicinity of the sciatic notch, and the dislocation on to the dorsum ilii in more advanced forms. The more the thigh is flexed and rotated inwards, the more will the head of the bone pass directly backwards, and if the flexion be acute enough, and the force continue, there is no reason why the head of the bone should not pass backwards and actually downwards towards the tuber ischii. In moderate flexion of the thigh with inward rotation, the head of the femur will plough its way upwards and backwards from the obturator foramen to the dorsum ilii. According to this view, all backward dislocations of the hip are primarily below the tendon of the obturator internus, and there is no need of the classification of Bigelow, of dislocations above and below the tendon, since the dislocation above it cannot primarily occur.

That this is the correct explanation of the mechanism of dislocations of the hip, as argued by Fabbri, Tillaux, Coote, and more recently with undeniable force and clearness by Morris, it is idle to deny. At the same time, it is possible that in exceptional circumstances the head of the bone may be thrust through the posterior part of the capsule, beneath the strong superior fibres by direct violence. In such cases the lip of the acetabulum is usually broken, a portion of bone being carried away with the capsule, attached to the neck of the femur. MacCormac, however, cites one case of direct backward dislocation without fracture, and the clinical evidence of this accident is sometimes very suggestive. I have seen the cases of two men, sitting in a railway carriage, with the right leg crossed over the left, in the position, therefore, of flexion and adduction. The carriage was smashed in, and both of them sustained dorsal dislocations of the femur, with a large hæmatoma on the front of the knee, marking the application of the force. In such cases, the inference that the head of the femur is thrust out through the posterior part of the capsule, is strong. There was no fracture in either of these cases, which were treated by the ordinary methods of manipulation with success. The advocates of the first theory would here object, that though the thigh was adducted, the pelvis might have been violently abducted during the crushing force, and so the head of the bone would really burst through the capsule inferiorly.

In the present state of our knowledge it is safe to assume that in the vast majority of cases, dislocations of the hip are primarily downwards. Direct backward dislocations are generally associated with fracture of the rim of the acetabulum; and that this displacement can take place without fracture is only maintained by scanty pathological proof, and by clinical observation, which, without dissection, may prove erroneous. Primary direct dorsal dislocation above the tendon of the obturator internus cannot occur without fracture, since here the head of the bone encounters the deep rim of the acetabulum, and part of the strongest portion of the capsule.

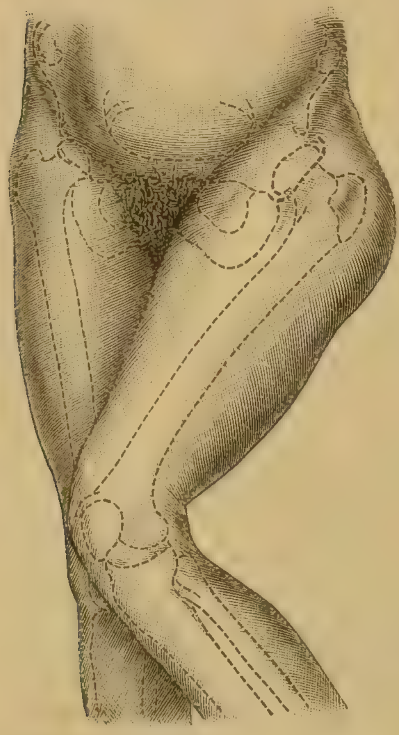

Fig. 402.—Dislocation of the Femur backwards.

Obstacles to reduction, especially in backward dislocations.—The upper and anterior part of the capsule being of extreme power, is rendered tense on extension, but is relaxed on flexion. The tension of the untorn part of the capsule is one of the great obstacles in drawing the head of the femur back to the acetabulum. A flap of torn capsule frequently becomes interposed between the acetabulum and the head of the femur. The head of the bone being thrust through or among the short rotator muscles, is often tightly girt by them and by tendinous fibres. The tendon of the obturator externus, if not ruptured, may bind down the neck of the femur like a cord, and the great sciatic nerve also may be entangled. All such structures as these are tightened in direct extension, and great force is needful to "overcome their resistance," or, in other words, to tear, and pull the head of the bone from amongst them. Hence the importance of free flexion to relax such important resistant structures as those enumerated.

Condition of the soft parts in backward dislocations of the hip.—It has already been pointed out that the rent of the capsule is inferiorly near the transverse notch. The tear is usually an extensive one, and extends towards the posterior surface of the femoral neck, the capsule being torn from the femur posteriorly, and not from the acetabulum, where it is comparatively strong. The short rotator muscles, the obturators, quadratus, the gemelli, and pyriformis are torn or violently stretched. The tendon of the obturator internus usually remains intact, but in marked cases all these muscles may be completely lacerated. The pectineus is generally torn, the psoas and iliacus are greatly stretched. The great sciatic nerve may be contused or even lacerated, and is frequently pressed upon by the head of the bone, or looped round its neck. The head of the bone ploughs its way beneath the glutæi, but it may lacerate them extensively. The greater the violence, the farther the head of the bone ascends on the dorsum, the more extensive will all these lesions be. The ligamentum teres generally tears a scale of bone away from the femur, or may itself be torn.

1. **Dislocation upwards and backwards on to the dorsum ilii** (Figs. 402, 403). *Causation.*—This, the most common dislocation of the hip, is produced by such accidents as heavy falls during abduction of the thighs, and especially when masses of metal or earth of great weight fall on the back when the thighs are abducted and flexed. This accident may also occur from the violent wrenches and twists of great machinery or railway accidents.

Symptoms.—The limb is shortened to an extent corresponding to the height the head of the bone has ascended. This is at least an inch and a half or two inches. The limb is flexed, adducted, and rotated inwards, so that the knee points across the lower third of the opposite thigh, and the ball of the great toe on the dorsum of the sound foot. There is a marked appearance of widening of the buttock, the trochanter being turned forwards, and approximated to the anterior superior spine above the line of Nélaton. The ilio-tibial fascia is relaxed, because of the approximation of the trochanter to the anterior superior spine. On feeling deeply in the groin, the femoral vessels, instead of beating in evident relief, will sink backwards into a hollow, and in thin subjects the rim of the acetabulum may even be felt. This is a symptom of great value. The fold of the buttock, on posterior inspection, is seen to be raised. There is considerable fixation of the limb, which can only be slightly flexed, adducted, and rotated inwards. Moderate traction fails to draw the limb down to its proper length, a valuable differential diagnostic sign from fracture. Under anæsthesia, the head of the bone may be felt in the buttock.

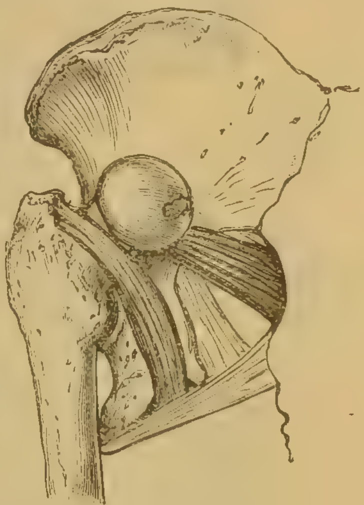

Fig. 403.—Dorsal Dislocation of the Hip. (After Pick.)

Treatment.—This should be undertaken as soon as possible. The patient, being laid perfectly flat on a hard mattress, is deeply anæsthetised with ether. The surgeon flexes the leg on the thigh to relax the hamstrings and great sciatic nerve. Continuing flexion, he bends the thigh on the pelvis until resistance is encountered. As the thigh is bent up it is carried towards the pubes in slight adduction. This manipulation relaxes the untorn part of the capsule or ilio-femoral ligament, the tension of which produces inversion. The thigh is now abducted to bring the head of the bone forwards, and to enlarge the rent in the capsule. It is next well rotated outwards ; this relaxes any of the tense untorn rotators, and slips the head of the bone into the acetabulum, the limb being finally brought down in extension. To sum up these movements, which should be executed powerfully in a continuous sweeping manner, flex in adduction, abduct, rotate outwards, extend (Fig. 404).

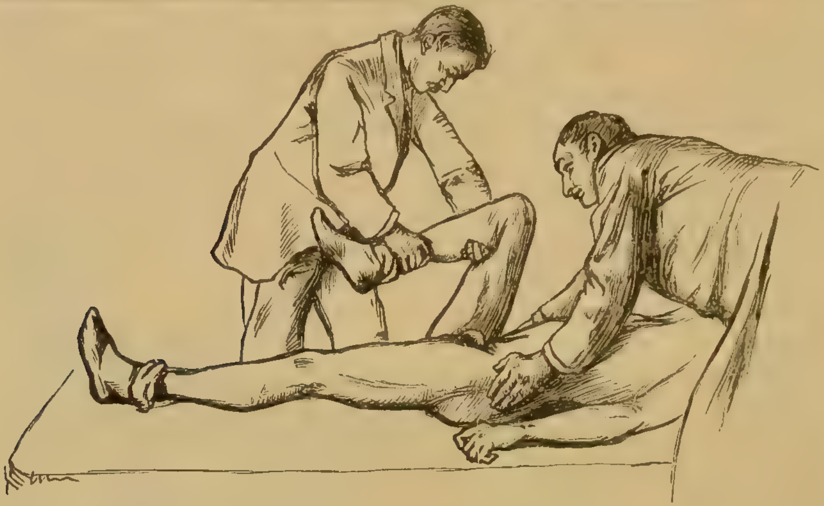

Fig. 404.—Flexion, Abduction and Rotation outwards, in Dorsal Dislocation of the Hip.

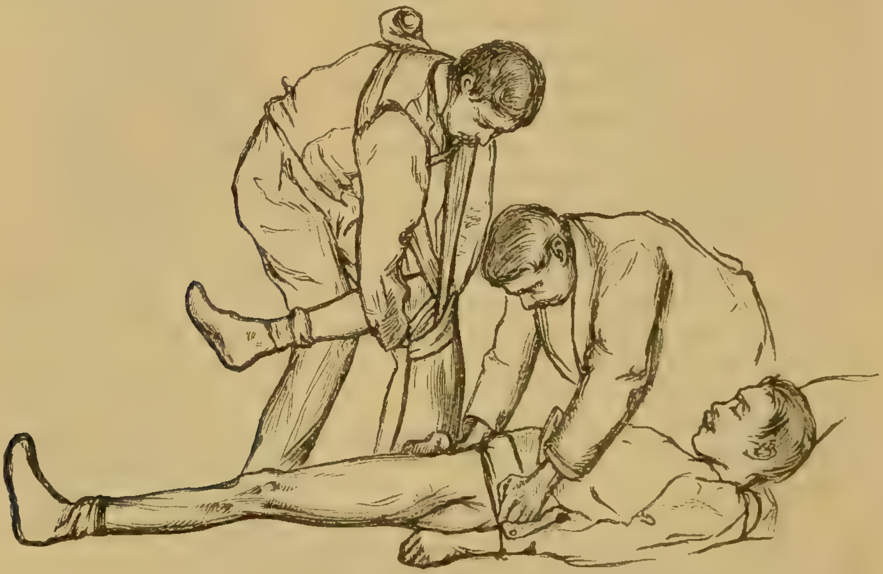

Fig. 405.—Forcibly lifting the Flexed Femur in Dorsal Dislocation.

Lifting movements are very useful in reducing backward dislocations of the hip, and these may well be combined with abduction and rotation outwards or inwards. The pelvis should be firmly fixed in all such manipulations by the hands of assistants, or a broad padded belt fastened to staples, or strongly strapped round the bed (Fig. 405). Provided the thigh be well flexed, considerable force may be exerted upon it by traction to lift the head of the bone into the acetabulum. Should manipulations fail, they may again be repeated on another occasion. There is seldom any difficulty in reducing a recent dislocation by manipulation if deep ether anæsthesia be induced.

Old dislocations of the femur backwards.—When the displacement has been left unreduced some weeks or months, great difficulties may be experienced, and I would not attempt to lay down

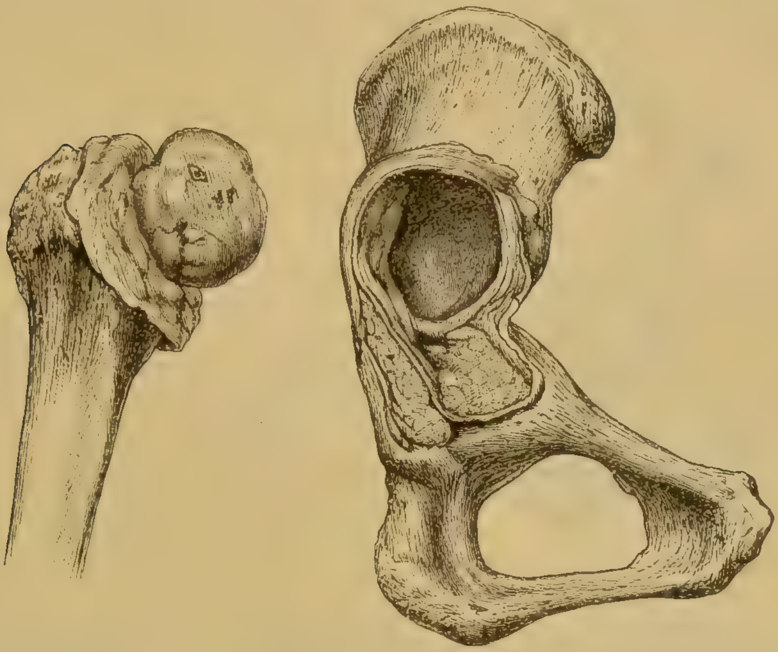

Fig. 406.—Unreduced Backward Dislocation of the Hip. The acetabulum is filled up and a new cavity is formed on the dorsum ilii. The head of the femur is altered in shape.

any time as to when it is right to attempt reduction. It may be pointed out that (1) manipulation is at least as likely to succeed as the use of pulleys or violent extension. (2) That the age, vigour, and occupation of the patient must be considered. (3) That manipulations, though they may fail in reduction, improve the case by breaking down adhesions. (4) That violent manipulations may cause such accidents as injuries to the sciatic nerve or vessels, extensive extravasation of blood and diffuse abscess, or fracture of the neck of the femur, the limb becoming mobile and everted with obvious crepitus. This latter accident I have myself witnessed. It would seem safer in these cases to attempt reduction by freely exposing the parts by a long incision posteriorly, and after dividing resistant structures to replace the head of the femur. Excision of the displaced head is also easily and certainly performed, and promises to be very successful. The greater number of old dislocations of the hip may well be left alone; and as the head of the bone works for itself a new socket, the tissues condense about it, and the lacerated muscles heal and recover contractility. A very useful limb will often ultimately result, the shortening being remedied by an appropriate boot. Muscle at first intervenes between the osseous surfaces, and a regular new bony socket seldom forms so quickly as is the case in the shoulder (Fig. 406).

The above remarks apply to any variety of ancient hip dislocation. If anchylosis has occurred in a bad position, Adam's operation of dividing the neck of the bone with a fine saw would probably give good results.

2. **Dislocation backwards into the vicinity of the sciatic notch** (Figs. 407, 408).—Much the same accidents that produce a dorsal dislocation will produce a dislocation near the sciatic notch, if the thigh be much flexed and rotated inwards. The amount of violence is generally less. The head of the bone does not pass so far upwards, lying beneath the obturator internus tendon, and usually upon the spine of the ischium.

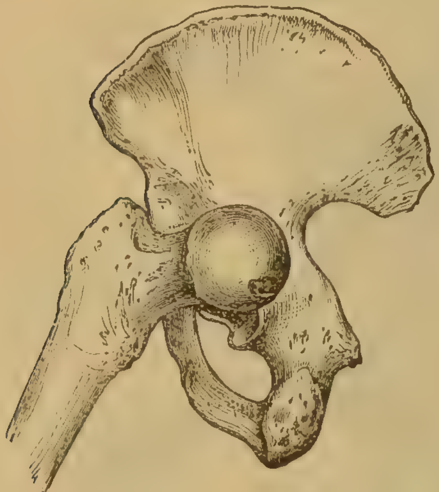
Fig. 407.—Sciatic Dislocation of the Hip. (After Pick.)

Symptoms.—These resemble the dorsal dislocation. As the head of the bone ascends but little, the shortening is less—usually under an inch. The thigh is flexed, adducted, and rotated inwards. The knee pointing to the knee of the sound side, and the ball of the toe towards the dorsum of the toe of the uninjured limb. The trochanter is a little raised, and the fold of the buttock also. The undue hollow of the groin from the sinking back of the femoral vessels may be detected. The slight shortening and the direction of the axis of the injured limb across the knee of the same thigh will differentiate this dislocation from the displacement on to the dorsum ilii. There is sometimes considerable flexion of the thigh in this accident, a symptom which is especially marked when the patient is lying upon his back. The shortening will then appear to be great; but on measurement, this is negatived. The head of the bone may be felt on deep manipulation, from the buttock, rectum, or vagina. This dislocation is often difficult to diagnose when there is much swelling, or in corpulent persons whose hip muscles and glutei are bulky and loaded with fat. It is not infrequently overlooked, and I have seen considerable mobility in sciatic dislocation, so that a superficial observer might easily have declared that no displacement could have existed.

Treatment.—A sciatic dislocation is to be treated in the same manner as a dorsal displacement. Lifting the head of the bone with abduction and external rotation is very useful. Too forcible abduction will land the head of the bone in the thyroid foramen.

Backward dislocations complicated by fracture.—In extreme cases of this nature the pelvis may be splintered into fragments. The most interesting complication is direct dorsal dislocation with fracture of the rim of the acetabulum, or, as related by Birkett, the head of the femur may be fractured as well, a portion remaining in the acetabulum. This accident may be suspected by the detection of crepitus, and a tendency to reproduction of the dislocation, which persistently occurs unless a weight be affixed to the foot and leg. Fracture of the shaft of the femur with dislocation is very rare. If the case be seen early, under deep anæsthesia, manipulation of the upper part of the shaft, with pressure on the displaced head, may perhaps bring about reduction. I see no reason why the method of McBurney, as advised in similar injuries of the shoulder, should not here be applicable. Extension of the thigh is, of course, useless; and if the shaft of the femur is allowed to unite firmly, it will probably be then too late to reduce the dislocation.

Especial difficulties in diagnosis of backward dislocations. — Traumatic backward dislocations of the hip have to be diagnosed from (1) impacted inverted fracture; (2) separation of the upper femoral epiphysis; (3) old hip disease; (4) rheumatoid arthritis with deformity; (5) congenital displacement.

In impacted inverted fracture, examined under anæsthesia, there is free movement. The femoral vessels have not receded into a hollow. The ilio-tibial band is markedly slackened in fractures of the neck of the femur. Crepitus from some giving way of the impaction will probably occur during manipulation, and the limb will become everted. Should the great trochanter be fractured and drawn away, it may be mistaken for the head of the femur on the buttock; but crepitus can usually be detected.

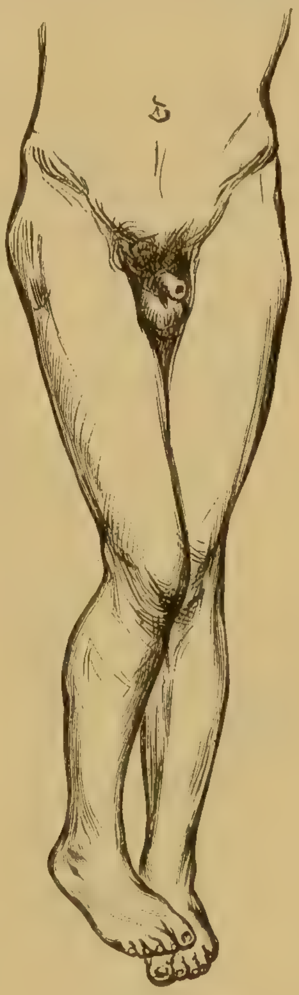
Fig. 408.—Sciatic Dislocation of the Hip.

Separation of the upper epiphysis may be suspected in a person under the age of eighteen years, and the younger the patient the more probable is the occurrence of what is, after all, an exceptional accident. Eversion of the foot is observed in these accidents, with, of course, free mobility under anæsthesia, and any shortening can be removed by drawing down the limb. On relaxing traction, the shortening and deformity are reproduced. The line of separation lies wholly within the capsule.

Old hip disease, or congenital displacement, can hardly be mistaken for traumatic hip dislocation. Errors in these directions usually occur when a person, the subject, let us say, of an old displacement of the hip from pyæmic inflammation, gets a heavy fall on the part, and is afterwards incoherent from alcoholism or concomitant head injury. It may then be hastily assumed that the displacement is recent. A warning of this error is sufficient to guard the reader against committing it.

In rheumatoid arthritis, the history of long-continued pain and lameness and the peculiar crepitus will serve as useful guides. Such cases are far more likely to be mistaken for fractures than dislocations.

Anomalous backward dislocations.—The head of the femur may lie just below the anterior superior spine and above the anterior inferior spine, the trochanter being turned backwards and sunk into a hollow. This is termed the supra-spinous dislocation. The head of the bone has also been found just above the acetabulum, and has passed backwards and downwards to the tuber ischii. In all these cases the head of the bone can be felt by manipulation under anæsthetics. There is considerable shortening, and in the dislocation downwards and backwards, much flexion of the thigh across the middle of the thigh of the sound side. In the supra-spinous, and even in some apparently ordinary dorsal dislocations, eversion sometimes occurs, and the case thus much resembles a fracture. Eversion has been attributed to a continuation of the force violently rotating the limb outwards after the head of the bone has reached the dorsum and rupturing some of the fibres of the ilio-femoral ligament, upon the tension of which inversion depends. These cases are to be diagnosed from fracture by the absence of crepitus, the sunken position of the trochanter, the impossibility of bringing the limb to its proper length by traction, and the detection of the head of the femur in an abnormal position. In the treatment, the head of the

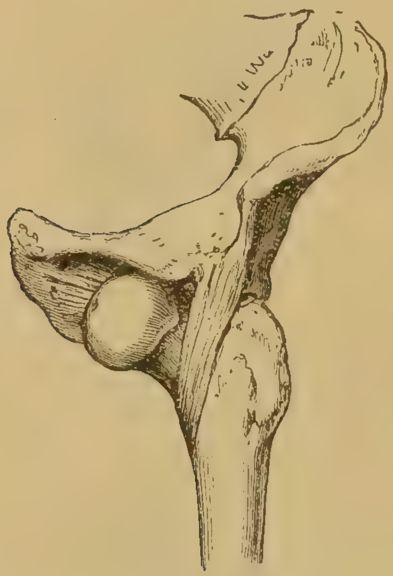

Fig. 409.—Obturator Dislocation of the Hip. (After Pick.)

bone should be brought as close to the acetabulum as possible by flexion and traction, and then the ordinary manipulation methods should be applied.

3. **Dislocation downwards and forwards into the obturator foramen** (Figs. 409 and 410). *Condition of soft parts.*—The capsule is torn inferiorly, and the rent extends forwards and upwards, instead of backwards and upwards, as in the case of the posterior dislocations. The ligamentum teres is torn away from the femur. The adductors, pectineus, and gracilis from the abduction of the femur are excessively tense and often lacerated. The ilio-psoas tendon is exceedingly tense; the glutei and obturator internus, with the gemelli and pyriformis, are much stretched. The head of the bone lies on the obturator externus muscle, pressing upon, tearing or stretching the obturator nerve.

Causation.—This is essentially the accident of extreme abduction. As has been explained, the first position of the head of the bone is downwards. If it move a little forwards, a thyroid dislocation occurs. This displacement can be readily manufactured in the cadaver by violent abduction. In practice it will be found to be caused by such accidents as falls with the legs widely apart, or stepping with one foot on to a boat which suddenly moves away from a pier. So, heavy weights falling upon the back, when the limbs are widely abducted, may produce an obturator dislocation.

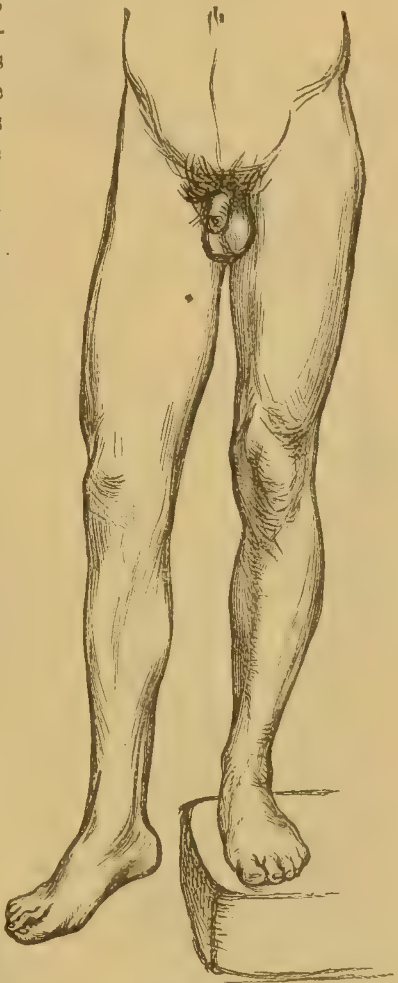

Fig. 410.—Obturator Dislocation of the Hip.

Symptoms.—The thigh is a little flexed, abducted, with the foot pointed forwards or everted (Fig. 410). There is lengthening, but this is seen to be apparent only from the tilting of the pelvis. In some cases, true lengthening is, however, found. Two marked signs are the tension of the adductor longus, which stands out like a tight cord, and the deep hollow left externally by the sinking inwards of the trochanter. The fold of the buttock is lowered, and much pain is complained of in the course of the obturator nerve. The head of the bone may or may not be detected, according to the extent it is thrown inwards.

Treatment.— Flex the leg on the thigh, and the thigh on the pelvis in slight abduction; adduct the thigh until opposite the umbilicus.

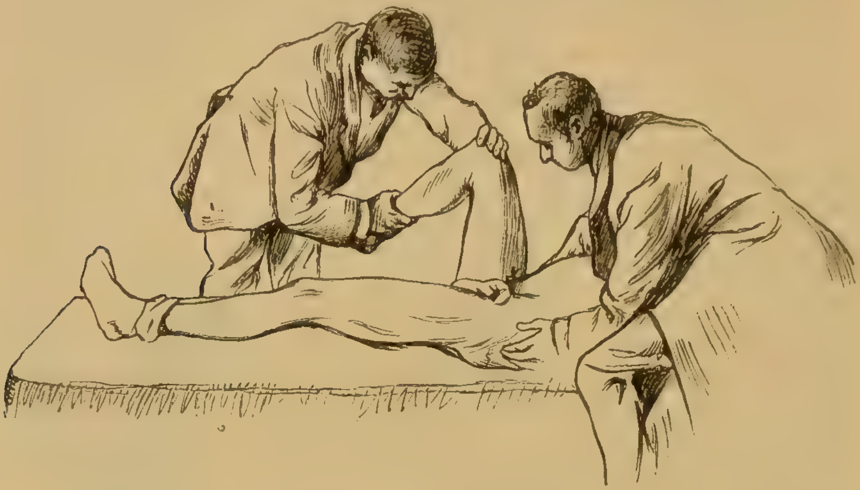

Fig. 411.—Flexion, Abduction and Rotation inwards in Pubic or Obturator Dislocations.

Rotate inwards and extend (Fig. 411). By this means the head of the bone retraces its steps into the acetabulum. In some cases of this manipulation, too forcible flexion of the thigh may press the displaced head backwards towards the sciatic region. Abduction and rotation outwards may then succeed, when the previous manipulation has failed. So, too, flexion of the thigh and lifting the head of the femur outwards and upwards into the acetabulum, combined with rotation inwards and outwards, have proved successful. While the surgeon is adducting the flexed limb, a powerful assistant may pass a jack-towel round the thigh, and the pelvis being firmly fixed, he, passing his shoulders through the towel, heaves upwards and outwards, while the surgeon adducts the limb and rotates it inwards. This dislocation has also been reduced by

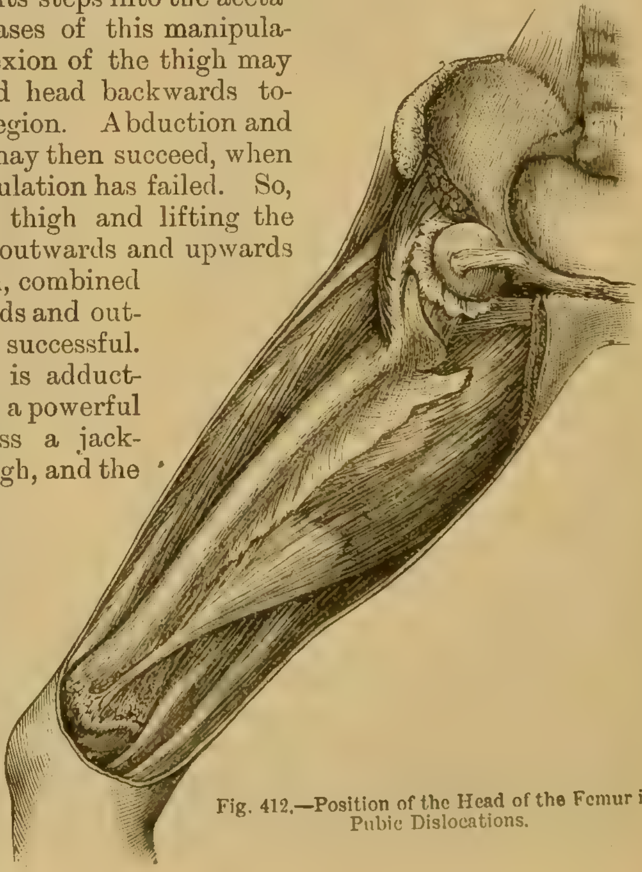

Fig. 412.—Position of the Head of the Femur in Pubic Dislocations.

placing a heavy sand-bag, or the well-padded bed-post, between the thighs and adducting the limb upon these as a fulcrum.

4. **Dislocation forwards and inwards on to the pubes, dislocation under the crural arch** (Fig. 412). *Condition of soft parts.*—The pubic dislocation may be looked upon as a further degree of obturator displacement. If violent extension and rotation outwards occurs, the head of the femur will mount upwards and forwards beneath Poupart's ligament. The soft parts are torn and lacerated to much the same extent as in the obturator dislocation. The capsule is torn inferiorly, but, according to Gunn, anteriorly also, to a marked extent. The head of the bone lies usually nearer the ilium than the pubes, to the outer side of the femoral vessels; the crural nerves are, therefore, often stretched considerably. The head of the femur may pass under Poupart's ligament for a considerable distance upwards into the pelvis. The tension of the ilio-psoas tendon and the untorn ilio-femoral ligament produces eversion.

Symptoms.—There is true shortening to the extent of about an inch, or even more. The limb is a little flexed, abducted, and everted (Fig. 413). It is very fixed, slight flexion being only permitted. There is pain in the course of the crural nerve, and the head of the femur makes a marked projection beneath Poupart's ligament, which is the distinguishing sign of the accident, and at once differentiates it from fracture.

Treatment.—The leg and thigh are flexed in slight abduction, and then swept inwards in adduction until near the median line; rotation inwards or rotation outwards may then be tried, the limb being extended at the same time. By these means the head of the bone retraces its steps round the acetabulum to the inner side and below, and the final manipulation slips it into the socket. Should the head of the bone have passed far under the crural arch, it would be right to flex the leg and thigh well, and draw or lift it downwards, until it is judged that the head of the bone is sufficiently disengaged to move freely on manipulation. The different methods of manipulation advised for this dislocation make it very probable that no one method will succeed for all cases

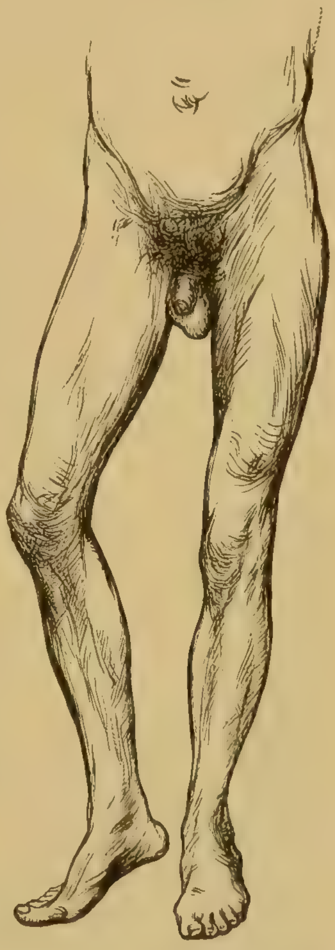

Fig. 413.—Pubic Dislocation of the Hip.

Anomalous forward dislocations.—The principal of these are as follows: Very violent and continued abduction may force the head of the femur into the perineum, where it may be felt, exercising pressure upon the urethra. All the symptoms of obturator dislocations are here very exaggerated. The head of the femur has been thrown forwards and directly upwards, and has been found resting upon the ilium, below the anterior inferior spine (subspinous). In all such displacements the detection of the head of the bone in its novel position will be the best guide to diagnosis, and the treatment can only be conducted by carefully studying the peculiarities of each individual case.

After consequences and after treatment of dislocations of the hip.—Should there have been extensive lesions of the soft parts, much weakness and lameness may long persist after dislocation of the hip. Should the sciatic nerve be injured, wasting of groups of muscles, especially the extensors of the foot upon the leg, may be observed. In the old and rheumatic, arthritis will probably supervene, with calcareous deposits about the articulation, and weakening of the large muscles about the hip. After reduction of a dislocation, the long splint should be applied for about fourteen days, and then daily manipulations, increasing in extent, should be carried out, with massage of the muscles, and galvanism, and this treatment should be long continued. If there has been any suspicion of fracture of the rim of the acetabulum, it must be remembered that if the patient places his weight on the foot too soon, the dislocation will be certainly reproduced. Treatment by extension and rest should be kept up for fully two months, in a heavy man, in these cases.

Compound dislocations of the hip.—The remarks concerning dislocations of large joints (page 957) may be studied in connection with this subject. Primary excision should always be preferred to amputation in compound dislocations of the hip.

Recapitulation.—To tabulate the methods of treatment of the four common dislocations of the hip by manipulation we may say:—

(1) Flex the thigh in adduction in the dorsal and sciatic dislocations. Flex the thigh in the abducted position in the obturator and pubic dislocations. The object of the flexion is to relax the Y-ligament.

(2) Abduct and rotate outwards in the dorsal and sciatic dislocations; adduct and rotate inwards in the obturator and pubic dislocations. These manipulations cause the head of the bone to retrace its steps through the capsular rent, and relax the short rotator muscles.

(3) Extend in all cases. This causes the head of the bone to enter the acetabulum.

Congenital dislocations of the hip.—Congenital displacements of the hip are comparatively common, and are of all kinds, yet the dorsal displacements are the more frequent. The affection is more common in females than in males. Congenital malformation would be a better term in many of these cases, which are often found combined with "monstrosities" of various kinds. The pelvis may be ill-developed, and I have seen congenital dislocation associated with deficiency of the symphysis pubis and extroversion of the bladder. The head of the femur is stunted in growth or flattened and oval in shape. The ligamentum teres is absent, or represented by a long atrophic cord. The capsule is replaced by ligamentous fibres. Some authors on the contrary describe the capsule in these cases as being unusually thick and strong. The anterior portion of it is often well developed, and markedly resists downward traction. The acetabulum is flattened and shallow, of triangular form, and its rim is deficient, the stunted femoral head thus readily slipping "out and in." A new socket lined with synovial membrane is sometimes found on the ilium. The muscles are frequently atrophic from chronic or infantile paralysis. Some are contracted, others degenerated, their true tissue being replaced by connective tissue and fat. Some difference of opinion exists as to whether this dislocation is unilateral or bilateral in order of frequency. From my own experience and the perusal of recorded cases, I am inclined to believe that the bilateral displacement is the more common. These cases must not be confounded with injuries of the hip-joints, produced by the efforts of the accoucheur in difficult labours.

Developmental peculiarities in congenital dislocations of the hip.—The idea that congenital dislocation of the hip is due to developmental error is far the most rational theory of the causation of this condition that we can adopt. In several recorded cases the iliac segment of the acetabulum has been absent, a shallow, triangular cavity being formed by the pubic and ischiatic segments. The essential point in the development of the acetabulum is a growth of pelvic cartilage round the head of the femur. This should be completed about the third month. The mesoblast about the joint becomes converted into the capsule and ligaments, while the mesoblast between the head of the femur and the acetabulum forms the ligamentum teres. Irregularities in the differentiation of the mesoblast may explain the fibrous bands sometimes met with, and the ill-formed capsule. In cases of congenital displacement of the head of the femur "an absence of the margin of the acetabulum is a prime feature" (Lockwood). Since motion is probably essential for the proper formation of a joint, it is interesting to observe that in some of these cases—notably in two recorded by Lockwood—the thighs have been acutely flexed, and the legs hyper-extended upon the thighs. Such a posture would prevent motion at the hip in intra-uterine life, and militate against proper development of the joint.

Symptoms.—The gait is waddling, with a characteristic roll; the buttocks and belly are extremely prominent. The heels are not well brought to the ground, the patient walking upon the "balls of the feet." There is very marked lordosis, the trochanters are much raised above the line of Nélaton (Fig. 414). There is free mobility, except in abduction, and traction in the flexed position restores the limb or limbs to their proper length. The displacement is at once reproduced, and there is a curious clicking sensation produced by the stunted head of the femur slipping "in and out" of its shallow cavity. The head of the bone is difficult to feel, since it is frequently atrophic and covered by large muscles. When the dislocation is unilateral, the patient walks with a limp, the hip is prominent, lordosis is not so marked. There is slight lateral curvature of the spine in the lumbar region, the convexity being towards the lame side. The trochanter lies well above Nélaton's line. The other symptoms will be the same. The lameness will have been noticed when the child began to walk. This negatives traumatic dislocation.

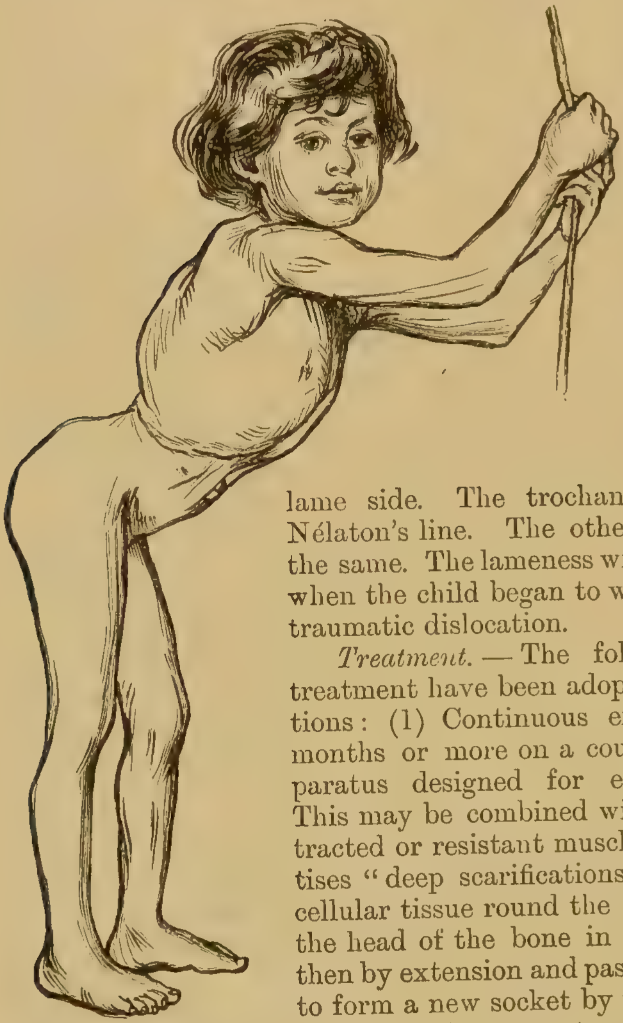

Fig. 414. — Bilateral Congenital Dislocation of the Hip. (From a case of Mr. Stanley Boyd's.)

Treatment. — The following methods of treatment have been adopted for these conditions: (1) Continuous extension for twelve months or more on a couch, and with an apparatus designed for each particular case. This may be combined with tenotomy of contracted or resistant muscles; (2) Guérin practises "deep scarifications" of the capsule and cellular tissue round the joint, in order to fix the head of the bone in a fresh position, and then by extension and passive motion attempts to form a new socket by pressure absorption.

Of continuous extension and the method of Guérin little need be said. Such plans of treatment are seldom now adopted, for the ultimate results are poor.

Operations for congenital dislocations of the hip principally owe their performance to the writings and teachings of Hoffa, published in 1890. Karewski in 1889 opened the joint by an anterior incision, and replaced the head of the bone, in a case of paralytic subluxation. He claims that this case was the first in which "bloody replacement" of a dislocated hip had been successfully accomplished.

The principles of Hoffa's operation may thus be summarised :—

(1) The joint is opened by a straight posterior incision, and the capsule is divided at its femoral attachment.

(2) The muscles attached to the trochanter major are divided as close to their periosteal attachments as possible. In children under five it is generally possible by flexion of the thigh, and direct pressure upon the head of the bone, to bring the latter into the situation of the old acetabulum. The hip and knee now remain flexed.

(4) The head of the bone being held firmly in its new position, an assistant gradually extends the thigh, so as to extend and stretch the biceps, semi-membranous, and semi-tendinous.

(5) A new acetabulum is formed, if needful, by the free use of Volkmann's spoon.

In children of over six years the hamstring muscles are divided near the tuber-ischii. The limb is next abducted, and the adductors divided subcutaneously. The limb is hyper-extended, and the fascia lata divided freely near the anterior superior spine. The head of the femur is now cut down upon by the posterior incision, the short muscles attached to the trochanter are divided, and the head of the femur is thrust out of the wound. The capsule must be very freely divided, especially the anterior part of it, and any ligamentum teres present cut away. Should the acetabulum be shallow or insufficient, a new cavity is formed by the free use of a sharp Volkmann's spoon. The cavity must be broad and deep. The head of the bone, by traction and manipulation, is fixed in this. The limb is placed in a fixed apparatus, with the thigh abducted, and careful asepsis must be aimed at. After four weeks, massage and cautious passive movements are carried out. Then an apparatus is constructed, which is worn for weeks or months. Cases of bilateral displacement wear a corset. Lorenz modifies Hoffa's operation by making an anterior incision. He divides the adductors and hamstrings subcutaneously. He lays stress on division of the anterior part of the capsule. Hoffa claims by his operation to minimise the waddling gait, and overcome the lordosis. He does not promise complete cure. Anchylosis only occurs if there is suppuration.

Paci recommends forcible manipulation of the limb as in a traumatic dislocation. Afterwards the limb is fixed in extension in a plaster-of-Paris splint for two months. On the removal of this an extension apparatus is applied. Kœnig's plan of raising a portion of bone and periosteum from the ilium has received little support. Hoffa has lost some of his cases from shock and hæmorrhage, and fatal peritonitis has occurred from attempts to form a new acetabulum with gouge and mallet, for the bone is thin and weak, and easily perforated. The position of the wounds renders perfect asepsis very difficult to maintain, and the occurrence of suppuration and necrosis would be disastrous. I can testify that the operation may be very difficult, and attended with troublesome bleeding. In England the conditions obtained by Hoffa's operation have been generally disappointing, and far inferior to the results claimed by the originator. An operation—like that of Lorenz—can only be carried out between the ages of four and seven years. The immediate results may be encouraging, but the ultimate results do not appear to have been at all satisfactory. Possibly improved methods of operating may yet succeed in ameliorating the crippled condition of these patients. At the present time most English surgeons are content to adopt some method of artificial support.

Perhaps one of the most efficient forms of apparatus for congenital dislocation of the hip was devised by the late Marcus Beck. It consists essentially of a padded metallic belt, which encircles the body between the trochanter and crest of the ilium, and is continuous with a moulded leather cap, which encloses the upper part of the femur. From this a light steel rod passes up into the axilla, and so to some extent the weight of the body is taken off the limb.

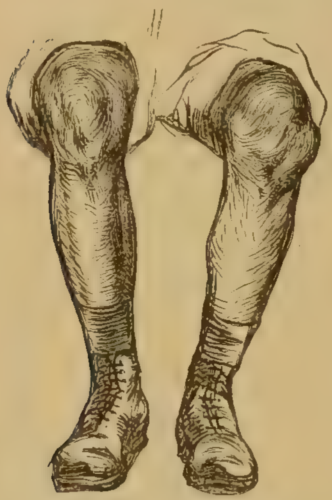

Fig. 415.—Congenital Dislocation of the Patella outwards.

Dislocation of the patella. Causation and classification. — Dislocation of the patella may be caused by muscular action when the limb is in the extended position. Owing to the oblique direction of the femur, there is a natural tendency for the quadriceps to pull the patella outwards. This is counteracted by the greater prominence of the external condyle, and, perhaps, also by the fibres of the vastus internus, which descend lower than the fibres of the vastus externus. The accident is predisposed to by lax ligaments, and is sometimes congenital. In congenital displacements, the vastus internus is frequently wasted and atrophic, and the external condyle of the femur deficient. The vastus externus consequently draws the patella outwards (Fig. 415). The patella itself is, in these cases, small and atrophic, and developmental error is obvious. Genu valgum is said by many authors to predispose to this accident. Direct blows on the inner edge of the patella may also dislocate it outwards.

The external displacement is far the more common, the internal very rare. Occasionally the patella is found displaced vertically, with its inner edge turned directly forwards. The upward and downward displacements, from rupture of the ligamentum patellæ or quadriceps extensor, should hardly be regarded as dislocations. Many of the dislocations are incomplete, but occasionally the bone is completely thrown to the side of the femur. In the incomplete displacements the capsule of the knee may remain intact, in the complete it is usually lacerated.

Symptoms.—The knee is fixed in semi-flexion, it has an appearance of widening, sometimes it is extended. The quadriceps stands out in bold relief, and is directed outwards or inwards according to the displacement. There is a depression over the inner condyle in the dislocation outwards, and a prominence over the outer condyle formed by the patella, with its external border projecting forwards under the skin, and its internal border buried in the inter-condyloid notch.

If the displacement is complete, the patella is seen as a hard, flat mass on the outer side of the joint, with its inner edge projecting forwards, while the front of the condyles is readily to be felt. In the rare inward displacement the same symptoms are observed, but of course, reversed. In the vertical displacement the limb is fixed in extension, the outer edge of the bone lies in the inter-condyloid notch, and the inner edge projects forwards against the skin (Fig. 416). Cases are recorded where the bone has been turned quite round, with the anterior surface against the front of the femur. The cause of this complete rotatory displacement is not clear.

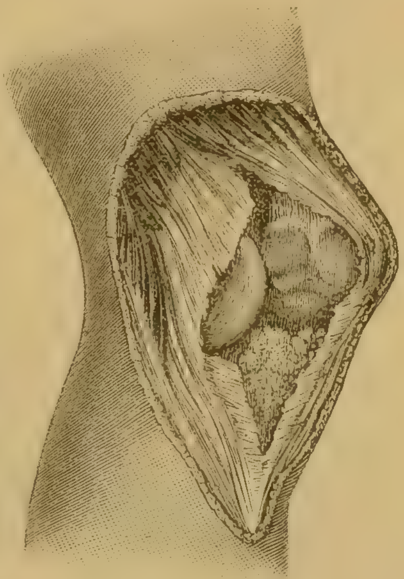

Fig. 416.—Vertical Displacement of the Patella.

Treatment.—The thigh should be flexed on the body, and the leg completely extended to relax the quadriceps; when under anæsthesia the surgeon will usually be able to press the bone back into position (Fig. 417). Flexion with pressure manipulation may also be tried. After reduction, rest on a splint and the ice-bag must be adopted. A strong knee-cap, well padded externally, should afterwards be constructed and worn, as there is a tendency to recurrence of this dislocation in those with lax ligaments. Remarkable difficulty has sometimes been experienced in the reduction of the rotatory vertical dislocation. This is usually declared by authors to be due to the wedging of the bone into the inter-condyloid notch. Considering the size of the latter as compared with the edge of the patella, this explanation seems to me hardly satisfactory. It is far more likely that the bone lacerates the capsule, and is tightly girt in its new position by the tough ligamentous fibres of this structure. In the cases I have seen, no difficulty was experienced in reduction, by very complete extension of the leg, and forcible pressure upon the prominent edge of the bone, under deep ether anæsthesia.

Supposing, however, the displacement remains irreducible, what course is to be adopted? Subcutaneous division of the ligamentum patellæ has not proved very satisfactory; the bone has remained obstinately fixed after this, and more extensive subcutaneous cutting measures have been vainly adopted. A fairly useful joint results if the case be left to nature. Bearing this in mind, the surgeon will do well to leave such a case alone, unless he has complete confidence in his practical knowledge of perfect aseptic surgery. With elaborate care and precaution, it would be justifiable freely to open the joint in such a case, and divide those structures which could thus be certainly felt and seen, preventing reduction. The wound in the capsule and soft parts over the joint should be accurately united with scrupulous attention to every detail that may directly or indirectly promote rapid and perfect union.

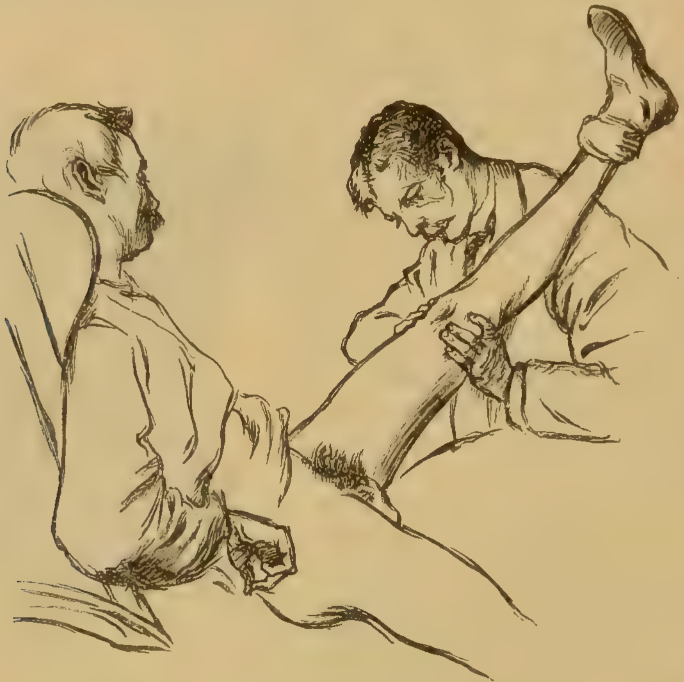

Fig. 417.—Reduction of Dislocation of the Patella by relaxing the Quadriceps and direct Pressure.

Dislocation of the fibula from the tibia. Causation and classification.—The upper end of the fibula may be displaced from the tibia forwards, backwards, or upwards. In the latter accident, which is very exceptional, one case having been recorded by Malgaigne, the whole bone is forced upwards, its lower extremity being also displaced. The usual cause of dislocation forwards or backwards is a heavy blow, or crushing violence directly applied to the head of the bone. Displacement may also be brought about by the violent action of the biceps. Dislocation of the lower end of the fibula from the tibia is said to have occurred from direct violence, and also in some dislocations of the astragalus. In "Dupuytren's fracture," the lower end of the fibula is torn away from the tibia. Dislocation of the upper end must not be confounded with (1) undue mobility of the head of the bone from laxity of the ligaments, or

(2) oblique fracture of a portion of the outer tuberosity of the tibia, the fragment being drawn upwards and backwards, carrying the fibula with it.

Symptoms.—The leg is partially flexed, and flexion and extension are much impaired, while severe pain may be felt from pressure on the popliteal nerve. There is a depression over the normal site of articulation, and the head of the bone will be felt in its new position. The tendon of the biceps is excessively prominent.

Treatment.—By flexion of the leg and thigh, and pressure on the head of the displaced fibula, reduction can usually be effected. It is difficult to keep the head of the bone in proper position; should it remain displaced, some permanent lameness may be apprehended. The plaster-of-Paris bandage accurately applied, the limb being kept extended for some six weeks, would seem to be the best treatment. Afterwards a supporting apparatus must be constructed and worn.

To present the dislocations of the fibula for consideration in a tabular form—(1) the upper end of the fibula may be dislocated from the tibia forwards, backwards, or upwards in certain dislocations of the ankle; (2) the lower end of the fibula may be displaced from the tibia in dislocations of the astragalus or ankle. It is also torn away from the tibia in "Dupuytren's fracture." Direct backward displacement of the lower end of the fibula has been related.

Dislocations of the knee-joint. Causation and classification.—The tibia is not displaced from the femur, except by extreme violence, since the ligaments are powerful, and the articular surfaces broad. Dislocations of the knee are found, therefore, in such accidents as those caused by powerful machinery, with violent wrenchings and twistings of the lower limb. Dislocations of this joint may be congenital, and are found in various diseases of the articulation, especially in severe cases of locomotor ataxy (Charcot's joint). The tibia may be thrown outwards, inwards, backwards, or forwards. The lateral dislocations are generally incomplete, the antero-posterior complete. The tibia is often also a little rotated on its longitudinal axis, and the head of the bone may be displaced forwards and outwards, or backwards and inwards, displacements compounded of the four first-named dislocations.

These dislocations may be thus tabulated in order of frequency:—(1) Outwards, inwards, usually partial; (2) forwards, backwards, usually complete; (3) oblique partial displacements, as forwards and outwards, or backwards and inwards.

Condition of the soft parts.—In the complete dislocations forwards or backwards, the capsule, lateral, and crucial ligaments are extensively lacerated. The hamstring muscles may be torn across, the popliteal vessels and nerves pressed upon or even ruptured. Gangrene of the foot and leg has consequently followed severe antero-posterior dislocations of the knee. In the side displacements the lateral ligaments are torn to a variable degree. The more complete the displacement, the more extensive the laceration of the ligaments, and also of the muscles and tendons about the joint.

Symptoms.—Great variety of position as regards flexion or extension of the leg has been observed. The complete antero-posterior displacements are frequently compound, and the bones of the leg being drawn upwards, considerable shortening is to be found. In dislocation backwards the head of the tibia makes a prominence in the popliteal space, resting against the back of the femoral condyles, while the condyles of the femur and the anterior surface of the patella resting upon them can plainly be seen and felt (Fig. 418). In the complete displacement forwards the conditions are reversed, the prominence in the popliteal region being caused by the lower end of the femur, that in front by the head and articular surfaces of the tibia (Fig. 419). In the lateral displacements the prominence of the upper tuberosities of the tibia and head of the fibula to one or the other side, and the corresponding depression opposite, with the condyle of the femur projecting, are sufficiently obvious signs. There is no difficulty about the diagnosis of these accidents, the nature of them being usually obvious at a glance. Separation of the lower epiphysis of the femur with displacement backwards has been mistaken for a dislocation. In such an injury the patient will be under twenty years of age, and the movements of the condyles with the tibia should guide the surgeon to a correct diagnosis. Careful comparative measurements should also be made between the condyles and the head of the tibia on either side.

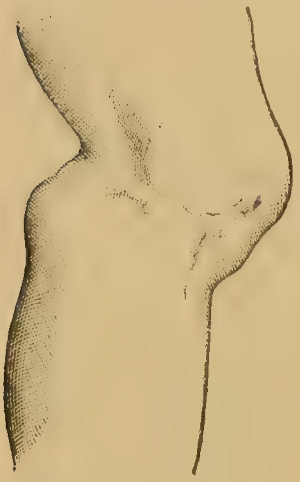

Fig. 418.—Appearance of Knee in Dislocation backwards. (After Pick.)

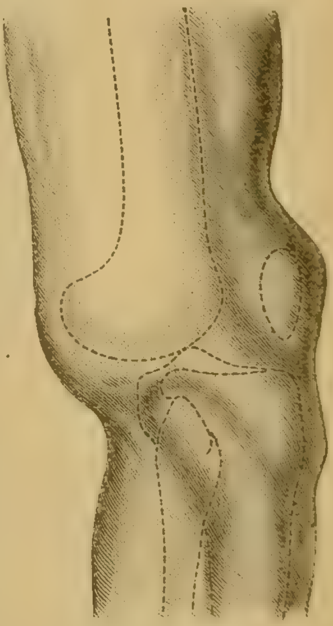

Fig. 419.—Dislocation of the Knee forwards.

Treatment.—By flexion of the leg and rotation, combined with traction in the flexed position and counter-extension from the thigh, these displacements may be reduced. In the complete dislocations powerful extension is often needful, counter-extension being made on the thigh. Afterwards careful splinting should be applied, and the ice-bag may be employed, but with caution. A strict watch should be kept on the toes and foot, should any injury to the vessels have occurred. The plaster-of-Paris dressing should soon be applied, and the part kept immovable for at least six weeks, when a leather splint may be substituted, and passive motion cautiously commenced. The patient must be warned that after so severe an accident, weakness and insecurity of the joint must long exist, with persistent œdema of the foot and leg, and a tendency to congestion and eczema of the skin. Dislocations of the knee are often compound; the accident is a most grave one, and if combined with laceration of the vessels, demands amputation.

Sub-luxation of the knee, internal derangement of the knee, dislocation of the semi-lunar cartilage.—Under this heading is described a peculiar condition of the knee-joint, which almost invariably is associated with a wrench, sprain, or other injury, and which is characterised by a sudden sensation of the joint being "put out," some fixation and impediment to the movements of the limb, more or less pain, and lastly by the functions of the articulation being suddenly and perfectly restored by an appropriate manipulation.

Causation and pathology.—The accident is usually produced by a twist to the leg, when the knee-joint is flexed and the tibia is fixed, the body and femur being suddenly rotated on the tibia. Thus the injury is experienced in swinging the body to make a stroke at "golf," or in the rotation which occurs in stepping out of a dogcart (Treves). On the other hand, violent rotation of the tibia when the femur is fixed, may produce displacements, though less commonly. In flexion and extension the cartilages move with the tibia. In rotation one or other cartilage is fixed, and the tibia rotates beneath them. In practice the internal cartilage will be found to be most generally displaced. This occurs in violent external rotation, principally performed by the biceps. During this movement the external cartilage is gripped firmly between the tibia and external condyle. The gap between the internal condyle and the tibia is, however, increased, and the movable internal cartilage is apt to slip between the internal condyle and the inner tuberosity of the tibia. During internal rotation the internal cartilage is firmly held between the bones. This movement is principally performed by the popliteus. The gap between the external condyle and external tuberosity of the tibia is now increased, but the external cartilage is smaller than the internal, is rounder and more movable, and is thus less likely to get nipped between the bones (Scott Lang).

Some authors take exactly the opposite view, and Godlee has argued from anatomical considerations that the external cartilage is more frequently displaced. Clinical experience points, however, undoubtedly to the greater frequency of the displacement of the internal cartilage, and practically this may be considered to be the cartilage nearly always at fault.

Marginal displacements of the cartilage are far the more common, but vertical displacements are not unknown (Fig. 420). In reference to this subject, it may be pointed out (1) that portions of the internal cartilage may be partially detached by violence, (2) that pre-existing inflammatory outgrowths found in chronic arthritis may also be detached, and (3) that these conditions may closely simulate in their symptoms a true displacement of the main body of the cartilage.

Symptoms.—After a sudden external twist of the knee, as in alighting from a vehicle, or turning rapidly while playing football, a sudden severe pain is felt in the joint, followed shortly by some effusion. This limb is usually held in the slightly flexed position, and complete flexion and extension cannot be performed. The patient can walk, but he keeps the knee slightly flexed and stiff, and is conscious that " all is not right " with the articulation. Passive flexion and extension can be performed, but not to the full degree. There are great differences in these cases in the severity of the symptoms complained of. A projection may be felt on the inner side of the joint, and pain is usually experienced here on pressure. In the case of vertical displacements, a depression may be detected. The reader will see that the group of symptoms above related closely simulate those of " loose cartilage " within the knee-joint, especially when a pre-existing inflammatory, pedunculated outgrowth is detached by a wrench or twist. Lateral movement of the tibia upon the femur may be detected on firmly grasping the leg and thigh, and making appropriate manipulations (Treves).

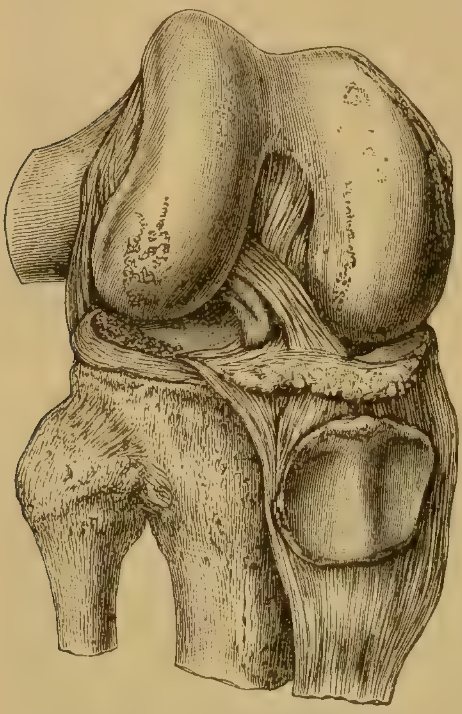

Fig. 420.—Vertical Displacement of part of the External Semi-lunar Cartilage. (After Godlee.)

Treatment.—Should any projection be felt at the inner side of the joint, the thumb should be firmly pressed upon it, and the leg flexed. The leg is now brought up suddenly in full extension, rotating inwards and outwards at the same time. This process is often agonisingly painful, and full anæsthesia with nitrous oxide gas is very advantageous. Afterwards, in recent cases, the joint should be fixed in plaster of Paris for at least five weeks, to allow the torn or displaced cartilage to unite. An apparatus must be worn for three months, or longer, should lateral movement of the tibia on the femur be obvious on manipulation (Fig. 421).

Recurrence of the symptoms is very frequent from any slight twist or muscular exertion beyond the common ; and sometimes this happens so frequently that the affection becomes chronic, and the patient is incapacitated for the duties of active life. The aid of a skilled surgical mechanician should be sought in these cases, and an apparatus should be constructed, designed to support the knee, and to make direct pressure on the site of displacement.

In case the apparatus fails or is ill-borne, the propriety of an operation may be considered. This consists in laying open the joint with all the precautions and in the same manner as is adopted in the ordinary operation for loose cartilage. The incision should be placed over the site of the supposed displaced cartilage. All bleeding vessels, even the smallest, should be clamped or twisted, and the wound being well held asunder with fine retractors, the disorganised part of the articulation is exposed to the eye and finger. Should a displaced cartilage be found, it is now brought into its place, and firmly fixed there by sutures of aseptic silk, which are passed with a strong curved needle through the periosteum and fibrous aponeurosis at the edge of the tibia. Should a portion only of cartilage be displaced, having been stripped away ribbon-like from the main cartilage, it had better be removed *in toto.* This operation, performed with all the elaborate care and attention to detail which its gravity warrants, has given excellent results in some otherwise obstinate cases. The necessity for it, if a proper apparatus be constructed, is probably rare. In some instances, perhaps more frequently than is generally known, the knee-joint has been opened, and no definite pathological lesion found. In a minority, disastrous results, as suppuration and fibrous anchylosis, in faulty positions, have occurred, so that no one should undertake this proceeding without careful consideration of all the circumstances of the case, and a full confidence in his own powers of producing and maintaining perfect asepticity. The uniting of the wound, including the synovial membrane, and the general after-treatment, are in all respects those with which we are familiar in the operative treatment of "loose cartilage" in the knee, and will not here be alluded to.

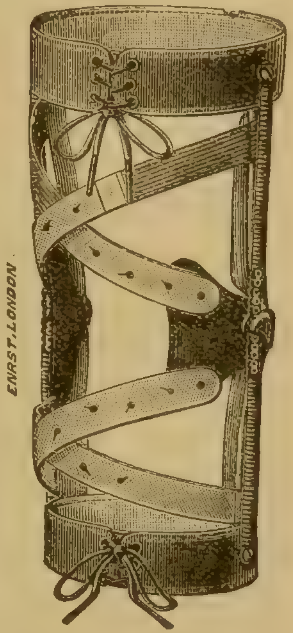

Fig. 421.—Ernst's Apparatus for Displaced Semi-lunar Cartilage.

Dislocations of the ankle-joint proper. *Causation and classification.*—These injuries, like most others about the foot, are caused by violent wrenches and twists, as when a heavy man "misses a step" in the dark, or jumps out of a vehicle in rapid motion. The antero-posterior displacements are usually caused by some sudden fixation of the foot when the body is in rapid motion. Thus, a man running may suddenly put his foot in a hole, and the weight and impetus of the body will force the tibia forwards on the scaphoid, and thus produce a dislocation of the foot backwards. Again, a person jumping from a great height, and alighting on the front of the foot, may readily suffer from a dislocation backwards. In exceptional instances these displacements are produced by direct violence, and the rare dislocation of the foot forwards has been described as occurring from the effects of a heavy blow on the front of the tibia, driving that bone backwards on to the upper surface of the os calcis. All dislocations of the ankle may be complete or incomplete, but the incomplete are far more common. A tabular view of dislocations of the ankle *in order of frequency* and importance would be as follows :—Outwards, inwards, backwards, forwards, and upwards between the tibia and fibula. The ankle is also displaced in the upward direction in Dupuytren's fracture, when the foot and astragalus, still connected with the lower fragment of the broken fibula, are forced upwards along the outer side of the tibia. In the lateral displacements fracture almost invariably co-exists.

Lateral dislocations of the ankle outwards or inwards. *Morbid anatomy.*—These displacements are almost always combined with fracture of the malleoli, the lateral ligaments being of great strength. The astragalus is also usually rotated on its antero-posterior axis, so that its lateral articular surfaces look obliquely upwards. It is exceptional for the astragalus and foot to be displaced horizontally outwards or inwards ; yet such dislocations occur, and the broken malleolus is usually displaced outwards or inwards with the foot. In the complete horizontal displacement of the foot outwards, the foot and lower fragment of the fibula may be drawn upwards along the outer side of the tibia until arrested by the lower end of the upper fragment of the fibula. This is termed Dupuytren's fracture. The common outward lateral displacement is incomplete. The fibula is broken about four inches from the tip of the outer malleolus ; the tip of the internal malleolus is torn off by the powerful deltoid ligament, and the astragalus and foot are rotated so that the outer articular facet of the astragalus looks obliquely upwards and outwards (Pott's fracture). (*See* page 868.) In the incomplete dislocation inwards, the external malleolus is broken off, or the external lateral ligament ruptured. The inner malleolus may remain intact or be separated obliquely from the tibia, and the astragalus is rotated so that its inner articular surface, carrying with it the inner malleolus, looks upwards and inwards. Lateral dislocations, especially outwards, are frequently compound. The complete outward or inward horizontal lateral displacements are exceedingly rare, and can hardly occur without fracture of the malleoli.

Symptoms.—In the dislocation outwards the foot is markedly everted by the peronei, its outer border looking upwards. The sharp broken edge of the inner malleolus is readily seen and felt at first, but soon becomes obscured by a fluctuating collection of blood. A depression about four inches up the outer side of the leg marks the site of the broken fibula. In the ~~lateral~~ dislocation inwards these symptoms are reversed. The outer malleolus is prominent, the inner buried, or frequently broken off. The foot is markedly inverted. In both these injuries there is an appearance of distortion, and widening about the front of the joint. Should a surgeon be called to see such a rare accident as a complete horizontal lateral dislocation, he would expect to find the following symptoms: The displacement will probably be in the outward direction. There will be striking deformity caused by the great widening of the parts. The lower end of the tibia and inner malleolus will be seen and felt internally, on the point of bursting through the skin. The everted foot and fractured external malleolus will be drawn upwards along the outer side of the leg. In connection with the preceding paragraphs, the article on fractures about the ankle may well be studied. In actual practice, fracture is usually combined with dislocations of the ankle.

Antero-posterior dislocations of the ankle.—Owing to the fact that the interval between the malleoli is greater in front than behind, and that the astragalus is a little narrower anteriorly, one would expect that dislocation forwards would be the more common of the antero-posterior displacements. Dislocation backwards is, as a matter of fact, the more frequent of these two very exceptional accidents, and this is due to such a cause as alighting on the front of the foot in jumping from a height, or from a vehicle in rapid transit. The dislocation backwards is of various degrees of completeness. Usually the tibia is forced forwards on the upper surface of the neck of the astragalus, impinging upon or overhanging the scaphoid bone (Fig. 422). The lateral ligaments are torn, more often the malleoli are fractured, and carried backwards with the foot and astragalus. The foot is pointed downwards, and greatly shortened, the prominence of the heel behind is markedly increased, and the tight tendo Achillis is concave backwards. Anteriorly, the transverse projection and marked ridge of the lower end of the tibia, with the skin, are thrown into folds beneath it. In the very rare dislocation forwards the foot is considerably elongated. The tendo Achillis is slackened and carried forwards and in front of it are the hard prominent lower

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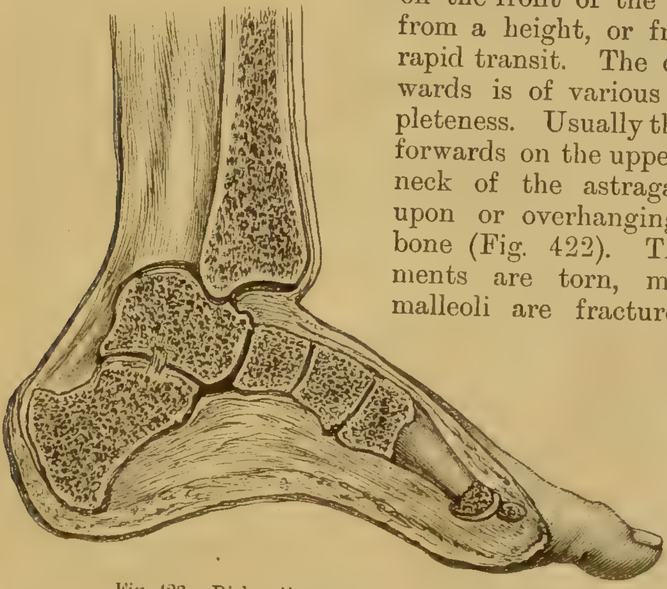

Fig. 422.—Dislocation of the Foot backwards.

ends of the tibia and fibula. Should the malleoli and lower end of the tibia move with the displaced foot, the case will be one of fracture or displacement forwards of the lower epiphyses of the tibia and fibula. This latter accident has been confounded with true dislocation, which at sight it much resembles.

Dislocation of the ankle upwards.—It has been pointed out that one variety of dislocation upwards is that described as *Dupuytren's* fracture, namely, when a complete outward horizontal displacement of the foot and lower end of a broken fibula are drawn upwards by the leg muscles. Occasionally it happens, however, that the strong ligaments uniting the tibia and fibula are ruptured, and the astragalus and foot pass directly upwards, separating the bones, and being jammed tightly between them. As might be expected, the enormous strength of these ligaments renders this an excessively rare accident. Fracture of the malleoli may complicate it. Should the reader meet with an instance of dislocation of the astragalus and foot upwards between the tibia and fibula, he would recognise it by the extraordinary widening of the joint, and the displacement outwards and inwards of the lower ends of the fibula and tibia, which are ready to burst through the skin on either side, and are also approximated to the sole of the foot, so that if the patient were to walk, the malleoli would touch the ground. The foot is quite immovable, being wedged between the leg bones, and this seems to have been a very marked sign in the recorded cases.

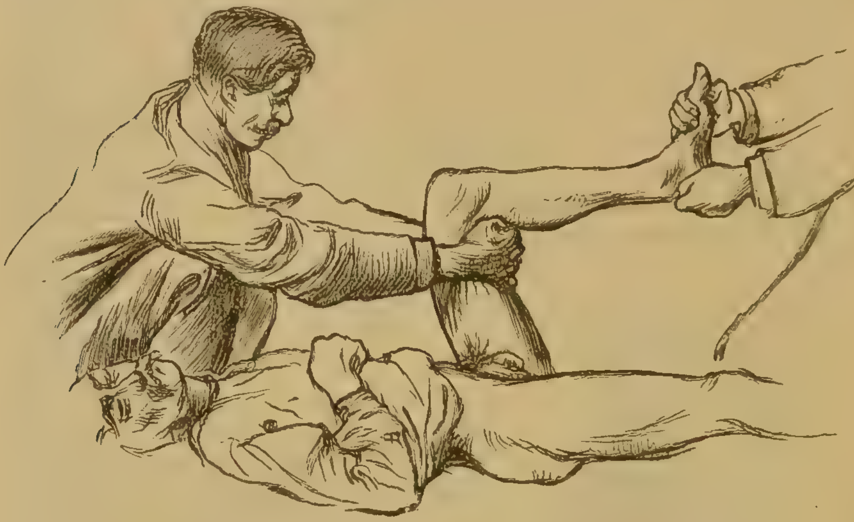

Fig. 423.—Manipulations for Dislocations of the Ankle.

Treatment of dislocations of the ankle.—The leg should be well bent upon the thigh, and the thigh on the abdomen, and counter-extension should be made on the thigh by the hands of an assistant, or a strong towel fastened behind the patient. The surgeon then seizes the foot and heel, and makes extension, inverting or everting the foot at the same time, as occasion requires (Fig. 423). The flexion of the leg relaxes the powerful calf muscles, and it is most important that this position be carefully observed. Anæsthesia is generally advisable, and the parts are wont to return into position gradually, and not with a distinct snap. The tendo Achillis may be subcutaneously divided in cases of great obstinacy, but this should be avoided if possible. In the dislocation of the foot backwards the surgeon should draw the front of the foot forwards and upwards, while he presses the tibia backwards with the other hand. The leg will then, of course, be thus acutely flexed on the thigh. Great difficulty has been experienced by those surgeons who have had the rare opportunity of treating a dislocation of the foot forwards. The tendo Achillis seems usually to prevent sufficient extension from being exerted to slip the astragalus backwards. The mode of reduction then is to divide this tendon if needful, and make powerful extension from the foot and heel, and when the foot is disengaged, press it backwards. In the dislocation upwards by separation, reduction has also been found impossible, but a useful limb has ultimately resulted. It is questionable in modern days if this dislocation should be left unreduced. An aseptic excision of the astragalus would probably leave a better foot. In neglected cases of displacement at the ankle, excision of the astragalus or amputation may subsequently become needful.

So soon as reduction is effected, the parts should be carefully fixed in splints while the leg is still in the flexed position, and the greatest care should be taken that the apparatus is well padded, and that no undue pressure be exercised, so as to cause the damaged skin to slough. The limb should be well elevated, and cold spirit lotion sedulously employed. Enough prominence has hitherto not been given to the tendency to displacement backwards in many of these cases, when combined with fracture of the malleoli. This is especially apt partially to occur in a dislocation backwards, when, as often happens, a piece of the lower end of the tibia is also broken off. As the limb lies on a posterior splint, such a displacement is readily overlooked, and the patient recovers with a disastrous antero-posterior deformity. This backward displacement is largely due to the tension of the tendo Achillis, which structure may need tenotomy; it is best guarded against by the treatment now to be described.

I have found the plaster-of-Paris dressing applied with scrupulous care and accuracy better than anything else in the after-treatment of dislocations about the ankle. Only those who have had practical experience of these troublesome injuries know the difficulties encountered in treating them with ordinary splints. So soon as the swelling and extravasation have subsided—say in about ten days—the foot should be held in absolutely correct position by a trustworthy assistant. He should see that the foot is at right angles to the leg, and that there is no "dropping" of the heel backwards. An anæsthetic will often facilitate and ensure accurate position. A fine flannel or domette bandage is carried from the toes to the centre of the leg, and over this the ordinary plaster dressing is firmly, not tightly, applied with narrow muslin bandages. In ten minutes the bandage will be "set," and if the assistant has maintained the foot in proper position, it will there remain, and healing will take place of the broken malleoli and ruptured ligaments, under the most favourable conditions. There is no objection to the application of plaster-of-Paris immediately on reduction, if the surgeon be careful to divide it anteriorly, to allow of swelling. Great care and caution must be exercised, however, as the swelling is very considerable, and gangrene may readily ensue from undue external pressure. In six weeks cautious passive motion and massage may be carried out. In elderly and heavy people, of the gouty or rheumatic taint, a dislocation at the ankle, combined with fracture, will leave a joint that for many months will be œdematous and insecure, entailing long, and perhaps permanent, lameness, and the prognosis of such injuries should always be very guarded.

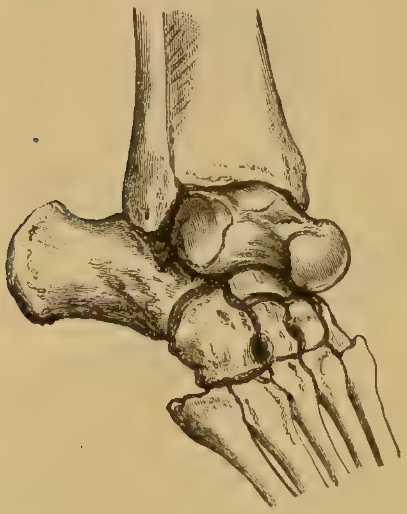
Fig. 424.—Partial Dislocation of the Astragalus forwards. (After Pick.)

Compound dislocations of the ankle-joint. — These very serious injuries are comparatively common, especially in the lateral dislocation outwards, where the sharp edge of the broken inner malleolus tears through the skin, and projects several inches, tightly girt by the blackened integument. If the soft parts are much crushed, and especially if the patient be aged, bloated, or intemperate, and the wounds full of dirt and grit, or such substances as machinery oil, I would strongly advise primary amputation of the leg. In some favourable circumstances, especially in the young, and in healthy country persons, an attempt may be made to save the foot. From a careful perusal of the recorded cases of this accident, and from personal notes of five instances of it, I think that it is always better to remove obliquely the projecting inner fragment with a fine saw, preserving the periosteum as much as possible. This affords free drainage, and a counter-opening may be made posteriorly if needful. In compound dislocations of the ankle, continuous irrigation with warm water is of striking utility. Great practical difficulty will be encountered in the proper disinfection of these complicated wounds, and the surgeon will be prepared to combat such septic disorders as spreading gangrene, abscess, cellulitis, and tetanus. As soon as practicable, the plaster-of-Paris dressing, with an opening cut at the site of injury, is an advantageous application.

Dislocations of the astragalus (Fig. 424). **Causation and classification.**—The fact that a bone deeply placed and strongly fixed, like the astragalus, can be forced out of its bed by injuries, is a striking instance of the curious results that may follow extreme and composite violence applied to joints. A typical injury to cause dislocation of the astragalus would be the entanglement of the foot in powerful revolving machinery. It may also be brought about by very extreme and violent extension, in which movement the head of the astragalus tends to start forwards, and by similar accidents to those which produce dislocations at the ankle: namely, heavy falls on the extended feet, which are twisted and wrenched severely at the same moment. Oblique dislocations forwards are the more common, and in *order of frequency* we may tabulate dislocations of the astragalus as follows: (1) Forwards and outwards; (2) forwards and inwards; (3) directly forwards (rare); (4) backwards and outwards or inwards; (5) directly backwards (very rare).

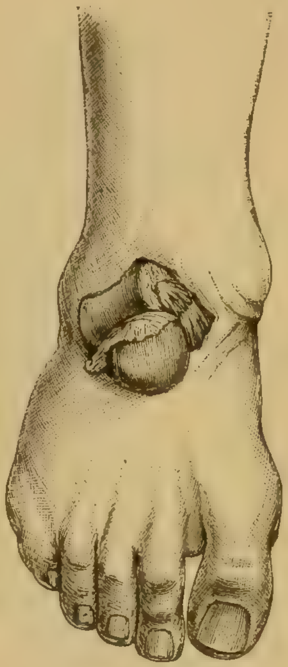

Fig. 425.—Compound Dislocation of the Astragalus.

In any of these displacements, rotation of the astragalus on its antero-posterior axis may also occur, and cases are related where the bone has been turned almost upside down. A curious displacement is also described, when the astragalus is rotated transversely across the joint. Such accidents are exceptional, and their symptoms are sufficiently obscure to render accurate diagnosis impossible. The forward displacements are assumed to occur when the foot is extended, and the astragalus advances forwards from under the tibio-fibular mortice. The dislocation backwards may take place in like manner when the foot is flexed forwards upon the leg, and the rotatory displacements in the horizontal or vertical axis are supposed to occur when the foot is at right angles to the leg. Dislocation directly outwards and inwards is also described, even without fracture of the malleoli, though this may well seem impossible (Fig. 425). A large number of dislocations of the astragalus are compound, the diagnosis being thus quite obvious, and the bone has even been thrown clean out of the foot, and found lying on the ground.

The literature of dislocations of the astragalus makes it clear that confusion has often arisen between this accident and the sub-astragaloid dislocation. The main distinction is the position of the head of the astragalus with regard to the malleoli, which is unaltered in the sub-astragaloid dislocation.

Morbid anatomy.—Dislocations of the astragalus may be complete or incomplete, and are sometimes combined with fracture of the malleoli. The neck of the bone may be broken, the head still retaining its position with the scaphoid (Fig. 426). The lateral, and strong calcaneo-astragaloid ligaments are more or less extensively torn, according to the completeness of the dislocation; and the tendons, vessels, and nerves are stretched, or even lacerated. The tendo Achillis and tibials draw up the calcaneum against the tibia in the complete dislocations, and the posterior tibial tendon may be partially hitched round the neck of the astragalus. The tension of these structures form the principal obstacles to reduction. The astragalo-scaphoid ligaments are always ruptured, unless fracture of the neck of the bone occurs, which complication is common in the backward displacement.

Symptoms.—The possible varieties of dislocations of the astragalus being so numerous, many pages of the described concomitant symptoms might easily be compiled from the records of reported cases.

The following rules for diagnosis will be found useful:—

(1) In complete and compound dislocations the diagnosis is usually obvious, the contour and shape of the bone being distinctly felt or seen.

(2) In incomplete dislocations the position of the head of the astragalus is the main guide to a correct diagnosis, as its distance and relation to the malleoli are altered, as compared to the sound side. The head of the astragalus is always to be detected when displaced from the scaphoid forwards and outwards, or inwards.

(3) In rotatory displacements pure and simple the diagnosis will be inferential, and is only to be approximately made by the possibility of feeling the displaced articular surfaces in deep manipulation under anæsthetics. This is usually impossible, from the rapid swelling. In the displacements forwards and outwards, or forwards and inwards, there is a manifest projection in front of the tibia, the articular end of the latter bone being sunk behind it, the malleoli nearly reaching the sole. In the dislocation outwards and forwards the foot is adducted, and the inner malleolus buried, while the head of the astragalus and external malleolus project externally. The reverse occurs in the forward and inward displacement.

(4) In dislocation backwards the displacement is usually oblique, as backwards and outwards, and the bone may also be twisted on its antero-posterior or transverse axis. The displaced bone makes a lumpy prominence above the heel, pushing the tendo Achillis backwards (Fig. 426); and on one side or the other the contour of the superior and lateral articulating surfaces of the astragalus can usually be made out, with the skin stretched tightly over them. The dorsal surface of the foot is shortened, and the tibia thrown forwards.

(5) The lateral dislocations of the astragalus are really usually exaggerated instances of the forward and lateral displacements. True horizontal complete displacements are, however, found. They

are usually compound, and combined with fracture of the malleoli; and I am not disposed to believe that complete displacement laterally of this bone can occur without fracture. The hollow on the one side of the foot, and the marked projection of the displaced bone on the other, will make the diagnosis clear, even though the injury is not compound.

(6) In all displacements of the astragalus the movements of the ankle are abolished, and much extravasation of blood occurs, with the most severe inflammation and consecutive swelling, so that unless the case is seen promptly it may be quite impossible to make out the exact nature of the injury.

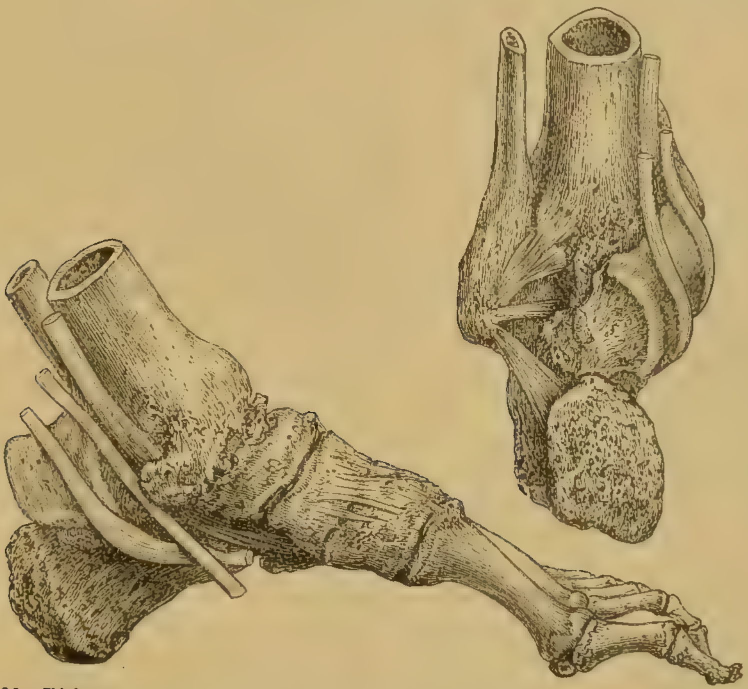

Fig. 426.—Dislocation of the Astragalus backwards, with fracture of the Neck. The head of the bone remaining *in situ*. (St. Thomas's Hospital Museum.)

Treatment.—Reduction should be attempted by well bending the knee, making counter-extension from the thigh, and drawing upon the foot by the heel and toes, while an assistant makes direct pressure upon the displaced bone. The foot may, at the same time, be firmly abducted or adducted. Tenotomy of the tendo Achillis and posterior tibial tendon is a valuable aid, and should be performed in obstinate cases.

Recent incomplete dislocations will usually be reduced by these means. In complete displacements such efforts will probably fail. What next is to be done? It is impossible to lay down definite rules for all varieties of these very variable accidents. It may here be observed that in unreduced dislocations backwards more recoveries with useful limbs have been recorded than in any other. The following *rules* are of general application only, and the results of modern aseptic surgery may serve as an apology for differing from such authorities as Broca, Morris, and Bryant :—

(1) In all complete compound dislocations of the astragalus excise the displaced bone at once, if reduction is impossible.

(2) In all irreducible complete simple dislocations excise the bone, as the injury generally becomes secondarily compound, with sloughing, fever, and severe constitutional disturbance.

(3) In backward irreducible dislocations or rotatory displacements, with abolition of the functions of the ankle joint, excise the bone in the young and healthy ; leave it alone in the aged, diseased, or intemperate.

When performing excision of the astragalus in these cases, it is the greatest error to gouge out the bone piecemeal through a small opening, as is too usually the practice. Bearing in mind the extreme violence that has been at work, and the probable extensive laceration of tissues, a free anterior incision should be made, the whole bone removed, and no fragments of it left to necrose and cause irritation. Effused blood should be flushed away with the hot douche, and fragments of torn ligaments, muscles, and tendons trimmed with a sharp scissors. All cut tendons should be united by catgut, and most free and efficient drainage provided in a dependent position. In the dislocation backwards an appropriate incision should be made posteriorly, if the bone is to be removed. The foot after excision of the astragalus is surprisingly useful ; and the above practice, if carefully carried out, will give good results.

After-treatment of reduced dislocations of the astragalus.—The foot should be kept at right angles on a well-padded splint, and spirit lotion constantly applied. The ice-bag should be used with caution, as the integrity of the skin is probably already severely tried. So soon as reduction of swelling will allow, the plaster dressing should always be employed, and in about six weeks passive motion may be commenced.

Subastragaloid dislocation of the foot. Causation and classification.—The confusion that has arisen between this displacement and dislocation of the astragalus renders their comparative frequency difficult to compute accurately. I would venture to question the statement usually laid down as to the great rarity of subastragaloid dislocations of the foot, and believe that these accidents are at least as common as dislocations of the astragalus pure and simple. The distinction is obvious and important. In subastragaloid dislocation the astragalus remains fixed in its box, the head of the bone maintaining its proper relation to the malleoli (Fig. 427). This dislocation is caused by the same class of injuries as before mentioned, and it seems a matter of trial of strength between groups of ligaments, as to whether the astragalus shall be thrown out of the ankle, or the os calcis displaced from under the astragalus. In *order of frequency*, these displacements may be (1) outwards or inwards, and at the same time backwards; (2) directly backwards; and lastly, (3) forwards, which is very rare. Subastragaloid dislocations are generally incomplete, and most commonly oblique. The direct displacement backwards is less frequent than dislocation backwards and outwards, or backwards and inwards.

Morbid anatomy.—In described cases the astragalo-scaphoid ligament has been found ruptured, and the lateral ligaments of the ankle also, to a variable extent. The interosseous ligament need not be entirely torn except in the complete displacements. The neck of the astragalus or one of the malleoli may be fractured. Blood-vessels, tendons, and nerves may be torn. The tendo Achillis is tense, drawing the calcaneum forcibly upwards, and one or other of the tibial tendons have been found tightly hitched against the neck of the astragalus. The tension of the tibial tendons, with tough ligamentous fibres torn and wrapped about the bones, is a powerful obstacle to reduction. The head of the astragalus may lie on the scaphoid, and project outwards or inwards; and the posterior border is often found tightly wedged in the groove between the articular facets of the calcaneum. In complete cases of displacement backwards, the head of the astragalus may rest on the cuneiform bones.

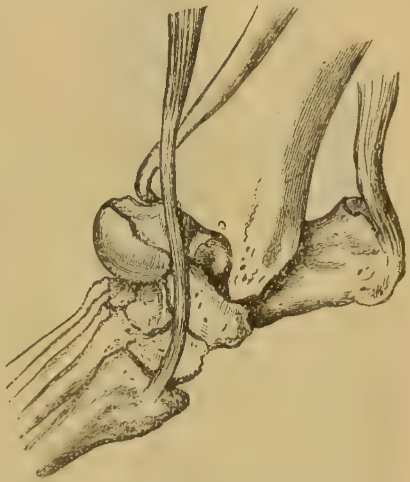

Fig. 427.—Subastragaloid Dislocation of the Foot backwards. (After Pick.)

Symptoms. — The literature of this dislocation makes it clear that various authors describe different dislocations under the same name; and the reader will do well, in perusing the following remarks, to remember especially that the astragalus retains its usual position to the bones of the leg, that there is a certain amount of movement in the ankle, and that any projection of the head of the astragalus is not due to a displacement of that bone, but to the rest of the foot from it.

Taking the symptoms of the oblique dislocation *backwards and outwards*, the foot will appear shortened dorsally, and the heel lengthened, while the tendo Achillis is pushed backwards. The foot is a little extended. The rounded prominence of the head of the astragalus will lie on the scaphoid or cuneiform bones, and project internally. The inner malleolus is also prominent, and reaches the sole. The foot is everted, so that its outer border looks upwards, and the external malleolus is buried, the appearance being that of extreme talipes valgus.

In the displacement *inwards and backwards* the head of the astragalus projects externally, and the foot is inverted, the external malleolus projecting nearly to the sole, and the inner being buried (Fig. 428). The position of the head of the astragalus may be looked upon as the key to the direction of these displacements. The more the foot is thrown directly backwards, the greater the elongation of the heel, and the more advanced the prominence of the astragalus dorsally. The tuberosity of the scaphoid will always be altered in position with regard to the head of the astragalus.

The *forward* subastragaloid dislocation is excessively rare, and the scope and aim of this article prevents further discussion of it. The malleoli are not so often fractured in this dislocation; but it may be compound, and when produced by machinery accidents is associated with much laceration and contusion of the soft parts.

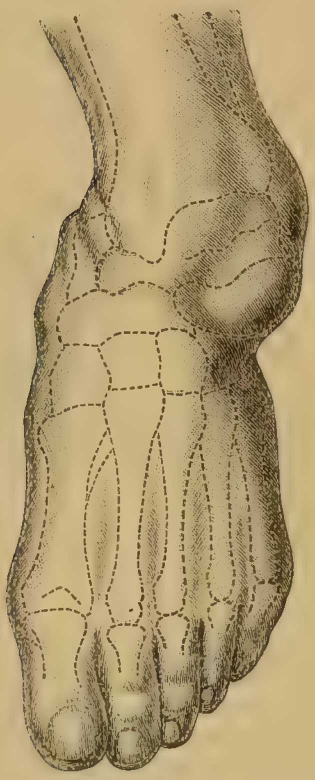
Fig. 428.—Subastragaloid Dislocation of the Foot inwards.

Treatment (Fig. 429).—Flexion of the leg and counter-extension from the thigh should be, as usual, effected. In those dislocations which have a backward direction, the operator may press the tibia backwards, and draw the front of the foot upwards and forwards, inverting or everting at the same time, according to the displacement; or the front of the foot may be powerfully drawn upon by a bandage, and the hand of the operator left free to manipulate and press upon the displaced calcaneum. Should great difficulty be experienced, the tendo Achillis and posterior tibial tendons will need division, after which reduction will generally be effected.

In *compound subastragaloid dislocations* it is advisable to remove the astragalus if it should be fractured, or if the soft parts are much injured, or there be difficulty in reduction. In the *irreducible simple dislocations* it has been generally advised that things should be left alone. The risk of sloughing of the skin over the prominent head of the astragalus seems very great, and the practice of modern surgery would tend towards excision of the astragalus or amputation, rather than leaving a painful distorted foot, which may entail serious permanent lameness. The subastragaloid amputation by a racket-shaped incision would be applicable to old irreducible subastragaloid dislocations, with distortion of the foot.

Dislocation at the medio-tarsal joint.—In the dislocation we are now considering, the astragalus and os calcis maintain their relation to each other, and to the ankle joint, the movements of which are free and unimpeded. The rest of the foot is displaced from its connection with these two bones. The displacements may be lateral or antero-posterior, and the diagnosis is made by observing the clubbed appearance of the foot, the projections of the displaced bones, and the position of the lesion. Reduction is to be effected by flexion of the leg, and extension of the front of the foot, with rotatory movements.

Displacements of individual tarsal bones or of the metatarsus or phalanges. — Dislocation of the os calcis alone is a rare accident, fracture being far more common. The probable symptoms will be a prominence of the bone laterally with a hollow on the opposite side, while the astragalus maintains its proper relation to the scaphoid. Any of the other tarsal bones may be completely or partially displaced by crushes or wrenches. The displacement is generally dorsal. Fracture is likewise found, and the displacements are often compound. The symptoms are plain from the position and relations of the displaced bone. In compound injuries, and if reduction be impossible in complete dislocations, resection should be practised.

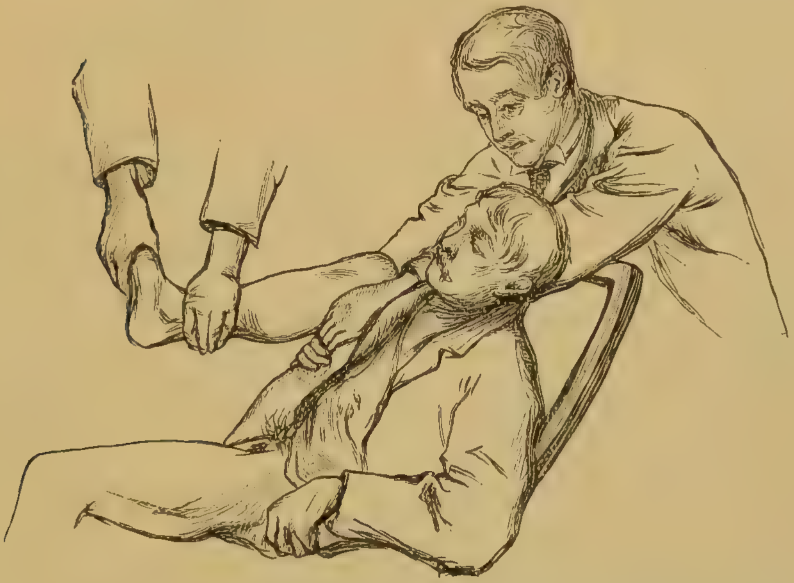

Fig. 429.—Pressing the Tibia backwards and pulling upon the Foot, in subastragaloid Dislocation backwards.

Any of the metatarsal bones, collectively or singly, may be displaced from the tarsus forwards, backwards, or laterally. The shortening and projections will indicate the nature of the injury. The internal cuneiform, with the metatarsal bones, may be dislocated (R. Smith).

The displacements of the phalanges resemble those of the upper extremity. The same difficulty has been found in the reduction of the first phalanx of the great toe as is encountered in the thumb. There is great practical difficulty in making extension upon a single metatarsal bone or toe phalanx, and I am inclined to think that the open method, by re-section, or the division of opposing ligaments, would be applicable to obstinate and inconvenient cases of these displacements.

XXXIII. DISEASES OF JOINTS.

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In studying the diseases of joints, it is necessary here to assume that their general anatomy is fully understood, and need not therefore be further alluded to. The details of the histology of the several tissues of the articulations must also be left untouched. But it must not be forgotten that their minuter structure has the most important bearing upon the pathological changes affecting the various component parts of the articulations. The synovial membrane, bones, cartilage, and ligaments behave quite differently towards the same morbid influence, whether it be traumatic, directly infective, or generally constitutional. This is only what might be expected when we consider that the synovial membrane is physiologically very active, the bones on the immediate aspect of joints are less so, while the cartilages and ligaments play an almost completely passive part in the economy. For it appears abundantly clear, from what we observe in all parts of the body, that where physiological activity is greatest there is to be seen the greatest vulnerability to disease of an active type. This rule is also observed to hold good in the age of tissues—those which are youngest, or, in other words, most energetic in growth, being most liable to active disease, while those which are more mature, or more highly differentiated, have the greatest power of resistance to morbid influences. We see, therefore, that, *ceteris paribus*, children are more liable to disease of the joints than adults, just as those tissues of joints are more vulnerable which possess the least highly differentiated structure. This law appears to be related in some way to the vascularity of the tissues, those being most liable to active disease in which the amount of blood is greatest, and *vice versâ.*

It is necessary to keep these points in mind, both on pathological and clinical grounds.

The actual consideration of the diseases affecting the joints of the body naturally divides itself into :—

(1) The study of the general pathology, symptoms, diagnosis, treatment, and prognosis of those morbid conditions met with

among the structures which enter into the formation of all the articulations.

(2) Their special clinical features as seen in particular joints, together with their diagnosis, symptoms, treatment, and prognosis as local affections.

GENERAL PATHOLOGY OF JOINT DISEASES.

Classification.—The morbid changes found here are, in the vast majority of cases, but the expression of one or other form of *local inflammation.*

But there are, besides, several diseased conditions due to *general degenerative* changes, either in the tissues of the joints themselves, or of more remote parts of the system, such as the nervous centres and vascular system.

Finally, the tissues of joints are occasionally the seat of *neoplasms* in the usual acceptation of the term.

Besides these morbid changes there are, of course, others the direct result of *violence,* such as dislocations, lacerations, gunshot wounds, and the like, but these are treated of in the article on INJURIES OF JOINTS, and will only be alluded to here so far as they give rise to the remote effects coming more naturally under the name of disease.

We have then before us three great groups of joint diseases—(*a*) *the inflammatory;* (*b*) *the degenerative;* (*c*) *the neoplastic.*

But, though practically convenient for the present, such a classification will probably require modification in the future, at all events as regards the diseases included in each group. For it is open to question under which of these headings some of the commonest joint affections ought to be placed, which have hitherto had their definite positions assigned to them. For instance, tuberculous disease has hitherto been placed among the inflammatory affections, but it might just as well be included among the neoplasms, to which it bears in many respects a very close resemblance, and there would be some practical advantages from such a nomenclature. And if it be proved, as appears likely to be the case before long, that what we now call new growths are masses of hyperplastic or heteroplastic tissue, produced under the irritation of living parasites, just as so-called tubercles are, and in the same way, the distinction between neoplasms and inflammatory products will be hard to draw.

Adopting, however, this classification for the present, we may proceed to consider the *inflammatory affections* of joints, first as they affect the synovial membrane, and then the ligaments, cartilages, and bones.

This anatomical arrangement is necessary, inasmuch as the various morbid processes behave, as has been already said, in a somewhat different manner in each of the different structures which enter into the formation of joints.

Taking all the primary affections of joints as a whole, it may be safely asserted that the synovial membrane is by far the most frequently diseased, the bones next, and the ligaments next, while the cartilages are very rarely the seat of primary change. This comparative liability to disease appears to depend upon the physiological activity of each structure, those tissues being most open to disease which are most active in their cell life, while those in which the latter is sluggish seem to enjoy comparative immunity. whether from acute inflammatory change or from heteroplastic new growths. This was pointed out as an axiom in pathology long ago by Virchow, and is a most important fact to bear in mind.

SIMPLE SYNOVITIS DUE TO CAUSES OPERATING FROM WITHOUT.

Inflammation of the synovial membrane, which is the first morbid process to be considered under our scheme, is a very common affection. It may be either *primary*, or *secondary*, either to other local primary disease, or to general conditions. It may, besides, be either *acute*, *subacute*, or *chronic*.

1. **Simple primary acute synovitis.** **Pathology.**—This affection as commonly met with is usually the result of over-use, injury, or cold, and in its simplest forms is best studied in relation to either of these exciting causes.

When a joint is violently overstrained or contused, or when it has been long exposed to cold and damp combined, the effect is usually to cause hyperæmia of the synovial membrane. This may vary from a transient flush to the most intense active congestion. The cause, pathologically expressed, is to be found in a more or less complete vasomotor paralysis affecting the smaller vessels of the synovial membrane. All the usual consequences of this acute congestion are soon seen in the form of increased heat, redness, tenderness, pain, and swelling. The increased heat is easily explained by the greater quantity of blood passing through the parts around than in the normal state. The redness is due to the same cause, as is also the hyperæsthesia. The swelling of the joint, on the other hand, is not only due to the increase in amount of blood in the vessels of the part, but also, in acute cases, to the exudation of serum from its vessels producing œdema, and also to hyper-secretion of synovium from the congested lining of the joint, or effusion of synovium.

Such a joint, if laid open, would exhibit the following changes:—The lining membrane would be somewhat swollen, deeply injected, its colour varying from light pink to deep purple, contrasting with the pearly-white, pale cartilage. It might also be thrown into folds if much swollen. At the same time, the capsule and parts around would also be found to be very vascular, and deeper in colour than normal. The synovial fluid in the joint in such cases is, as already remarked, increased in amount, and may be otherwise normal or thinned by the admixture of effused serum, or turbid from the presence of leucocytes and lymph. The latter condition, however, is rarely seen in simple cases. There is no sharp boundary line between each of these conditions, they run one into the other insensibly, according to the intensity of the hyperæmia.

The **symptoms** in such a simple acute case will be, subjectively, pain on movement or handling, a sense of heat in the joint, and perhaps some throbbing, with, in some cases, a little general pyrexia. Objectively, there will be general swelling, fluctuation of fluid in the cavity, also redness and altered position, due to the necessity of relaxing certain parts of the inflamed and tender capsule.

The fluctuation in these cases will always be best seen on those aspects of the joint at which the capsule is thinnest. Thus, in the knee it will be observed on either side of the joint in front, and in the elbow on either side behind.

In this state, if uncomplicated, the pressure of the cartilages one against the other will cause no pain, but any friction of the synovial membrane upon subjacent parts is acutely painful. This is a very important fact to bear in mind when endeavouring to determine whether the encrusting cartilages are involved or not.

The **diagnosis,** except for the last point, is not difficult, and is based upon the foregoing factors.

The rapid onset of the swelling in the joint after the operation of one or other of the exciting causes mentioned above distinguishes it from any of the *malignant tumours*, as would also in most cases the early increased heat, redness, and tenderness to the touch. The latter symptoms also serve to contrast it with the *subacute* and *chronic forms* of synovitis. It is, perhaps, more likely to be confounded with hæmarthrus or effusion of blood into the joint than with anything else where there has been a history of injury as the exciting cause. But in *hæmarthrus* the pouches of the joint are distended with blood within an hour or so of the receipt of the injury, while even the most acute simple synovitis from violence does not follow the latter for some hours, and its effusion is much slower. When blood is present in the joint, too, the heat, redness, and swelling are not so marked as in acute synovitis. And, moreover, after it has undergone coagulation the clot will give the usual tearing crepitation on palpation, which is characteristic, while in synovitis the fluctuation is soft or elastic alone. In many cases, too, of hæmarthrus the tint of the skin round the joint helps a differential diagnosis. To the eye at a little distance it is slightly darkened in hue by the underlying mass of blood, while otherwise normal. Of course, if any doubt still existed after consideration of all these points, the introduction of a sterilised aspirator needle would at once clear up the difficulty. This needle should be large enough to withdraw some of the clot if coagulation has taken place, otherwise the mistake of supposing there is no fluid in the joint may be made.

The **general treatment** of such a condition is based upon ordinary principles, and is for the most part simple. In the first place comes, as a matter of course, rest for the affected tissue specially, and for the system generally, so that all vascular excitement may be avoided. We may also do a good deal constitutionally by the use of saline purges, with a little antimony in sthenic cases to relieve vascular tension, and remove effete products from the blood, which might lower the vitality of the tissues and render them more vulnerable to morbid influences. In asthenic cases, on the other hand, we must be cautious with purgatives, and must remember that tonics and stimulants are indicated, and that where there is much pain, preventing sleep and interfering with the appetite, anodynes and sedatives are given with advantage.

The **local treatment** comes next in the form of blood-letting, either by means of leeches to the part, or wet cupping where the patient is strong enough to bear the loss of blood. Then, or in place of these measures, the application of cold by means of the ice-bag or evaporating lotions is of the greatest service. Where these means fail to check the inflammatory process, and the latter is running high, hot fomentations, with or without belladonna, will be found of the greatest use. In many cases, too, where there is much tension from effusion, aspiration of the joint with a sterilised hollow needle is the readiest way of giving relief. And this should be done before the capsule and ligaments have been overstretched, and the synovial membrane permanently altered. Carefully performed, this operation can do no harm, and may save much time to the patient and loss of function to the joint.

Prognosis.—Simple cases treated on one or other of these lines usually do well, and the prognosis is favourable. The active congestion becomes less and less, and with it the effusion of serum into the parts around, and the hyper-secretion of serum. With the disappearance of this swelling and tension all pain ceases, and local and general fever subside. As a rule, the effused fluid is soon absorbed, and a complete *restitutio ad integrum* takes place. In a few cases, however, in debilitated individuals absorption is slow, especially from the joint cavity, and the capsule may be much weakened by the distension if prolonged. This condition—known as *hydrops articuli*—is, however, more likely to follow the subacute and chronic affections than the acute, and particularly among those who have been living under defective hygienic conditions, and whose blood may consequently contain organised impurities. If it resist the treatment indicated above, and the tension threatens permanently to stretch and weaken the capsule, it may be well to drain off the fluid from the joint by aspiration, every care in the direction of asepsis being taken. If there be any suspicion of the inflammation of the cavity being kept up by more or less septic matter derived from the blood, it may be well to supplement this aspiration by washing out the joint with some non-irritating antiseptic fluid, such as boric lotion, or even sterilised water, or the part may be opened by lateral incisions, and drained for a time with benefit. This, if carefully done, is usually followed by complete restoration of function. But where the measure has been too long delayed the synovial membrane has often become thickened and shrunken, and has lost its elasticity and smooth surface, and the subsequent movements of the articulation are impaired. It is well, therefore, not to delay aspiration or incision too long. Much of this stiffness may be removed after all inflammation has subsided by regular massage and slightly-forced passive movements. Active movement, too, should not be put off too long, but should be gently and cautiously employed.

It is very rare for such a simple attack of acute synovitis, due to injury, cold, or wet, to terminate in actual *suppuration*, but it is possible in individuals who are in bad health, or who have been living under vicious hygienic conditions. In this case there is probably a septic element in operation, the local injury only determining the spot at which this septic matter in the blood shall find a suitable soil for its development. If such suppuration do take place as indicated by rigors, marked œdema, and redness of the part, and very high body temperature, the sooner the joint is freely opened, drained, and washed out, the better. The incisions should be made in the axis of the limb on both sides, towards the back part of the lateral pockets, and should be tolerably free. The pus should then be washed out with a stream of sterilised water, or 1 in 1,000 solution of perchloride of mercury, 1 in 20 solution of carbolic acid, and should then be drained with a tube on both sides. Such joints thus treated may recover their functions completely if the operation has been done before the cartilages and other surfaces have been extensively ulcerated or covered with organised lymph.

2. **Simple subacute synovitis.**—Here the *causes* operating are often the same, and differ only in degree. The vascular excitement is less, and therefore no redness of the skin, very little swelling of the soft parts, and but slight effusion of synovium into the joint are present. The fluid in such a case is clear and thin, and contains few or no corpuscular elements. Pain is present in no marked degree, and there is but little alteration in the position of the joint, which may be handled without giving rise to any suffering. The chief complaint of the patient is that the joint feels "stiff" and "weak."

Treatment.—These subacute cases are treated on the same general principles as the acute, but with less vigour. They require, however, close attention, for if neglected the affection is apt to become chronic, and is then very troublesome. In the initial stage leeches and the application of cold are advisable, and when all heat has disappeared the part must be blistered freely. Of course, absolute rest is secured by splints suited to the joint, or by position when the patient is confined to bed. The chief difficulty in these cases, as a rule, is the removal of the fluid from the joint, and the prevention of its re-accumulation. For, even without any return of inflammation, the tendency in these cases is towards re-secretion of superabundant synovium. Either the vascularity of the synovial membrane remains slightly above normal, or its absorbents are in some way interfered with, and the balance between secretion and absorption is thus upset. Aspiration in such cases may be repeated several times, and yet without avail; the joint, though painless, refills each time. Here we will find much benefit from repeated blistering at short intervals, with firm elastic pressure. The blisters should be applied in constant succession on different aspects of the joint every alternate day or so, and the pressure may be kept up by bandages applied over thick wool laid upon the blisters. Later, if the fluid is completely removed, or almost so, the joint may be strapped with plaster in the usual way as soon as the blistered surfaces are healed. This will promote absorption of any remaining fluid, and will often prevent its return when the patient is beginning to use the affected part.

If these means fail, it may be desirable to aspirate and wash out the cavity with some mild antiseptic, such as $2\frac{1}{2}$ per cent. of carbolic acid, or a weak iodine solution. This is frequently perfectly successful where other milder means have been ineffectual; but it is not clear exactly why, unless there be irritant products in the synovial fluid which is evacuated, but of this there is no evidence at present in ordinary subacute synovitis following injury or cold.

In many cases great benefit is derived from massage regularly applied. Fluid is by this means removed, and the stiffness of the joint overcome. Patients having had subacute attacks of synovitis should be careful in all cases to wear over the joint, for some weeks or months after all swelling has disappeared, a covering of elastic webbing, and should guard the part against overstrain or exposure to cold or wet, which is very likely to bring on the trouble again.

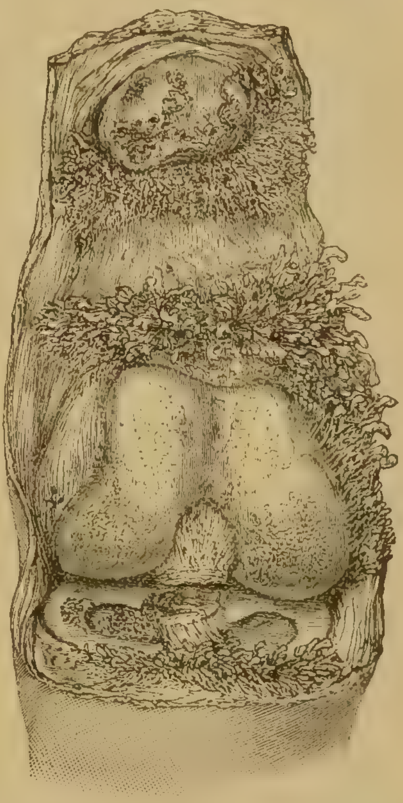

Fig. 430.—Pendulous Folds of Synovial Membrane, due to chronic Inflammation. (University College Museum.)

3. **Simple chronic synovitis.**—This form differs but little from the subacute, except in degree. The same effects are produced by similar causes. But, owing to some peculiarity in the patient, usually a defective vitality, the effects last longer, and as a consequence are somewhat modified. Again, want of rest may convert a subacute into a chronic case. Thus, though the

hyperæmia and the fluid accumulation differ in few respects from what is observed in the subacute form of the disease, the persistence of the congestion of the synovial membrane for long periods leads to its plastic thickening. It thus becomes altered in consistence, being voluminous and often irregularly hypertrophied in all its parts. When its borders are particularly affected, pendulous masses or folds are often formed within it, which hang loose about the sides of the joint (Fig. 430). These may give rise to considerable modification of the movements of the articulation, and may cause a sense of weakness in the latter, or, if the pedicle of such a pendulous mass be torn through during movement, a loose body is produced, with many serious consequences. The ligamentous structures of the joint are often affected at the same time in various ways, to be alluded to presently.

The **symptoms** of simple chronic synovitis have much in common with those of the subacute variety, and it would not be necessary to consider the two forms apart were it not for the fact that the plastic changes produced in chronic inflammation of the synovial membrane seriously interfere in many cases with the functions of the joint if neglected.

The **treatment,** too, up to a certain point, is the same in both affections. But in addition, we have to endeavour to help in the absorption of the plastic matter, or actually to remove it. In the first instance we make use of strapping, with friction combined, with painting of iodine over the part, or inunction of mercurial ointment. Rest during this treatment is desirable, and later, cautious passive movement. But for a long time after the patient is able to get about it is well to keep the part enveloped in an elastic bandage, so as to prevent that passive congestion and swelling which is so apt to reappear on resumption of active movement.

But sometimes in simple chronic synovitis all our efforts to restore the normal conditions by ordinary methods fail completely; the joint remains weak, painful, and swollen. In such cases I have had the happiest results from freely opening the joints on their lateral aspects, and excising as much of the hypertrophied synovial membrane as could be reached from the wounds. The latter must be made carefully with due regard to the integrity of the lateral and patellar ligaments. The tissue thus removed has been found by the writer and others to be hyperæmic and fibroid, without any trace of tuberculous material, being simply affected by chronic hyperplasia. It is hard to account for the marked improvement which follows in such cases; but it has been proved by many surgeons, at home and abroad. And when we have had a larger experience of the treatment we shall accumulate data upon which to found a theory as to the *modus operandi* of the method. All this should be followed by massage and douching, with change of air, good diet, tonics, and sea-bathing if possible.

SYNOVITIS DUE TO CAUSES OPERATING THROUGH THE BLOOD.

Classification.—The three forms of simple synovitis—the acute, the subacute and the chronic—produced by causes operating *from without*, may be taken as types of inflammatory change in the synovial membrane of joints.

But there are other forms of disease of the same structure, due to causes operating *from within*, which remain for our consideration.

Among these may be mentioned those inflammations set up by poisons carried to the synovial membrane through the blood, which irritate the former. Of these there are many, such as the *pyæmic*, *gonorrhœal*, *puerperal*, *scarlatinal*, *acute rheumatic*, and the affections of joints which follow in many cases on *small-pox*, *typhoid fever*, *dysentery*, and *renal disease*, together with the synovitis of *tubercle*, *syphilis*, and *gout*.

We will do well however, at all events for the present, to group a number of the above affections under one heading, and speak of them simply as the *septic inflammations of the synovial membrane.* For as yet it is hardly possible to draw any distinction as to ætiology between the inflammations produced in joints by the septic matter introduced into the blood from definite putrid wounds on the surface, and those caused by toxic agents reaching the circulation through other less evident centres of infection. The effects, at all events, are practically the same; and it seems probable that, when in the course of puerperal or typhoid fever, of scarlatina, gonorrhœa, acute rheumatism, dysentery, and small-pox, the synovial membrane inflames, it is again merely a question of infection of the system generally from some raw or ulcerating surface, be it in the throat, urethra, intestine, or uterus. The specific poisons in each of these cases, if not identical, have, nevertheless, so much in common that we may put them in the same group, and speak of them as the septic irritants of the synovial membranes.

Probably, if not certainly, the poison operating in many of these affections is a mixed one, consisting of the toxic products of several forms of fission fungi. This is the case, we know, in gonorrhœal synovitis, in which the specific organism of the urethral catarrh is, so far as we know, aërobic, and not propagated in the blood, or otherwise than on mucous surfaces exposed to air. And yet gonorrhœa is frequently followed by severe pyæmic affections of joints, with the production in them of several forms of pathogenic organisms.

The same may probably be said of the synovitis which follows scarlatina, typhoid, dysentery, and other similar disorders.

In tubercle, on the other hand, we have, as a rule, a pure unmixed affection, as also in syphilis.

In gout, again, we have a definite specific poison, in the form of a chemical irritant—urate of soda. And the mode in which this acts in setting up synovitis throws considerable light on the other processes in which the irritant cannot be so easily isolated.

The long list, then, given above may now be reduced to (1) the *synovitis of septic infection*, (2) *of syphilis*, (3) *of tubercle*, and (4) *of gout*.

Another point strikes the observer at once in looking over the above list of synovial diseases, namely, that they naturally arrange themselves into *three* groups, according to the processes which underlie the grosser changes present. One group, including the above acute and subacute septic affections, is generally characterised by the presence of *active* hyperæmia, rapid effusion, and cell-proliferation; the next, made up of the tuberculous and syphilitic, by a more *passive* hyperæmia and more sluggish cell-proliferation; the last, including the gouty and so-called chronic rheumatic inflammations, rather by *degenerative* changes, accompanied or followed by passive congestions of usually moderate intensity, and not marked primarily by either excessive effusion or cell-proliferation. The varieties of disease in each of these groups display a close parallelism in their natural history. For instance, it requires minute scrutiny to detect differences between chronic pulpy degeneration of the synovial membrane due to tubercle, and the analogous changes of structure in the synovial membrane brought about by syphilis. The same may be said of the other affections belonging to the acute and subacute pyæmic class, while between the primary degenerative changes of gout and chronic rheumatic arthritis there often seems to be a close relation.

It is not intended to imply here that in chronic rheumatic arthritis the synovial membrane is often primarily affected. Probably this is rare, the changes in the synovial membrane being in most cases secondary to degenerative disease starting in the cartilages and ligaments. This disease appears to hold an intermediate position between gout, in which the cause of the lowered vitality is the presence of a definite chemical compound in the tissues, and Charcot's disease, in which, be the original cause what it may, the proximal cause appears to be a profound affection of the nervous centres manifesting itself in the osseous system by degenerative changes due to defective innervation.

These varieties of synovitis, due to causes operating through the blood, may now be considered.

1. **Acute septic synovitis.** **Pathology.**—This form of synovitis, as already stated (page 1039), is met with in the course of many local and constitutional diseases, hitherto regarded as quite distinct. But it is almost certain that the poison which produces the joint trouble is analogous if not identical in all, and is due to the presence in the blood of one or other of the well-known forms of staphylococci, which have actually been found in the joint effusions in such cases. It is easy to understand the mode of entrance of these into the system when we remember the foul suppurative surfaces present in different parts of the body in the diseases in question—in the urethra in gonorrhœal synovitis, in the throat and ear in scarlet fever, in the intestine in typhoid, in the uterus in puerperal fever, and so on.

Once introduced into the circulation, the capacity of these fungi to increase within the system is well known, until the whole organism may be overpowered by the poison generated by them.

The first evidence of a dangerous general intoxication with the latter is usually a sudden rise of temperature, followed by a rigor, and then abrupt defervescence. It is after this that we usually meet with the septic joint affections in the form of simple synovial, serous, or purulent effusion into the cavity of the articulations.

This synovitis is brought about by the irritating effect of the organisms or their products upon the capillaries of the membrane, in which colonies of staphylococci may be entangled. Hyperæmia follows, and, as a consequence of this, increased secretion takes place from the surface of the sac, and the joint is distended. In the milder cases the fluid poured out may be but slightly altered synovium, and may be soon absorbed again. This, however, is rare, the effusion in clearly septic cases being usually either turbid serum or actual pus. The reason for this is not far to seek. Simple hypersecretion from a synovial membrane invaded by septic irritants would naturally carry a certain number of the organisms into the joint. As there is no means of escape from the latter for the fluid except by the absorbents (probably also damaged in their functions by the same inflammation, and, at all events, inadequate to the extra work thrown upon them), an accumulation of septic matter takes place in the joint, and this re-acting upon the surfaces around, intensifies the hyperæmia, and soon leads to suppuration in most cases.

It would seem, too, as if the organisms in the vitiated synovium in such cases had the power of directly attacking the tissues, and also of preventing any of that organisation of fibrin which is so frequently seen to be present in the neighbourhood of non-septic inflammations. This is indicated by the peculiar appearance of the synovial membrane and the cartilages in such cases. These seem to the naked eye as though they had been subjected to some digesting process, which has liquefied them, and at the same time left them clean and smooth, and without a trace of lymph upon them. In many cases the injection of the synovial membrane appears after death to be but slight, and out of all proportion to the large amount of pus in the joint.

A microscopical examination of the membrane, however, will show exudation of small round cells in its most superficial layers, which are thus softened and sodden on the surface, and perfectly destitute of any trace of plastic material.

The cartilage, too, is seen under the microscope to be altered, in its superficial layers at all events. Here the tissue is softened and rapidly liquefying, as though under the action of a chemical solvent, but, as a rule, so evenly and uniformly, that only close scrutiny detects the thinning of any particular part. The capsules near the surface are packed with small round cells, but those in the deeper part of the cartilaginous crust are normal. Indeed, the whole appearance of the tissue gives the impression that so rapid and universal was the solvent action of the secretion in the joint upon the cartilage, that it had but little time to develop any tissue change in its deeper parts.

Symptoms.—The clinical features of acute septic synovitis, as contrasted with the simpler forms, are easy to draw. Not only have we the general condition to guide us, but we have the fact that frequently more than one joint is affected spontaneously and simultaneously, and without any local external exciting cause. Not uncommonly, too, effusion appears in one joint and then disappears, while another joint is becoming acutely affected. This in its turn may recover, while a third, fourth, or fifth may become successively involved in the same process. Some of these joints may completely recover, while others in the same individual may go on to the most destructive suppuration.

Again, the swellings in the joints are often accompanied by circumscribed dusky flushes of the skin, which contrast strongly with the faded clay-like colour of the general surface of the body in septic infection. These somewhat livid blushes come and go, and are, besides, quite unlike the diffused bright flush of simple synovitis.

In very severe cases the effusion tends to rupture the synovial pouch very early, and to diffuse itself freely among the surrounding tissues, setting up in them, too, the same suppurative processes. The fact that the toxic agent acts as a solvent upon any fresh plastic matter which otherwise would tend to limit the process, explains the rapid destructive changes produced in this form of disease.

The subjective symptoms are the same as those accompanying the simple form of synovitis, running on to suppuration (page 1036); but the constitutional condition is quite different. There is the high but oscillating temperature, the rigors, delirium, restlessness, great wasting, and often vomiting; in fact, the symptoms are more or less of marked general pyæmia. (*See* PYÆMIA, page 183.)

Treatment.—The treatment locally is carried out on the same principles which guide us in dealing with simple acute synovitis of the suppurative form; but we must remember that early evacuation of the septic effusion into the joint is here more than ever of importance. Here, too, the washing out of the cavity is more pressingly called for than when the pus is not septic, and the fluid employed may be more strongly antiseptic. At the same time, it should be borne in mind that the mechanical flushing effect of the fluid passing through the joint is probably of more importance than its germicide properties. Indeed, more will be achieved by using a large quantity of simply sterilised water to carry away every trace of the irritating pus from the joint than by using a limited quantity of a strong bactericidal solution, and the prognosis as to movement in the articulation of those who recover will be better. For there can be no doubt that germicides strong enough to destroy bacteria in the joints, and in the tissues from which they are derived, must also be strong enough to injure the vitality of the superficial cells of the joint surfaces upon which we rely for the repair of the lesion.

The **prognosis** in these cases of acute septic synovitis is very unfavourable, both as regards the life of the patient and the functions of the part if he survive. When septic matter is present in the blood in sufficient amount to cause suppuration in the joints, it is only too likely to be abundant enough to overwhelm the vital powers completely. And yet every surgeon of experience has seen cases in which, after free opening and evacuation of such purulent collections in joints, the patient has survived, and, moreover, sometimes with the movement of the joint but little impaired.

The **subacute form of septic synovitis** differs from that just described only in the degree of inflammation induced in the joint. Either the poison is not so virulent, or it is not in such large amount, or the vitality of the patient is greater.

The *symptoms* are also the same, though less marked, and the constitutional disturbance is less severe.

Here the temperature is not so high, and does not oscillate so much, and rigors are few or entirely absent. The pain, however, in these subacute cases is often more severe than in the acute, and the tendency to affect several joints one after the other is greater. The possibility of the smaller and deeper synovial tracts being attacked must never be lost sight of in this form. Such affections may often be observed, for instance, in deep tendon-sheaths and the smaller joints of the ribs, or wrist and ankle, or, again, in synovial bursæ.

The *treatment* in this case need not be so radical in the first instance. Here we may trust to hot fomentations, combined with belladonna paint for a time, with complete immobilisation of the limb. But if these means fail there is no choice, the joint must be opened, washed out, and in some cases drained.

In such cases the *prognosis* is favourable, and the functions of the part are often completely restored if treated early enough.

In the **chronic form of septic synovitis** we have a very troublesome affection, if not actually very dangerous to life. Here there may be only a very moderate amount of effusion into the joint, but the inflammation lingers so long, that grave secondary structural changes are induced, which frequently destroy all the functions of the part in the end. The synovial membrane becomes thickened and fibrous, and the cartilages roughened, while the ligaments are softened at first, and then shrink and contract, so that great deformity is frequently the result if the inflammatory process be recovered from.

The *treatment* in such cases is the same as for the subacute form in the main; but we must not forget that the longer such a process lasts the graver will be the structural lesions. We must not therefore hesitate, where other means fail, to open and wash out these joints, and carry out free drainage. And at the same time we must be particularly careful to support the joint in the best position when its ligaments are softening, so as, on the one hand, to prevent its component bones from being dislocated one upon the other; and, on the other, to secure that, if it should become stiff, it shall anchylose in the most favourable position for future use.

When the part recovers with only fibrous thickening, much may be done to bring back its movements by massage, combined with passive motion and cold douches. These measures, patiently continued for long periods, are productive of the greatest benefit. Forcible movement, on the other hand, under an anæsthetic is to be deprecated where the disease has been very chronic. If this be done, the disease may be set alight again, or the fused surfaces, being torn apart, will only unite again, or, finally, the atrophied bones may be broken by the force applied.

(2) **Syphilitic and** (3) **tuberculous synovial disease.**—The group of synovial affections, characterised in the main by more passive hyperæmia and sluggish cell proliferation, must now engage our attention. This will include syphilitic and tuberculous disease.

2. **Syphilitic synovial disease.**—It is necessary to remark here, however, that in this group is not included that form of subacute synovitis often noticed early in the *secondary stage* of syphilis, which differs but little from, if it be not identical with, what we have been considering under the heading of subacute septic synovitis. The congestion and hyper-secretion here seen in the joints are analogous to what is often seen at the same time in the mucous membrane of the throat, larynx, and bronchi at the outset of the secondary sequelæ, which last for a short time, and then disappear spontaneously, but only to be followed soon by the more obvious cutaneous syphilides. The synovitis here is probably either due to ordinary septic infection, produced at the same time that the specific syphilitic poison is grafted on the system, or to the irritation of the syphilitic poison, as yet not sufficiently developed to produce those plastic changes familiar to us in the later secondary and tertiary stages. It is probably this milder form of synovial affection without marked effusion which gives rise to those so-called "prodromal pains" in the joints and deeper parts of the back, so often met with at the outset of syphilitic infection. Possibly the joints of the spine are the seat of some of them, as well as those larger articulations which we can see, and which are also the seat of these pains. (*See also* pages 430 and 1061.)

Pathology.—The syphilitic synovial disease, which is here contrasted with tuberculous disease, is not like that just alluded to. It is a slow hyperplastic change in the synovial membrane, which comes on late in the course of the constitutional affection, whether it be acquired or congenital. In the latter form it is quite as common as in the former, if not more so. All the components of the joint may be affected, but the change is generally seen in the soft parts and, above all, in the synovial tissues.

The change consists in a small-celled infiltration—in other words, in a diffused gummatous formation. To the naked eye the latter has a close resemblance to the granulation tissue of tuberculous disease. When the cells are aggregated in masses, we have before us a true gumma. But, as a rule, the infiltration is diffused evenly, like ordinary chronic inflammation, and not in "systems" like tubercle, though there is only a sluggish so-called passive hyperæmia present.

Thus it may remain for an indefinite time if not relieved by treatment, or may undergo further secondary changes. The latter are quite similar to those seen in tubercular granulation. It may *resolve* or may undergo *fibroid organisation*, or may *liquefy*, *caseate*, or *cretify*. Thus, as we shall see presently, there is, in both syphilis and tubercle, either (1) a return to a healthy state, with or without permanent thickening; or (2) liquefaction, larger or smaller collections of pulpy broken-down material forming, and, perhaps, opening through the skin or into the joint; or (3) the granulation material may caseate, owing to fatty degeneration; or (4) it may dry up and cretify. Sometimes this deposit is associated with effusion into the joint, but this is not common.

These tertiary syphilitic deposits in and about joints have long been recognised clinically, but Lanceraux was one of the first to demonstrate them in the *post-mortem* room. In his interesting description the clearest proof is given that the tertiary gummata or inflammatory deposits in the articular structures present the characters and undergo the same changes as in the other parts of the body.

Symptoms.—The *subjective symptoms* of this late syphilitic affection of the synovial membrane are not usually well marked in the early stages of the affection. The change may have advanced far before much pain, weakness, or stiffness are complained of; and a patient will often continue to use the part when these factors are present, which, were the disease tuberculous, would indicate very extensive and disabling disorganisation, and be accompanied by considerable suffering and loss of power.

The *objective symptoms* are: swelling generally, far less uniform than in the tuberculous affection, and possibly localised at one or two clearly defined spots. These will have a peculiarly elastic or doughy feel to the touch, according to the condition of the focus. If consisting of fresh granulation tissue it will be elastic; if of softening gumma, doughy; if liquefied, of course, fluctuating. The temperature of the part is not raised appreciably, and the surface of the joint shows no signs of congestion, unless a gummatous deposit be breaking down on its way to the surface. Then the skin will gradually assume a brownish or more or less purple colour, which contrasts markedly with the pale earthy tint so frequently seen in late syphilis.

When other parts of the body exhibit unmistakable evidence of syphilitic disease—such as old circular scars, "pegged teeth," or interstitial keratitis—there can be but little difficulty in the diagnosis.

Of the **treatment** of this affection it may be fairly said that it is much more satisfactory, as a rule, than is that for tuberculous synovitis, unless it has been neglected and allowed to advance to an extreme degree of cachexia before being taken in hand. In younger individuals suffering chiefly from the congenital variety, our chief reliance will be upon mercury given in small doses for extended periods. But in addition it must be remembered that, as want of vitality lies at the root of the advance of the disease, so we must improve the patient's general health in every way in our power by nourishing food, fresh air, cod-liver oil, and tonics. In some cases where the disease is not far advanced the use of the joint in moderation need not be prohibited, and limited movement will probably be beneficial. Among the more aged and cachectic, iodide of potassium or ammonium and iron will take the place of mercury. In both massage and douches will be of the greatest use, as tending to improve the vitality of the tissues and promote absorption. Where resolution takes place the surface of the joint will be marked by pits and contractions, corresponding to the foci of granulation tissue, which have shrunken and organised or degenerated.

3 **Tuberculous synovial disease.** **Pathology.**—The next pathological condition on our list is the tuberculous affection of the synovial membrane. And in considering this disease it will be necessary to assume that the general pathology of tubercle is understood. (*See* page 340.)

It is essentially a disease of the two extremes of life. In other words, it is met with at a time when the general vitality of the body is feeble, and when the tissues are particularly vulnerable to injurious influences, operating either from within the body or from without. Again, it is essentially a disease of the poorer classes, who live under the depressing influences of mal-hygiene, although it may be met with among those who live in the midst of luxury, and are consequently debilitated in other ways, or perhaps, strongly predisposed by heredity.

But wherever and whenever met with, it is always due to the presence of a specific poison generated by an organism, to which the name of bacillus tuberculosis has been given.

Tuberculous synovitis is simply an inflammation set up in the synovial membrane by the presence of this particular organism. This is brought to the part in the vast majority of cases with the blood, although in some exceptional cases the joint is directly infected from without. But for a general tuberculous infection of the synovial membrane, something else is usually necessary in addition to the mere presence of the organism. There is reason to believe that in many individuals who are tuberculous the bacilli or their spores circulate freely in the body with the blood, and yet these patients do not necessarily get tuberculous joint affections. As a rule, some local influence which lowers the vitality of the particular structure affected is necessary in order to render it vulnerable to the attacks of the bacillus. Such influences are met with in injuries such as blows or strains, and in cold and wet. These may act in three ways, at least, in favouring the growth of the parasite. In the first place, an injury may directly lower the vital power of the part, and in the next it may secondarily injure its nutrition by inducing active or passive hyperæmia. Finally, with the increase of blood in the part we can imagine a greater amount of the tuberculous poison to be carried to the region involved, ready to settle upon the most damaged spot.

As a matter of fact, there is in many cases of tuberculous synovitis such a history of a local external determining cause as well as of a hereditary disposition and often of general infection indicated by evidence of the same disease in other parts, such as the lymphatic glands or lungs, though, of course, it is a common experience to meet with cases in which the joint affection appears to be primary and unassociated with any other demonstrable affection of the same nature.

The changes produced in the synovial membrane by the development in it of tubercle vary with the rapidity of the growth of the latter and the stage at which it has arrived. When rapid and extensive invasion takes place, there is considerable active hyperæmia in the synovial membrane and external swelling. This, however, is comparatively rare, but is met with in those uncommon cases in which direct infection of a joint with tuberculous matter from without has taken place. When, on the other hand, the invasion is limited and slow, the joint shows but little of the ordinary signs of inflammation, either externally or on being laid open. But if the immigration of the bacilli be but sparse, and the vital resistance of the tissues be considerable, the most chronic of all the forms of synovitis is produced, in which almost all the clinical signs of inflammation, except swelling, are in abeyance If such a joint be laid open, as is often the case, little or no active hyperæmia is observed. But, on the other hand, the synovial membrane is frequently found to be the seat of passive congestion, and to be immensely hypertrophied, being thicker in all its parts and more or less gelatinous or fibrous.

Symptoms and morbid changes in the chronic form.—Thus, although occasionally instances of acute and subacute tuberculous synovitis are encountered, it is with the distinctly chronic affection that we have to do in the vast majority of cases; and it is this form of disease which we must now study somewhat more in detail.

Chronic tuberculous synovitis, the "white swelling" of the older authors, usually dates from some injury, and is most frequently met with in children and in older individuals of delicate constitution and often with hereditary predisposition to tubercle. The first sign is usually some stiffness and swelling in the joint, and a sense of weakness in it. Occasionally there is a slight sense of heat, appreciable to the patient himself and to the hand of the surgeon, but this is often absent. As time goes on, the swelling increases and the sense of weakness becomes more marked. If left to itself the joint becomes flexed and otherwise deformed, as will be described in considering the case of special joints. At those joints where it is least covered by soft parts the synovial membrane will be found to have become voluminous, and to be elastic or doughy to the finger. This "pulpy swelling" is due to the formation, in the most superficial layers of the membrane, of a quantity of granulation tissue, the product of chronic inflammation with exudation of leucocytes.

A section (Fig. 63, page 344) through the membrane at this stage will show the superficial cell layer intact in many instances, and underneath this innumerable leucocytes grouped in special zones, in the centre of which are larger "epithelioid" cells surrounding in most cases a large irregular mass of protoplasm with peripheral nuclei, the so-called "giant cell," the whole constituting a "tubercle system" or simply a tubercle (Figs. 65 and 66, pages 347 and 348). Of these several may lie within the field of the microscope with a low power, or in other cases only one. Besides these tubercles the synovial membrane will be seen to contain thin-walled vessels in abundance surrounded by multitudes of indifferent exudation cells, and to possess a larger amount of new fibrous tissue in its substance than in the normal state (Fig. 431).

This stage may last almost for an indefinite time in some cases, but as a rule the new tissue undergoes further changes before long. In favourable cases the whole process may be arrested at this point, and resolution may take place. By this is meant that proliferation ceases, probably owing to death of the bacillus, if not of its spores, and the exuded matter becomes absorbed, most of the leucocytes wandering farther, and probably in the end entering the circulation through the lymphatics. Some of them may become organised too; but in any case the whole tuberculised area is restored to an almost normal condition, with perhaps some fibrous thickening.

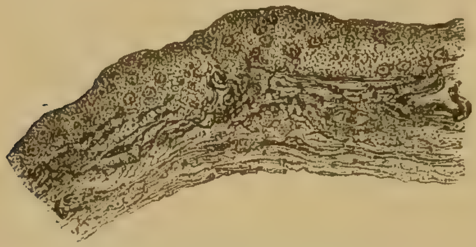

Fig. 431.—Synovial Membrane, thickened and infiltrated with tubercles. (From a Photograph by Prof. Cheyne.)

But, unfortunately, this is not the rule, and in the majority of cases further changes take place in the granulation tissue and tubercles themselves. As the cells become heaped up round the centres of infection, the latter are farther and farther removed from their vascular supply, and eventually to such an extent as to be incapable of further existence. They, therefore, undergo fatty degeneration, and form small centres of caseous material in the centres of the tubercle systems (Fig. 67, page 349). On section of the membrane at this stage, these foci of caseation stand out to the naked eye as white beads in the centres of masses of red or pink granulation tissue. Some of them may have been situated so close to the joint surface of the membrane as to have involved the latter, on which they may stand out either as white prominences or, having broken, as round white-walled cavities open to the joint. Every size, from a pin point to the volume of a grape, may be seen in the same specimen. But without involving the surfaces of the synovial membrane, such foci may go on enlarging and liquefying until large, soft masses of caseous material are formed, known as cold abscesses. These may spread in any direction, and often ultimately burst into the joint, but as often make their way outwards through the capsule, and form large peri-articular abscesses. Again, this tuberculous inflammation of the synovial membrane may so hypertrophy the latter as to throw it into folds and fringes, which spread out between the ends of the bones. If the process still advance, the pathogenic matter in the fringes may commence to attack the underlying cartilage and lead to its ulceration and destruction, as is well seen in Fig. 433, and to the left-hand side. In this way the whole cartilage may be gradually destroyed until the bones are laid bare; but in many cases it only shows small pits upon its surface filled with tuberculous granulation matter.

It is important, however, to remember that synovial disease of this kind does not always involve the surfaces of the joint, but may go on to the production of large extra-articular abscesses without doing so. Again after developing into caseous masses, these tubercles may become quiescent, and the dead tissue dry up into hard material in which the calcareous salts become concreted into chalk-like material. When this change sets in early, the functions of the joint may become restored, but it is rare for the disease to stop at this point when once it has reached the stage of caseation. Most frequently some of the caseous foci break into the joint, and the infective matter contained in them is thus brought into contact with sound tissue, and infects it over a large area.

These various changes are not accompanied by any special objective and subjective symptoms which enable us to recognise them with certainty. We are driven to study the whole aspect of the case: the time the disease has lasted, the strength and constitution of the patient, and the amount of swelling and interference with function, and to cast up the general evidence bearing upon the probable state of the parts within. But it is a common experience that we find as a rule more disease in such a joint than the aggregate of symptoms would have led us to expect.

Treatment.—The general treatment of tuberculous synovitis is practically the same as that suitable for syphilitic disease of the same structure. Everything which can by local or general means improve the vitality of the tissues should be employed. Thus abundance of digestible food, fresh dry air, good clothing, and moderate exercise are called for, together with the use of tonics and the preparations of iodine. (*See* page 359.)

Locally, we also apply iodine tincture, or blisters in the earlier stages; and if there is much pain, the part must be thoroughly immobilised. In very mild cases it is a question whether gentle exercise is not of some benefit so long as the part is not strained or rendered painful; but much caution is necessary, and in doubtful cases it is better completely to fix the joint in the position of greatest ease. In some early cases the use of the actual cautery to the skin over the joint, to cause rapid vesication or an actual sore, is of much benefit. Sometimes mercurial inunction, combined with firm strapping, is followed by improvement, but is not now so much believed in as some years ago. Again, the application of an elastic bandage for several consecutive hours daily, followed by massage, has been recommended as a means of reducing the passive engorgement of the synovial membrane. This method is of much benefit in early cases, and ought to be more widely employed than is at present the case.

Another mode of treatment has of late come much into use, especially in Germany; and at a recent meeting of the Surgical Society in Berlin I had the opportunity of hearing the opinions as to its value expressed by many prominent surgeons. Almost all were agreed that it had made the treatment of tuberculous disease of the synovial membrane far more satisfactory than formerly was the case, and had rendered operative interference far less necessary than hitherto. This method is the injection of iodoform powder, suspended in glycerine, into the affected tissues. The influence of this drug in checking the growth of the bacillus tuberculosis has been much disputed by many bacteriologists and pathologists, but I have been always struck with the always unanimous verdict in its favour of those surgeons who have had the largest practical experience of its use in clinical work. It is used in these cases as follows: Ten per cent. of finely powdered iodoform is suspended in pure glycerine, with 20 per cent. of sterilised water; and of this fluid about a drachm is injected into various parts of the diseased synovial membrane with a coarse hypodermic needle, until about one ounce has been used. This injection may be repeated at intervals of about two weeks. In some cases symptoms of iodoform intoxication have been observed, but this is rare. This treatment is usually combined with immobilisation of the part and massage.

Another mode also of dealing with these cases has been devised and practised by Dr. Bier of Kiel. This is known as the method of "Hyperæmic Engorgement," and consists in applying an elastic bandage over the extremity as far as the affected joint, leaving the latter bare, and again applying a bandage above the joint in such a way as to produce an engorgement of all the vessels in and around the joint for some hours daily. This, it is stated, is followed by arrest of the tubercular process and death of any tubercular foci already present in the part. Those who have tried this plan speak highly of it, but I gather from others who have employed it and the iodoform treatment, that the latter has satisfied them better. It is not to be supposed, however, that either of these two methods will supersede the opening of tuberculous abscesses when they have formed, nor the removal of diseased bone in certain cases. This will always be proper in any case. But where the disease is fairly early in development, both methods deserve a further trial.

4. **Gouty and chronic rheumatic joint affections.**—There naturally follow upon the syphilitic and tuberculous group of synovial affections another set which exhibit their chief peculiarities in degenerative changes, although cell-proliferation is also present as a secondary process. These affections are, however, by

no means limited to the synovial membrane, but involve the cartilages, bones, and ligaments equally, if not to a greater extent. This is especially true of gout, which must now engage our attention for a few moments.

Gout.—This disease, as is well known, depends upon the presence in the system of a definite chemical body, the result of faulty metabolism, which in certain tissues leads either to loss of vitality or to low inflammatory changes due to the latter. This chemical substance is *urate of soda*, which tends to deposit itself in those tissues which naturally possess the least vital energy, namely, in the cartilages, ligaments, and bones. The synovial structures, however, are also affected. The question then suggests itself whether this deposition may not be due, at all events in part, to lack of vitality in the tissues first mentioned, owing to which they are unable to eliminate or resist the power of the salt to crystallise in them, which it therefore does in tufts of acicular prisms of great delicacy. In solution, too, it passes into the joint with the synovial secretion, and is there precipitated in the crystalline form, and often in sufficient amount to distend the sac with a mass resembling chalk to the eye. It is the presence of this product which is supposed to provoke that hyperæmia culminating in those peculiar explosive attacks of inflammation of the peri-articular structures so familiar to physicians. It is very likely, too, that its presence in the tissues in solution produces a loss of energy, laying them open to the degenerative changes which are found in them.

The **symptoms**, as seen in the synovial membrane in gouty change, are hyperæmia accompanied by heat, redness, and a puffy swelling, with throbbing pain most marked at night.

These symptoms may be observed in almost any joint, but are undoubtedly most frequently seen in those which are most exposed. Among these may be mentioned the knee, elbow, ankle wrist, and above all the metacarpo-phalangeal joint of the great toe.

The **treatment** consists in regulation of diet, the use of diluents, and the administration of laxatives and colchicum in free doses. Locally, there must be rest for the part involved, with elevation and the protection of it from external irritation in the form of rapid alternation of heat and cold. This is best met by wrapping up the part in warm cotton-wool or in moist compresses. Later, gentle massage may be employed with benefit. It acts in unloading the engorged vessels, and in promoting the absorption of the urate deposited in the tissues. This disease is so commonly considered as belonging to the department of medicine, that its further treatment may be relegated to the physician.

Chronic osteo-arthritis.—It will be convenient to consider now a disease which in some respects appears to have many features in common with gouty arthritis, although it cannot be said to range itself easily among the synovial affections. This is the so-called "chronic rheumatic arthritis," for which a number of other names have been proposed, such as "arthritis deformans," "rheumatic gout," "chronic osteo-arthritis," "arthritis senilis," "arthritis sicca" or "nodosa," and lately "polypanarthritis."

The number of these designations indicates in itself that none of them are held to convey any clear characterisation of the disease. But in the present imperfect state of our knowledge as to the pathology of the condition, it is just as well to employ some term which implies no particular theory as to ultimate causation. The term "osteo-arthritis" is probably less open to objection than any of the others mentioned, and is, perhaps, the most commonly used at present.

Pathology.—Osteo-arthritis is essentially a disease of later life, and occurs, in the great preponderance of cases, after fifty, though occasionally found among feeble individuals at an earlier age. It is far more common among the ill-fed and badly-clothed than among the more favoured classes. It has thus come to be called "poor man's gout." Again, habitual exposure to severe changes of weather appears to dispose to it, not only by acting locally upon the joints, but also by lowering the general nutrition of the body. The disease, indeed, appears to depend, at all events in its beginnings, upon some loss of vitality in the tissues of the body, generally due to several depressing causes combined, which leads up to degenerative changes. But as the condition advances we recognise the introduction of other factors, in the shape of subacute inflammatory processes, due to the disturbance of function depending upon the degenerative changes in the first instance. It would be incorrect, then, to describe this affection, in the form in which it is usually met, as purely degenerative, seeing that its more marked results are those produced by secondary inflammation, not always destructive, but often of a plastic character.

If we examine a joint in which this affection is in its earliest stages, many striking alterations are evident, even to the naked eye. In the first place, the articular cartilages will appear roughened, and on close inspection fibrillated, the fibrils standing vertical to the surface, but all this without any evidence of inflammation. This alteration is now believed to be the result of degeneration of the cartilage cells, and their removal leaving behind nothing of the original structure but the intercellular matrix in the form of columns and fibres. This degeneration may be due to age, exposure, unhealthy blood-supply to all the tissues, reducing their vitality, or to all these causes combined. Finally, it has been suggested that it may be a trophic nerve change, induced, in the first instance, by the causes just mentioned operating upon the nervous system, and leading to defective innervation, using the term in its widest sense. However this may be, the first change of all in the disease is degenerative, and before it has progressed very far it leads to actual loss of substance in the cartilage. For the latter, in its weakened state, appears unable to fulfil its ordinary functions, and is gradually worn away in the movements of the joint, until at last the two surfaces of the bone are actually in contact

Then another condition begins to be noticed around the borders of the encrusting cartilages, advancing *pari passu* with the using up of the latter. This is an irregular swelling of the tissue, producing eminences of a pearly hyaline look or yellowish colour. These are noticed at those aspects of the joint where the movements are least felt—namely, at the borders of the encrusting tissue, where it touches the periosteal and synovial vascular supply, and where the nutrition of the cartilage may be said to be at its best. These nodules only interfere with the free play of the parts during the extraordinary movements of the joint. They are, beyond all question, due to hyperplasia of the cartilage, which latter is explained either by irritation produced by the play of those parts of the surfaces of the articulation which are undergoing degeneration one against the other in their roughened state, or by the presence in the blood of some irritant substance. The first of these is the most likely hypothesis as far as we know at present. Together with this obvious hyperplasia, there is usually a certain amount of hyperæmia of the nearest vascular tissue—*i.e.* the synovial membrane—but only in sufficient quantity to produce a slight amount of increased secretion. This was formerly supposed to be the initial change, but is now recognised as probably only secondary to the earlier degenerative disorganisation. For it is easy to understand that, when hard osseous tissue comes to rub against osseous tissue, without the interposition of cartilage, there must be an amount of friction, and consequent irritation not present beforehand.

Then, as this disease advances, the bone thus injured becomes, in its turn, hyperæmic, and begins to throw out new osseous tissue, especially at the periphery of the joint, where it is not subjected to pressure. In the first instance, this hyperæmia leads to softening of the bone by the well-known processes of rarefaction, so that it is less resistant to pressure, and soon becomes altered in shape on its surfaces being moulded and rubbed down by the opposite bone (Fig. 432). The new material, too, at its edges is at first soft, and may assume various shapes, according to the forces brought to bear upon it. As a rule, however, it presents itself in the form of lobes and irregular nodules, which have been compared by Billroth to "tallow drippings." The effect of the chronic hyperæmia upon the synovial membrane is to cause its gradual thickening. At the same time, it becomes rough on the surface, and frequently throws out hyperplastic fringes or pendulous growths. Sometimes these latter are torn off by the movements of the joint, and so form loose bodies; or they may become ossified or calcified, and then seriously interfere with the functions of the part. Every museum contains examples of these loose bodies.

It is the presence of this new bone round the borders of the joint which gives it the lumpy appearance seen and felt through the skin, and which so seriously impedes its movements, and gives rise to the grating so familiar in these cases. But, though there may be much material thrown out in such cases, it rarely, if ever, leads in simple chronic osteo-arthritis to fusion or synovitis of the two bones. The latter may be firmly locked one into the other, but they are not fused, as in cases of ordinary inflammation. The reparative powers of the part are, in fact, below par, and the plastic processes only go to a certain stage, and no farther.

In some cases, however, the process just described is slightly modified, though essentially the same. For some unexplained reason the degenerative process seems to progress more slowly, and the cartilage is only tardily worn away. The underlying bone at the same time is only very gradually irritated as its surfaces become more and more bared of cartilage. The consequence is an inflammation of a lower degree of intensity, which, instead of leading to softening of the bone, is the cause of a sclerosing osteitis in its superficial layers. The bone thus hardened is more slowly ground and worn away than when rarefied, and as a consequence has a smoother surface at the worn points of contact. These frequently become actually polished, when the whole surface assumes the appearance of porcelain or ivory. In this state the movements of the joints may be fairly free and almost without grating, the surfaces being so perfectly smooth.

It is said that occasionally the process of degeneration may lead in cases of osteo-arthritis to the formation of caseous material, which will float about in the synovium or lie embedded in the tissues forming the surfaces of the joint. But this is certainly very rare, and when met with suggests very strongly that there is a tuberculous or syphilitic element in the case, tuberculous arthritis being by no means so uncommon in elderly patients as is commonly supposed. At all events, if such caseous degeneration does take place in chronic rheumatic arthritis, the course which the disease pursues is precisely that of the tuberculous foci of arthritis. The joint becomes swollen, doughy, and tender, in addition to the usual signs and symptoms of the ordinary form of the affection, and sooner or later soft spots are felt under the skin, and the latter breaks, discharging curdy broken-down material. In this way the joint may be opened up, and if the sinuses should then become infected ordinary suppuration with all its dangers is grafted upon the degenerative process, and very soon leads to the utter disorganisation of the whole articulation and to the most grave constitutional disturbance. Such cases, as already stated, are very rare, and if the softening process become developed in a case in which osteo-arthritis had been previously present in one or more of the joints, careful inquiry and examination should be made, in view of the possibility of syphilis or tubercle, before it is admitted that the process is due to the original disease.

Symptoms.—The symptoms in all the various phases of osteo-arthritis are both subjective or objective. In the earliest stages when the cartilage is degenerating the patient will notice first some creaking in the joint, with slight insecurity in using it. There is, as a rule, but little pain at first, and no tenderness. At this stage nothing objective can be made out, except that the roughened surfaces do creak on movement under the hand. Later, when the low inflammatory action is beginning, and the border of the articular cartilage is becoming nodular while the synovial tissue is irritated, the patient complains of dull aching pain, aggravated by motion. Greater creaking or actual crackling will now be noticed, and the lumpy enlargement at the borders of the articulation can be made out with the hand. Probably, too, the joint will contain a certain amount of fluid, and there may be a certain elevation of temperature without redness. This condition, of course, interferes with the movement of the joint, not only because of the pain, but also owing to the alteration of the shape of its surfaces, due to the bony growths. Any attempt to carry the bones through their usual movements leads, of course, to stretching of the ligaments, with great increase of suffering. With the advance of the disease all these symptoms become aggravated, especially the last. But, ultimately, if the joint surfaces become locked in one another, movement being arrested, the pain generally ceases, except that at times, especially at night, the bones may ache.

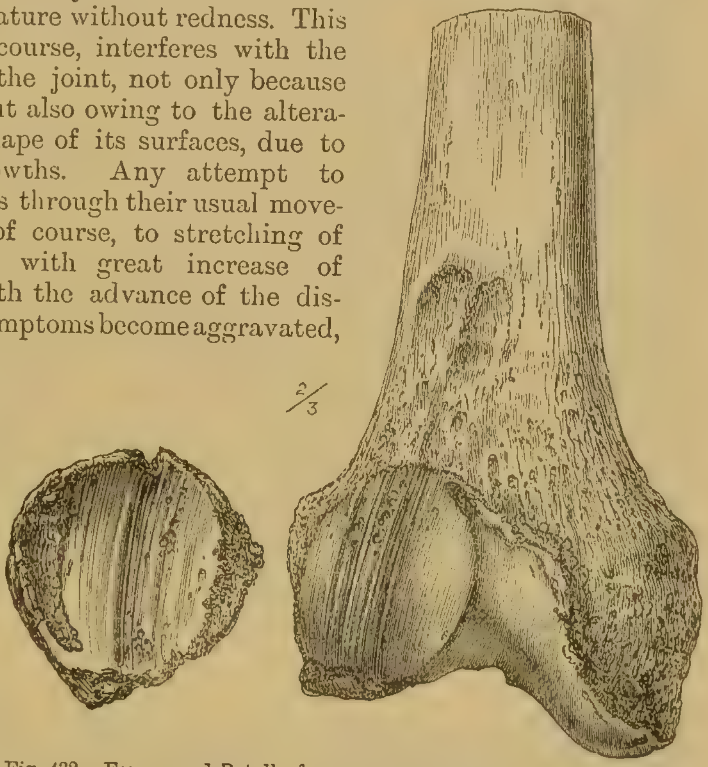

Fig. 432.—Femur and Patella from a case of chronic Osteo-arthritis, showing the effects of friction.

So far as we know, all the joints of the body may be affected, either singly or together, by osteo-arthritis. But there are some which appear to be peculiarly liable to the disease. Among the larger articulations the following is about the order of frequency: knee, hip, shoulder, elbow, ankle, sterno-clavicular, and temporo-maxillary, the smaller joints showing it only a little less frequently. Even the smaller articulations of the vertebræ have no immunity from the disease, but may be extensively involved.

Both sexes appear to be equally affected by osteo-arthritis; perhaps the larger joints are more frequently attacked in males, the smaller in females.

The **treatment** of this affection is, on the whole, unsatisfactory. Depending as it does primarily upon degenerative processes, it is almost too much to hope that we can effect much by remedial agents. Cure of the condition when well marked there is none known; the structural changes cannot be repaired. We may, perhaps, arrest the rapid progress of degeneration at the outset by appropriate treatment, or when advanced relieve the suffering caused by it, but more than this we cannot claim.

The general strength of the system may be improved by nourishing food of a light digestible kind, and by warm clothing, fresh air, and tonics. Again, if the blood be loaded with effete products, the result of improper food or excess in eating and drinking, we may by simplifying the diet and the use of laxatives and emunctories, and by encouraging the action of the skin by the employment of Turkish baths, massage, etc., effect a good deal.

Locally, the nutrition of the part involved may be much helped by protecting it from the vicissitudes of the weather by warm coverings of cotton-wool or flannel. Douches and hot baths are also sometimes of use, and counter-irritation may even be resorted to occasionally with benefit. Pain may be relieved by anodyne applications, such as belladonna or opium. I have also found that carbonate of lithia and iodide of potassium possess the power of relieving the pain of this disease, although an explanation of the fact is not easy.

Of the benefits accruing from the use of certain mineral baths there can be no doubt. Thus I have known patients very much improved by residence at Wildbad, and the use of its natural springs, when other treatment had been productive of no benefit. The other resorts generally regarded as of special use in this disease are Buxton, in England, Suchon, in the Pyrenees, Vichy, Ems, Wiesbaden, and Carlsbad. But of all I give the preference to Wildbad, which for some reason or other appears peculiarly suited as a resort for those affected with osteo-arthritis, gout and rheumatism.

DISEASES OF LIGAMENTS.

Primary affections of the purely ligamentous structures of joints are exceedingly rare. When ligaments are diseased, it is nearly always due to extension from the neighbouring synovial membrane. This is only what we might expect when we remember that the structures in question play a more or less passive part in the functions of the joint, and consist of a highly differentiated tissue.

Inflammation.—When ligaments are, however, severely strained, or actually torn, they may become inflamed. The process is, as a rule, sluggish, the vascular supply being very limited, and the actual cellular elements in the tissue few. They will be found, in such a case, more injected than usual, the vessels passing among their fibres being dilated or, perhaps, increased in number. If this hyperæmia lasts long, the whole ligament becomes permeated with leucocytes, and thereby assumes a softened, pulpy consistence, its individual fibres, too, being softened at the same time.

The **diagnosis** of such a condition would not be difficult, in view of the injury. There would probably be some ecchymosis, localised swelling, tenderness, and pain, aggravated by particular movements. There would also, in all probability, be some effusion into the joint, due to the hyperæmia having reached the synovial membrane, or the latter might have been injured at the same time.

The **treatment** of inflammation of the ligaments of joints in the early stages does not differ from that described as suitable for synovitis (page 1035), and need not detain us. But in these cases the structures will require to be supported by strapping and bandages for longer periods than if the synovial membrane was alone affected.

Inflammation of ligaments, once established, is very prone to become chronic, and to leave much weakness in the part affected.

New growths.—Ligaments are not likely to be the seat of new growths, and are very rarely so affected; but they are occasionally the seat of cartilaginous or bony deposits, which may require operative treatment, on account of their interfering with the movements of the articulation. These are met with usually close to the insertion of the ligaments, so that it is difficult to say whether they have started in the latter or in the subjacent bone. (*See* page 907.) Unless these are actually interfering with the joint, it is well to abstain from operation upon them, inasmuch as they are not prone to grow to any magnitude, and usually, after a short period of activity, appear to remain stationary. In this they resemble the small ossifying chondromata, which are so frequently found growing about the ends of the bones, close to the joints, and their pathology is probably the same.

Secondary diseases of the ligaments are common, and are considered with the affections with which they are associated.

DISEASES OF THE CARTILAGES.

Here, again, we find that primary disease is rare. Cartilage plays but a subordinate part in the economy, its functions being almost passive, and it is a highly differentiated tissue, and therefore exempted from fluctuations of circulation, which affect the more embryonic connective tissues. When it is diseased the affection is usually either the result of degenerative changes, or is secondary to inflammation in adjacent structures. New growths, again, starting from articular cartilage are almost unknown.

Degenerative changes.—Degenerative changes in cartilage, such as those seen in osteo-arthritis and in Charcot's disease, or in the debility of extreme old age, commence usually as a fatty degeneration of the protoplasm of the cells, which ultimately involves the whole body of the corpuscles. The latter, having in this way lost all their vitality, or having conversely undergone the change in consequence of the loss of vital energy, break up. The cells having thus been destroyed, the matrix or less vital part soon follows, breaking up first into columns and bands, and finally liquefying and exfoliating into the joint, until the surface of the bone is left bare.

Sometimes there are seen in the matrix itself rows of fatty granules, vertical to the surface, which ultimately separate columns of the matrix one from the other, until the whole tissue has the appearance of a "pile" fabric like coarse velvet, and, when stroked over with the finger, all the fibres are bent in one direction, as a field of corn beaten down by the wind.

These changes may depend upon age, damaged innervation, insufficient food, or the circulation in the blood of deleterious materials, damaging the nutrition of the tissues. That the latter may operate in this way is shown by the somewhat analogous condition of cartilage, produced by the presence of urate of soda. Here, as the result of mal-assimilation or bad elimination, or of both, a harmful material accumulates in the blood, and is carried through all the tissues of the body, inducing the changes already alluded to (page 1051). When very abundant in the blood, this salt is deposited in the cartilage in the form of bunches of a circular form, and may give the tissue a chalky appearance. Its presence here leads to loss of vitality of the tissue, and consequent degenerative changes, the matrix breaking up as in the fatty change just described, until it is completely destroyed. Analogous materials of a non-crystalline form may also produce similar changes, without being precipitated from the blood as a solid. This seems likely to be the primary cause of the degenerative changes seen in osteo-arthritis. The pearly swelling, on the other hand, seen at the borders of the encrusting cartilage, is probably secondary, and due to the irritation of friction between surfaces now roughened, and consisting of material never designed to bear the movements of the articulation.

Secondary affections of cartilage.—Secondarily, joint cartilage is also otherwise affected in many ways, like other forms of connective tissue. That is to say, if any powerful irritant be brought into contact with either its free or attached surface, its intercellular substance and cells are attacked and destroyed in a similar way.

Allusion has already been made to the effect of septic matter within a joint upon its encrusting cartilage, and also to the consequences of simple suppurative synovitis. The differences between these, if any, are only those of degree. In both cases a substance capable of attacking and devitalising the cells and matrix of the cartilage is brought into contact with the latter. As a result, the corpuscles are destroyed, and the capsules open towards the joint, producing little pits upon the surface. Through these depressions the irritant reaches a deeper layer of "mother capsules," producing the same changes in them, until they likewise burst towards the joint, and the whole cartilage is "honeycombed," even as far as the underlying bone.

In septic synovitis this process of destruction is usually rapid and uniform over all the surfaces, which appear as if digested in some solvent fluid. In syphilitic disease, and in tuberculosis, on the other hand, where the infection is slower and the irritation localised, the cartilage is often only pitted with small ulcers, leaving healthy tissue between them, or the margins are destroyed at spots corresponding to the fringes of infected synovial membrane, which overlap them (Fig. 433).

In the same way the cartilage may be destroyed by irritation reaching it from below. The process may be rapid or slow, which is the more common. In a case, for instance, in which a rarefying non-suppurative osteitis exists in the end of a bone, the inflamed medulla may press upon the under surface of the cartilage. In this case the cells of the latter are soon destroyed, as already described, and the capsules ultimately burst, but this time towards the bone. The inflamed medulla now enters the pit thus formed, and exerts its irritating effect upon a deeper layer of cells, causing in them the same degenerative changes as before, with the same result. Thus the whole encrusting cartilage may be tunnelled through from below, until the inflamed medulla projects into the joint in the form of bosses of granulations, projecting through otherwise healthy cartilage (Fig. 434). Masses of the latter may in this way be cut off all round by granulations, and be at the same time separated from the bone below by layers of the same material. In this case flakes of cartilage will be separated and shed off into the joint, in the form of white chips of varying size. This exfoliation is, on the whole, not infrequent. All the varieties of destruction of cartilage alluded to may be met with in the same joint.

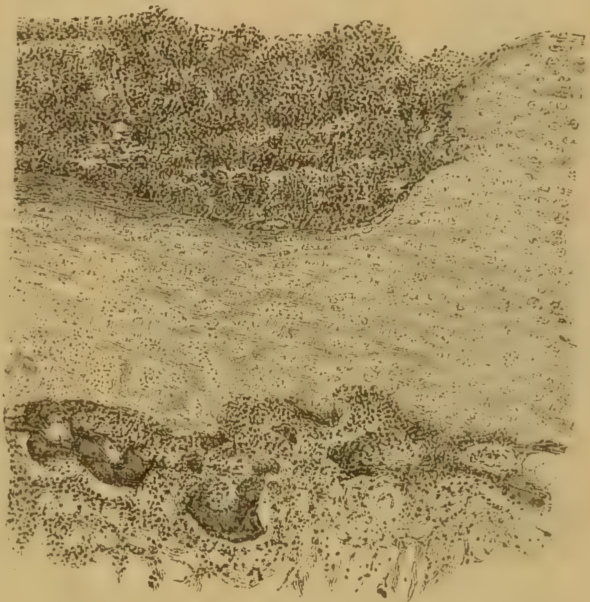

Fig. 433.—Tubercle attacking the Cartilage of a Joint Surface. (From a Photograph by Prof. Cheyne.)

The symptoms of destruction of cartilage, apart from those of the disease of the joint which lead to it, are few. Objectively, we have in the degenerative form an unevenness in the movements of the surfaces one upon the other, or even creaking or grating. In the acute inflammatory forms alluded to, the painful condition of the joint will usually prevent all movement, unless the patient be anæsthetised, in which case the roughened condition of the cartilages may be made out by gentle flexion and extension, or, perhaps, by lateral movement. An old criterion of cartilage destruction has always been a certain "starting" of the limb with acute pain, especially when the patient is dropping off to sleep, and there is a certain amount of value in the sign. The suffering caused by these sudden involuntary movements of the joint is very great, and the patient is unable to control them. They are not usually present, however, until the ulceration has progressed to a considerable extent. The explanation of the phenomenon is probably this: cartilage is not a sensitive tissue, but the other structures of the joint are pre-eminently so. When the former is ulcerated over two bones at their point of contact, the muscles governing the joint will involuntarily do all in their power to prevent the inflamed surfaces from rubbing one against the other, and will keep them firmly fixed in one position, simple contact of the diseased surfaces producing no pain. When, however, the muscles become fatigued from time to time, or the patient is dropping off to sleep, the former relax a little, and immediately the surfaces slide one upon the other, causing sudden pain at the moment. This leads to reflex contraction of the muscles once again, and the spasm produces acute pain in all the structures that are diseased.

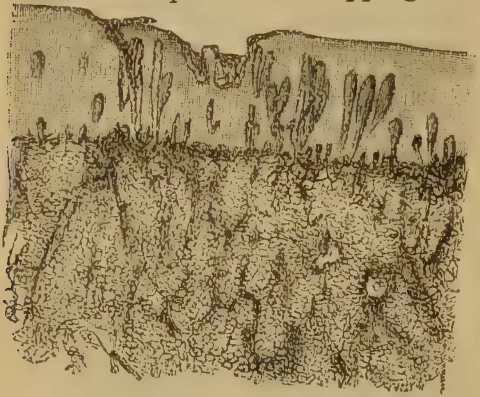

Fig. 434.—Destruction of Cartilage by flask-shaped Prolongations of Granulation Tissue from below. (From a Photograph by Prof. Cheyne.)

This symptom of starting is, however, not invariably present with all forms of destruction of cartilage. In osteo-arthritis it is absent, also in gout and in Charcot's disease; and even in the acute change which accompanies pyæmia it is not a noticeable feature in the destruction of the cartilage.

The **treatment** of diseases of cartilage has nothing special about it apart from that of the disease in which it is only a single factor.

The **prognosis,** however, in any case where the surfaces of a joint have been destroyed by acute or chronic inflammation, is always grave, in the first place often as regards the patient's life, for this indicates very serious disease, and next as regards the functions of the articulation. For once the cartilage is destroyed, it is not replaced by the usual hyaline material which covers the ends of bones, but by fibrous tissue; and as this is equally developed on both surfaces, it forms a bond between the two, which prevents movement. (*See* Anchylosis, page 1067.)

JOINT DISEASE COMMENCING IN THE BONES.

In the article on Diseases of Bone (page 873) will be considered the details of the various morbid processes found in the osseous system. We shall, therefore, in this place only consider the outlines of the changes as they more particularly affect the joints.

The separate diseases of bones which affect the joints are few, and excluding the results of injury, the vast majority of them will come under the heading of either syphilis, tubercle, osteo-arthritis, or Charcot's disease. There are, of course, others, such as the acute septic inflammations of the bones, which occasionally play a part here; but as a matter of fact, these almost invariably stop short at the epiphysial lines, and as the latter in most cases lie outside the capsule of the articulations, the chance of their invading the joints is small.

Syphilitic disease.—This disease, as it affects the ends of the bones entering into the structure of joints, is usually a question of a growing gumma (*see* Fig. 82, page 408), which spreads through the articular cartilage, just as in other cases it advances through the skin, and finally gives way on its surface. In such a case, if we examine the bone we find the syphilitic granuloma in the cancellous tissue of its end presenting very much the appearance of inflamed medulla. This may or may not be undergoing caseous change in one or other spot. The bone around is rarefied in most cases, but sometimes sclerosed if the process be more chronic. The cartilage around the borders of the gumma is normal, unless where undermined, when it is yellow or grey and shreddy.

Tertiary disease of the end of a bone may also affect the joint when it takes on the form of a chronic osteitis, with the formation of osteophytes projecting more or less into the joint; or a syphilitic sclerosing osteitis may gradually lead to the formation of sequestra, which may work their way into the cavity of the articulation, and set up there a disorganising inflammation.

The **diagnosis** of these conditions is not difficult, resting as it does upon the recognition of the constitutional condition, and a comparison of the local symptoms with those of the other possible affections of the joint. There will also in many cases be evidence of local syphilis elsewhere, in the form of scars and nodes, interstitial keratitis, or stunted teeth.

The **treatment** is that of syphilis in its most advanced stages, and it is only necessary to emphasise the fact that here it is of quite as much importance to attend to diet and everything which will improve the general nutrition of the body, as to give the usual specific remedies of syphilis, such as iodide of potassium and mercury, inasmuch as the varieties of this disease, which reach the joints as above described, are associated, as a rule, with great debility, and probably depend in a large measure upon it for their extremely destructive characters.

Tuberculous disease commencing in the bones entering into the composition of joints is, perhaps, the commonest of all the affections of these structures, and seems to be especially so in England. (*See* page 879.) It is frequently a primary affection, appearing in the ends of the bones quite independently of any other infection of the system which we can detect by our present methods of diagnosis. It is, of course, also at times a secondary affection, in many cases appearing in the joints after a wider distribution through the system. Attempts have been made to estimate the relative frequency of primary and secondary tuberculous bone disease, but so far they have failed, and in the nature of things they are likely to fail for some time to come. When tuberculous disease attacks the ends of bones it most usually has its first seat in or about the epiphysial line, where there is much young growing tissue (Fig. 436). This form of disease is often referred to as "tuberculous epiphysitis," a name which has a certain amount of convenience, and which may be employed with the proviso that it be understood that the affection starts rather at the junction of the epiphysis and diaphysis than in the actual epiphysis itself, although it has a tendency in most cases to spread into the latter. But tuberculous disease may also start in the end of a bone under the encrusting cartilage, where there is also a quantity of young embryonic tissue, peculiarly vulnerable to this specific infection. In either case, the morbid process consists in an infection of the medullary tissue with the bacillus tuberculosis, which soon excites in the latter a low form of inflammation, characterised by cell multiplication round certain centres, until nodules of soft granulation are formed, which, as they increase at the periphery, undergo degenerative changes towards their centre (Fig. 437). This process may extend from the epiphysial line or under the encrusting cartilage through the latter, and so invade the whole joint in all its parts. But it must not be forgotten that it may also extend in a lateral direction altogether, and though very close to the joint surfaces, may never penetrate through them, but may open on the surface of the limb hard by, leaving the joint proper intact (Fig. 435).

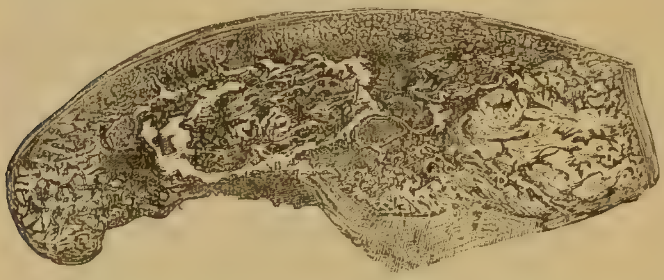

Fig. 435.—Metacarpal Bone, with tuberculous sequestrum in centre, cut off from the inflamed bone by granulations. The thickening of the periosteum over the inflamed area is well seen, but both joint surfaces are free. (From a Photograph by Prof. Cheyne.)

Limbs have over and over again been removed under the impression, on the part of the surgeon, that the joint was gravely affected because sinuses ran down from the surface almost into it, when it has been found afterwards that the joint proper was in no wise involved, and that the focus of disease might have been reached without any interference with its surfaces. On the other hand, we must remember that chronic tuberculous disease is frequently so quiet in its progress, that it may have passed from the bone through the cartilage and extensively invaded the joint cavity, without having produced much disturbance in the latter. In such a case we often find that there are bosses of tuberculous granulations projecting through holes in the cartilage from below (Fig. 434), where the chief centres of disease lie, while the cartilage itself, on the rest of its surface, seems quite unaffected, and the synovial membrane almost or entirely healthy. In other instances, when such an invasion of the joint takes place from the bones, the granulation, once having entered the joint, spreads rapidly over its surfaces, and commences at once to attack the cartilages on their free surfaces as well as the synovial membrane. This is only seen where the infection is more of the acute and rapidly progressive character, indicating virulence in the poison, and great vulnerability of the individual. (*See also* page 1047.)

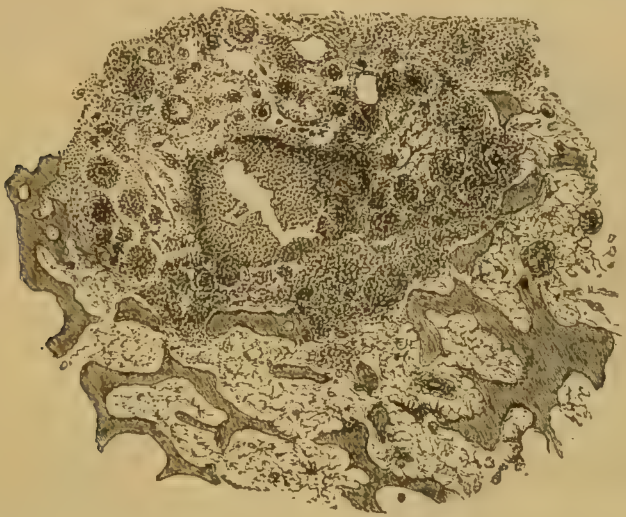

Fig. 436.—Focus of Tuberculous Disease in the lower Epiphysis of the Femur. (From a Photograph by Prof. Cheyne.)

The **symptoms** of tuberculous disease in the ends of bones, taken with the patient's past history, are usually easily recognisable. In the first place, the age of the individual will guide us somewhat, the affection being met with mostly at the two extremes of life. We have, besides, frequently, though not always, a history of injury, and often of a plain hereditary predisposition to guide us. The subjective evidence as to the local complaint is usually clear: there is stiffness and tenderness of the part, with pain felt chiefly at night, and of a gnawing character. Gentle percussion with the finger over the end of the bone will often show the spot at which the process is most marked, or, indeed, discover it when otherwise doubtful. Objectively, we see swelling in and often a trace of œdema over the end of the bone increasing its bulk considerably, and, as the disease advances, often a doughy area where caseation of the tuberculous material is in progress. The colour of the skin is rarely altered, but the veins may be more than usually evident. When the surfaces of the joint are affected we have its position altered, the patient putting it in that which gives the greatest ease. If allowed to run on without suitable treatment, abscesses will often form, and sinuses when the latter burst, after which the condition of the end of the bone can be easily made out as a rule with probe or finger.

The **treatment**, constitutionally, is that of tubercle generally—*i.e.* everything which may improve the general nutrition of the body, in the form of good food and fresh air, tonics, and warm clothing (page 359). Locally, the chief thing at the outset is rest, with counter-irritation in the form of blisters and tincture of iodine. Later, when it is quite evident that the process is increasing, we cannot do better than cut down upon the focus of disease in the end of the bone, and remove it by the gouge or chisel. If this be done at a comparatively early stage, we have the comfortable assurance that it may prevent invasion of the joint, and, if carefully carried out, such an operation ought to be followed by union by first intention. And even where extensive abscesses have formed in connection with such disease in the ends of the bones, we must be cautious in assuming that the joint proper is affected, and must proceed with the utmost caution, so as by all means to avoid interfering with the latter. It is very striking how much may be done in many apparently unfavourable cases in this direction, and our aim should be here, as in all tubercular diseases, to go to all lengths in the direction of conservative surgery compatible with proper eradication of local foci.

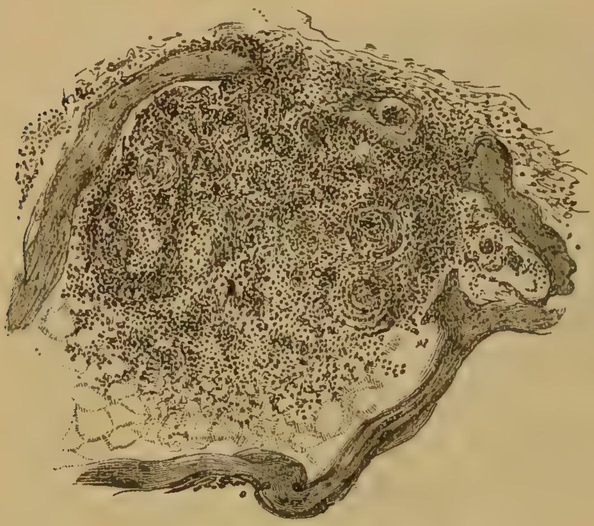
Fig. 437.—Focus of Tuberculous Disease in Bone. (From a Photograph by Prof. Cheyne.)

The **prognosis** of tuberculous disease of the ends of bones is favourable, on the whole, if the affection is seen and treated with or without operation fairly early in patients not altogether too debilitated, and we must never forget the strong tendency there is in all young subjects of tolerable physique to shake off tubercle if only the local and general powers are placed under favourable conditions.

Charcot's disease.—The degenerative changes of osteo-arthritis and of Charcot's disease are, as has already been pointed out (page 1051), followed in some cases by hyperplastic processes in those areas of the bone bordering on the part actually breaking down. These manifest themselves in the first place usually as a rarefying osteitis, which softens the osseous tissue about the joint to such an extent that it is capable of moulding itself into various altered shapes, depending upon the movements of the bone resting against it. In other cases the hyperplasia assumes the bone-forming type, and masses of new osseous tissue are thrown out, especially round the borders of the articulation, until its movements are seriously interfered with. (*See* page 701.) Again, there may be in either of these diseases a certain amount of sclerosing osteitis at one or other of the limits of the degenerative process, by which the breaking down of the end of the bone is arrested. Thus we may have the three well-known forms of bone inflammation in different stages of the same affection, that is the "rarefying," "bone-forming," and "sclerosing" osteitis. This simply means that these processes are only due to varieties in the amount and quality of the hyperæmia present.

Acute epiphysitis.—There is a group of acute affections of the ends of the bones in very young children, which, as they may simulate or actually lead to joint disease, require a few words of notice here, although properly coming under the heading of diseases of the bones. (*See* page 883.)

Mr. Thomas Smith in a series of twenty-two cases shows that eight occurred in the first month of life, nineteen within the first six months, and three more within the first year.

The **ætiology** of these cases is still obscure, but there seems to be a considerable amount of evidence in favour of the view that in the majority of cases, at all events, the affection is due to septic infection through the sloughing remains of the umbilical cord. As a determining cause it is possible that injury plays a part by lowering the vitality of the epiphysial tissues. Such an injury may be received in utero or during labour, either from the natural efforts of the uterus or from instrumental delivery. But be this as it may, the disease in all its features is one of septic infection.

Symptoms.—The disease is characterised by the rapid development of inflammation at the lines of growth of the diaphysis and epiphysis, often within a few days of birth. This has a great tendency to run on rapidly to suppuration, the pus making its way either directly through the end of the bone into the joint, or more frequently extending across the whole plane of the epiphysis, and separating the latter from the diaphysis, finally bursting the periosteum laterally. Again, it may run up the shaft more or less. When it escapes laterally under the periosteum, it depends, of course, upon the relation of the epiphysis to the capsule, whether the pus shall make its way into the latter or not. In the hip, for instance, the joint is inevitably involved, while in the case of the knee the pus may escape without entering the articulation.

When the joint is invaded, rapid solution of all the cartilages is usually the result, the bone, too, in many cases, suffering severely. In other cases the capsule ruptures early, and the pus discharges through the skin for a time, and finally the whole process subsides. And from the fact that the movements of the part are restored, we are forced to the conclusion that the surfaces have not become extensively diseased. The rapidity with which the pus forms in such instances is remarkable, but the possibilities of repair after it has been evacuated are even more so. Thus in some cases where the affection has run a rapid course, and the pus has been drained off early, the patient may recover with an excellent joint, with all its movements free, or even with increased mobility in others, though usually with shortening of the limb from interference with the growth of the epiphysis.

As in the syphilitic and tuberculous form of epiphysial disease, this affection will be found most often in those bones in which growth is most rapid. Thus in the series just alluded to, in those which proved fatal, out of twenty-two joints affected in thirteen infants, the hip was the seat of the disease in ten, the knee five times, the shoulder four, the elbow, ankle and sacro-iliac joints each once. Of those nine cases which recovered, out of twelve joints affected there were five knees, three hips, two elbows, one shoulder, and one ankle. This rough estimate in a limited number of cases is probably nearly what would be found to be correct in a larger number.

Subacute epiphysitis is, as a rule, either of tuberculous or syphilitic origin, and as far as the first variety is concerned, requires no further description here after what has been already said upon the subject above (page 1062).

But as regards the syphilitic forms of the affection, a few words may be added to what has been prefaced. This affection has recently been studied very closely. The disease may be the result of either hereditary syphilis or that acquired within the first few years of childhood. It consists in an inflammation of the epiphysial lines, chiefly of the long bones, producing separation in many cases of the end from the shaft, demonstrable either before or after death. This may be accompanied by the formation of abscesses with external sinuses, or may run its whole course even to great swelling of the ends of the bones and separation with abscess, and yet be recovered from in a short time. The inflammation is regarded by Wegner as secondary to the calcification, to a large extent, of the epiphysial cartilage, due to the irritation of the syphilitic poison, such calcification going on faster than the usual vascularisation, the earthy matter acting as a foreign body and thus leading to inflammation. Ranvier, on the other hand, regards the inflammation as primary in the growing line, and produced by the irritant of the syphilitic poison upon the growing vascular tissue. But one cannot avoid the thought that in these cases, although the patients are undoubtedly infected with syphilis, perhaps the affection of the epiphysis may after all be independent of the specific poison of that disease, and be due simply to one or other of the ordinary septic processes, attacking those whose vitality is seriously lowered by the constitutional disease—in other words, it may be a mixed infection.

A very remarkable characteristic of this form of bone disease is the possibility of complete recovery of the joint when the inflammatory products have been thoroughly and early let out.

ANCHYLOSIS OF JOINTS.

Varieties.—In all joints whose surfaces have been altered by inflammation the movements may become limited to any degree after the subsidence of the process, and as a consequence of it. This limitation of movement will vary from trifling stiffness to actual suspension of all motion. In the milder cases the difficulty in moving the joint is usually due to disuse, and to plastic exudation into the peri-articular structures, the surfaces remaining intact. But in the more marked cases the latter are more or less modified, and their free play one over the other is permanently impaired. This may be due either to actual adhesions of the articular ends of the bones to one another, or to parts of the capsular structures, against which they ought to play freely under normal conditions. In the latter case the movements are usually only limited to a moderate extent, and often not permanently, but in the former the interference with motion is usually very decided and permanent. Here we have the encrusting cartilages more or less altered or destroyed, and their place taken by plastic material, which has organised either into fibrous tissue or bone (Fig. 438). In either case, we speak of the joint being anchylosed, and divide the condition into fibrous and bony anchylosis.

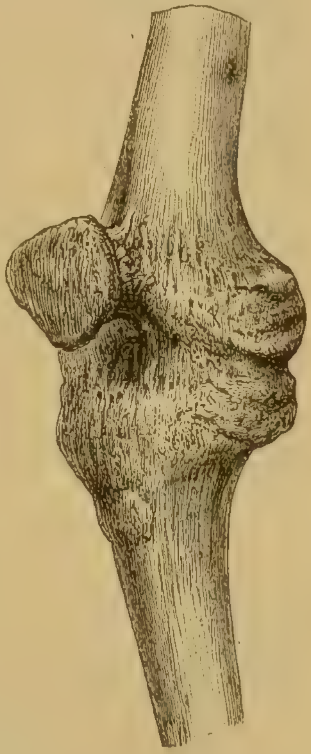
Fig. 438.—Bony Anchylosis of the Knee Joint in the best possible Position. (From Univ. College Museum.)

The first of these conditions usually results from removal of the superficial layers of the encrusting cartilage by ulceration, but may follow the complete destruction of the latter, while bony anchylosis or synostosis invariably implies the removal of the cartilages to such an extent, that bone comes into contact with bone, and in many cases it is the result of destruction of the actual bony surfaces, as well as the cartilages.

Treatment.—It is important to bear all these distinctions in mind in cases in which stiffness follows upon inflammatory joint affections. For it is quite plain that measures which would be quite proper for restoring movement in the one case would be highly improper in the other. For instance, the stiffness, the result of disuse and some periarticular exudation, can usually be soon overcome by regular passive movement and massage, followed by active use. This would be quite inadequate where there was much adhesion between the ends of the bones and the inner surfaces of the capsular structures. Here forced movements of flexion and extension are necessary, as a rule, under an anæsthetic, in order to tear through the recent adhesions and allow the unaltered cartilaginous surfaces to play freely. But such forced flexion and extension would be quite improper where there was actual fibrous adhesion between bones deprived of their natural smooth coverings. In the first place, the bond of union would be very strong, following, as it necessarily would do, upon a very severe destructive inflammation, and to tear it across would require very great force, which would be often sufficient to fracture the bones atrophied already by disuse, rather than break the bond of union. And, again, if the latter did give way, the surfaces left would be but raw fibrous material, unsuited to the movements of a joint and quite unlike the normal cartilage, and ever ready to unite again, as all raw surfaces are when in close apposition, as these would be. I have seen disaster follow where good fibrous anchylosis following upon destructive inflammation of the surfaces of a joint has been taken in hand by a bone-setter and treated in the usual way by very forcible passive movements, with the object of restoring its functions: suppuration has followed with the loss of the joint. And, if the band of union were bony, no fresh joint could be formed, and the line of fracture would be just as likely to lie outside the joint line as in it.

NEOPLASMS OF JOINTS.

Strictly speaking, the joints are rarely the seat of neoplastic tumours: that is to say, the structures actually forming the articulation are infrequently the starting-point of new growths. When such are found in joints, they have almost invariably only invaded them in a secondary way. Thus we find in some cases that an *osteo-sarcoma* starting in the head of a bone may ultimately spread towards its cartilaginous covering, and may stretch and distort the latter, but rarely perforate it. The same may be said of the other forms of *sarcoma* and *chondroma* growing from the bones and periosteum, and even indeed of those which start in the soft parts near joints.

But growths having their initial lesion in either the encrusting cartilage or the synovial membrane are almost unknown. The hypertrophy of the synovial fringes, either as the result of tuberculous irritation or the development in them of small masses of fibrous or hyaline cartilage from remnants of the same structures dating from the embryo period, might possibly be held to be an exception to this rule, but the question is whether these ought to be spoken of as neoplasms at all. Be this as it may, they are non-malignant and call for no more detailed consideration in this place.

Carcinoma is, even as a secondary affection of joints, one of the rarest of diseases. Of course, when an articulation is secondarily involved in the growth of either a sarcoma or carcinoma, the interest in the joint is merged in the more general question regarding the neoplasm, and becomes altogether subordinate to the latter. In these cases it is a question of saving life, and this can only be done in almost every instance of the kind by the sacrifice of the joint.

Cysts in and about joints.—These are few in number, and are almost invariably connected with the synovial sacs in or about the articulation. At the same time, we must recognise the fact that no part of the body appears to have immunity from invasion by the cysticercus but this; the true hydatid must be very rare as a disease of joints.

Seat.—The commonest forms of synovial cysts of the kind in question are those consisting of simple dilatation of normal bursæ from excessive secretion. Such bursæ exist in abundance in the neighbourhood of many joints, and are affected by the same causes of disease as the latter. The commonest seat, as we find them, is in the front of the hip joint, where the bursa under the common tendon of the psoas and iliacus tendons may be enlarged, the back of the knee, where the bursa between the semi-membranosus tendon and the head of the gastrocnemius is distended, or the inner aspect of the same joint, in which case the synovial sacs connected with the inner hamstring tendons are affected. Again, the bursa just above the insertion of the ligamentum patellæ may become enlarged. Finally, the proper bursæ patellæ may be affected in various ways. The ankle, shoulder, elbow, and wrist are also surrounded with bursæ and tendon sheaths, any of which may become distended.

Ætiology.—The cause of enlargement may be of at least three kinds. It may be due to hyper-secretion, the result of hyperæmia, resulting from simple local mechanical irritation, as, for instance, the pressure of kneeling on the bursæ patellæ; or it may be due to hyperplasia and hyper-secretion, resulting from tuberculous irritation; or it may be due to increased secretion, depending upon the presence in the blood of deleterious matters of various kinds. This is as much as to say that the commonest forms of bursal enlargement are all due to one or other species of synovitis.

It must not be forgotten, too, that occasionally pouches of the normal synovial membrane of joints are formed, which protrude through the capsular structures, and show themselves as cystic structures on one or other aspects of the articulation. These are not infrequently mistaken for independent cysts, and are only recognised as connected with the joint when they have been opened. This error is not so serious now as in the days before complete aseptic operation was understood. But even now it is at times a grave one to make.

Symptoms.—Most of these cysts offer, as a rule, but little difficulty in diagnosis. The slow growth and position at once suggest their origin. The total absence of all the signs of active inflammation distinguishes them at once from abscesses, and the only condition they are likely to be confounded with is a soft new growth. Such growths, however, are not common in the neighbourhood of joints, and, as a rule, are much more rapidly formed than the cysts in question. They will also be found in some cases, where very vascular, to pulsate, whereas the cysts are quite free from this symptom. Fatty tumours are sometimes difficult to distinguish from these cystic swellings when seated in the flexures of joints, but their lobulated outline, and the fact that they are movable under the skin in all attitudes of the joint, serve to distinguish them from the bursal tumours, which are less sharply defined, and in some positions of the joint are firmly fixed.

The **treatment** of these cysts is either palliative or radical. Sometimes they will become reduced in size under the action of blisters or discutients, such as iodine, combined with strapping or bandaging, and this should always be tried first. But when this course fails, they may be aspirated and strapped, and may not refill. Both these methods of treatment, however, fail in a large proportion of cases, and nothing is left but their careful extirpation by the knife. This is done with great care, so as to secure perfect asepsis and primary union of the whole wound. An incision is made, so as to expose the cyst, and this is carefully isolated by dissection in the loose areolar tissue lying outside the fibrous investment of the cyst. The latter is now separated all round and gradually drawn out of the wound, if possible, unruptured. Many of these structures can thus be removed as a whole, but in some cases the capsule is very thin and bursts, or can only be followed a certain way among the tendons, and has to be removed incompletely. This, however, will be enough in many cases to effect a cure, for the cicatrisation of the residuum may be counted on. This treatment by excision is suitable to all the cysts in connection with joints, and, properly performed, offers the best prospect of a permanent cure with the minimum of risk.

Loose bodies in joints.—Reference has already been frequently made in the foregoing pages to what are known as "loose bodies" in joints, and it becomes necessary now to examine more particularly into their clinical history, varieties, and treatment.

Clinical history.—Not infrequently patients, otherwise in excellent health, are suddenly seized with violent pain in a joint, and are immediately unable to move it in any direction. This comes on usually while the joint is being moved from the attitude of greatest relaxation to that of extension. The pain is excruciating, and sufficient, in many cases, to produce faintness, followed by vomiting. The articulation becomes suddenly fixed, and is kept so by the involuntary action of the muscles on guard against any further movement. In most cases the part appears at first perfectly normal, but sooner or later, after such an attack, it is affected with acute or subacute synovitis. (*See* page 1033.) Later, on the subsidence of the latter.

movement is again restored, and the function may be as perfect as before, until a similar attack, like the first in every way, comes on.

When a patient with such a history presents himself, one of two hypotheses at once suggests itself. Either the joint is the seat of a "loose body," or, as in the case of the knee, some part of the normal capsular appendages has become loose and has got nipped between the bones in their movement from flexion to extension. In the first case there is some mass within the articulation capable of moving about more or less freely between the bones, either attached by a pedicle or actually separated from all connection with surrounding structures, and capable of slipping to any part of the cavity. In the second instance, one or other of the semi-lunar cartilages is loosened at its capsular attachment, and moves in between the joint surfaces proper (Fig. 420). As a consequence of either of these two accidents a great strain is put upon the lateral ligaments as the limb is brought into the straight position, the long leverage of the bone with the foreign body as its fulcrum forcing the attachments of the ligaments apart. (*See* page 1015.)

The **diagnosis** between these two conditions is sometimes a matter of considerable difficulty. When the attack is due to a loose body of any size, however, the latter may often be felt by the patient or surgeon when the synovitis has subsided. In the knee it may be made out with the fingers, slipping about usually in one of the pouches above the patella, as a round or nodulated body, from the size of a pea to that of a filbert. It is usually very difficult to restrain, and while manipulated will often pass with a slip into some part of the joint in which it can no longer be felt by either the patient or surgeon. Flexion and extension will often again dislodge it and bring it within reach of the fingers, but not always; and it may be lost, so to speak, for days or weeks, only giving evidence of its presence at last by a fresh attack like the first.

When a semi-lunar cartilage, on the other hand, becomes loosened from its capsular attachments, and moves in between the femur and tibia, although the symptoms are very much the same as in the other case, no projecting body can be felt at any time, but on the contrary a depression may be discovered by careful comparison with the opposite side just above the articular border of the tibia. The patient's sensations may also guide us in some cases. He may be conscious of something moving about in the joint, even before he is able to localise the loose body with his fingers, and when there is no pain to guide him.

Varieties.—There are several varieties of loose bodies now recognised. They may consist either of hyaline cartilage, of fibro-cartilage, of bone, or of fibrinous material. The first two forms are derived from the synovial fringes of the joint, and are produced, as a rule, from remnants of embryonic tissue which have been left behind, as it were, in the development of the sac. Later, these remnants of embryonic tissue have grown with stored-up energy, and have formed more or less pendulous tags projecting into the joint. In this state they may remain for an indefinite time, or become detached and float loose in the joint. Similar pendulous tags may be formed in the synovial fringes by any form of chronic inflammation, and are doubtless in some cases tuberculous. The bony masses, on the other hand, are sometimes produced from nodules at the borders of the bones due to rheumatoid arthritis (page 1053), which have broken off and been set free. Again, they may be derived from the actual normal surfaces of the joint by injury, a fragment being chipped off, showing on one surface the line of fracture and on the other the normal encrusting cartilage. Finally, they may be produced from the surfaces of the articulation by a process of what has been described as "quiet necrosis," that is, the separation of a fragment of sclerosed bone from that underlying it by a rarefactive process like that of rarefactive osteitis, but unaccompanied by any suppuration. (*See* page 887.) The fibrinous bodies are probably formed from the fibrin of blood extravasated into the joint, as the result of injury, and rolled into pellets by the movement of its surfaces.

The **treatment** of any or all of these loose bodies is practically the same in most cases.

Sometimes they will form adhesions for themselves in some part of the synovial pouches out of the way of any particular pressure, and if so give rise to little or no inconvenience. This adhesion may be encouraged by bandaging the joint firmly when the loose body is giving no trouble, and is, therefore, presumably out of the way. If such a bandage, preferably an elastic one, be worn for a long period, the final fixation of the body may be accomplished and a more or less permanent cure result.

But these methods are very uncertain, and the patient has, as a rule, no time sufficient to give them a fair trial. Fortunately, in these days of aseptic surgery the risks of removing these bodies once and for all from the joint are very small, and this is the course which is approved by most modern surgeons. The foreign body is simply fixed in one of the lateral synovial pouches by the finger and thumb of the operator, and a bold stroke is made through all the structures with a knife until it is reached. As a rule, it will then slip out easily through the wound, aided, perhaps, by a little extra pressure with the finger and thumb. Sometimes several loose bodies may be thus squeezed out of the same opening. It should be a rule, however, with all operators never to make an incision into a joint to remove a loose body unless the latter can be felt and held *in situ.* If this is not adhered to, it may be impossible after the opening is made to find the foreign body at all. Again, there are cases in which, though the hard mass may appear to move freely about, it may be really pedunculated, and may not slip out readily on the incision being made. In this extremity it must be carefully seized in forceps, and its pedicle must be divided carefully with scissors. It is better in all cases to avoid putting the fingers into the joint, or indeed anything that can be avoided, lest the cavity be infected

When the body or bodies have been removed, the opening should be accurately closed from end to end, all excess of synovium having first been squeezed out. No drainage is required in cases operated on with strict asepsis.

The treatment of loose semi-lunar cartilage which has slipped in between the surfaces of tibia and femur is not so easy a matter as in the last case. Sometimes, it is true, we may achieve a practical cure by rest and careful support of the joint for long periods, while at the same time the patient is generally strengthened by tonics and fresh air, and the joint locally by cold douches. For it is a well-known fact that this displacement is usually met with in anæmic individuals of slack fibre. But these means are frequently inadequate to prevent recurrence of the condition, and with each recurrence the tendency to the reproduction of the trouble is greater. Indeed, in some instances where one of the semi-lunar cartilages has slipped bodily in between the condyles and lies crumpled up there, as was the case in the joint from which Fig. 420 is drawn, nothing short of opening the joint and readjusting the cartilage would have any chance of success. But even in less marked instances of displacement the frequent recurrence of pain and synovitis in spite of supports of various kinds is often felt by the patient to justify him in running the small risk of having the fibro-cartilage secured in its normal position by operation. When the latter is determined on, of course every precaution is taken to secure perfect asepsis round the field of operation. The patient is also kept at rest for some days, especially if traces of synovitis remain from one of the attacks. Indeed, until all trace of the latter has gone, no operation should be undertaken. Then an incision running obliquely across the line of the displaced body is made through all the structures overlying its outer border. When this is exposed it is caught up with three or four stitches, and secured to the capsule of the joint in its normal position. If it be totally displaced, it is necessary to open the joint more freely, then, by alternate flexion and extension, the crumpled cartilage may be brought into its normal position, and be secured as just described. No drainage is required, and the skin wound is completely closed by sutures at once, the limb being put up on a back-splint until healing is complete.

HÆMOPHILIA AFFECTING JOINTS.

Hæmophilia, for the detailed description of which the reader is referred to page 376, is, without doubt, at times the cause of serious joint disease. When affecting the joints, it is seen in the form of effusion of a large amount of blood into the synovial sac following injuries to the part, often of a very trifling character. The first effect of this effusion is simple distension of the pouches of the joint with blood, the symptoms of which have already been considered. (*See* Hæmarthrus, page 1034). Certain secondary changes, however, are also observed in some cases, at all events, which may interfere with the functions of the articulation permanently. These are subacute inflammations of the synovial membrane and cartilage, leading to their degeneration. The synovial membrane, in other words, becomes thickened and roughened by exudation under the irritation of the clotted blood, part of which becomes apparently organised. The cartilage, too, becomes fibrillated and roughened, and is thus thinned until, in some cases, the underlying bone is exposed. If, in these circumstances, the clot and exudation become organised, adhesions are formed which seriously interfere with the movements of the articulation.

The *symptoms* of the condition are common to those of effusion into the joint of blood from any cause (*see* Hæmarthrus, page 1034), and the sequelæ are the same.

The *treatment* consists in cold applications in the first place, and subsequently of strapping or elastic support of the whole joint with elastic bandages. Combined with this, massage may do much good in helping to remove the clot. On no account ought any operation—not even aspiration—be done for the removal of the latter.

The *prognosis* in ordinary cases, as far as the joint is concerned, is good, but as to the general condition, we know of no treatment that cures it.

NEURALGIA OF JOINTS.

Like other highly-sensitive parts of the body, joints are occasionally, though on the whole rarely, the seat of real pain that does not appear to be associated with any tangible structural lesion. The exact pathology of the phenomenon is still unknown. Whether it be due to some alteration in the peripheral portion of the nerves of the part themselves, or to morbid conditions in the central nervous system, will probably for a long time be a moot point. But without attempting to settle this question, we may now consider the clinical facts relating to these neuralgias, and contrast them with those recognised as factors in true structural change.

Symptoms.—These pains are usually noticed in joints which at one time or another have received some injury, such as a strain or a blow, but which have recovered all their functions completely. They are mostly of a keen, sharp character, or are described as "burning." They are not associated with any throbbing or sense of heat actually in the part, nor is the local temperature found to be altered. Occasionally the pain is lancinating, being intermittent; sometimes it is continuous. It is generally met with in those who for one reason or another have run down and become anæmic, and is most felt towards evening after the work of the day has used up the patient's surplus energy. Again, the pain is often most severe during bad weather, which depresses the vital energy. Patients affected by these pains are often hereditarily neurotic, and have been lately through the same experiences which lead up to the nervous mimicry of joint disease. (*See* page 381.) They differ, however, from the latter class of patients, inasmuch as they are real sufferers from a distinct pain which is anything but imaginary.

The condition is distinguished from structural change by the absence of all objective signs of disease in the joint, such as swelling, heat, altered colour, or deformity, and by the character of the suffering. Such patients, too, will use the joint affected as well as the other, and even may experience a sense of relief in so doing. After rest of the whole body, however, the pain is usually relieved, as also after the enjoyment of food and sleep.

Treatment.—The treatment of this neuralgia will be general and local. In the first place every means must be adopted to strengthen the whole body. All exhausting work must be given up, and the patient must take abundance of sleep and regular exercise in fresh air, with plenty of suitable digestible food. Stimulants are to be avoided, but tonic medicines, especially nux vomica and iron, will do much to assist the digestion and restore general tone.

Locally, blisters or rubefacients are useful. But in many cases nothing is so efficacious as the application of the actual cautery in the form of the hot button over the seat of pain. The part should also be protected against exposure to extremes, either of heat or of cold, by coverings of flannel or wool. Friction with liniments containing chloroform and belladonna is often efficacious. In many cases, too, a regular course of massage may be recommended where other means fail.

AFFECTIONS OF THE HIP JOINT.

This joint claims our first attention, not only on account of its size and importance for progression, but also on account of the frequency with which it is the seat of disease.

Simple acute synovitis of the hip.—This simplest form of acute disease is not as common, however, here as in other joints, owing to the fact that the articulation is protected, in a measure, against external influences by its great depth among the muscles. Nevertheless, it is occasionally seen in its purest form as the result of overstrain or exposure.

Symptoms.—The subjective symptoms are heat and stiffness, followed by throbbing pain, referred to the joint itself, and also to the knee. This latter symptom is explained by the connection between the two joints through the obturator nerve. Tenderness and pain are most marked over the front and back of the joint, and cause limping very early, and, later, complete inability to use the limb.

Objectively, swelling is also noticed in two situations—*i.e.* over the front and back of the capsule—and is due partly to effusion into the synovial sac, and partly to œdema. Redness is rarely seen, on account of the depth of the part beneath the surface. Next to this, the most striking symptom is the position of the thigh. If the patient be young, and the inflammation recent, strong flexion, abduction, and eversion will be present. The causes of this displacement are the following:—In the first place, we must remember that the synovial membrane is now inflamed, swollen, and tender, and that hyper-secretion is taking place. In the fully extended position of the limb the parts of the capsule and ligaments most on the stretch are those on the anterior aspect of the limb. In the inflamed condition, these parts of the capsular structures are most in need of relaxation. As a consequence, the patient involuntarily relaxes the front of the capsule by flexion, in order to relieve the pain. As the Y-ligament is in close relation to the anterior part of the joint, it also requires to be relaxed in all its parts, as a whole, by flexion, its inner arm by eversion, and its outer by abduction. The posterior part of the capsule being thin and loose, yields readily to these movements, until the tension is distributed evenly all over the capsular structures, when further displacement is unnecessary. In short, the patient places the limb involuntarily in the position of the greatest ease for himself, and thus it is fixed by the muscles, so that no rubbing of the surfaces may increase the suffering. (*See* Fig. 443.)

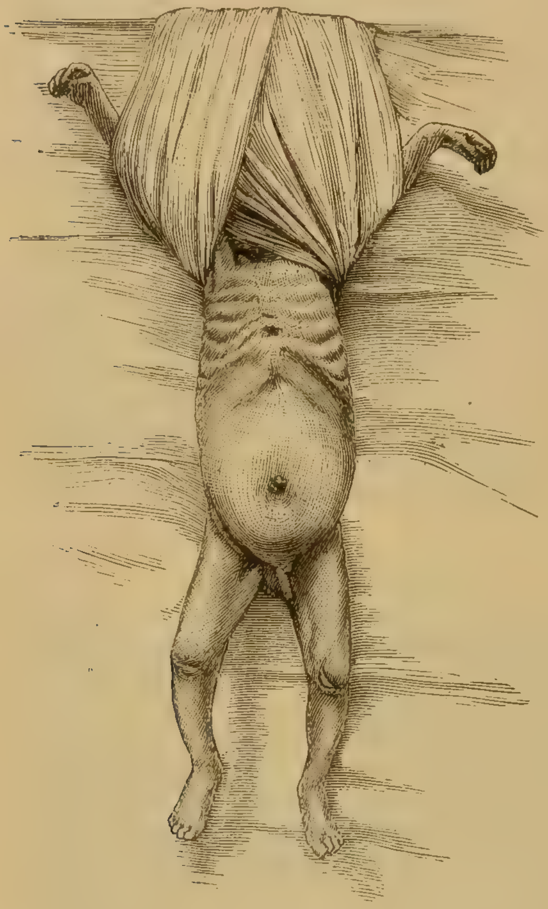
Fig. 439.—Position of Hip Joint when forcibly distended with Water.

That this position of the limb is assumed in order to reduce the strain upon inflamed parts, and not, as was formerly thought, as the result of reflex irritation of muscles, is shown to be almost certain by experiments on the dead body. Figs. 439 and 440 are from photographs of the body of a child with healthy hip joints. Into one of the latter (right) I injected water with a screw-piston through a hole bored in the pubic bone. As the water was forced into the joint the thigh rose steadily into the position of flexion, abduction, and eversion (Fig. 439), and when the water was allowed to flow out again the limb returned to its normal position. In this case, of course, the distending force was felt on all aspects of the joint alike, but the looser posterior part yielded first, the strong anterior unyielding portion could only be relaxed by flexion, abduction, and eversion. The only difference between what takes place in this case after death and when the joint is inflamed during life is that, in the latter case, the patient relaxes the painful parts, while in the former the distension is the mechanical cause of the relaxation of the Y-ligament and capsule. The principle is the same, and explains the whole phenomenon without invoking reflexes. This experiment was originally made by Bonnet many years ago, but its correct interpretation was not forthcoming for a long time afterwards.

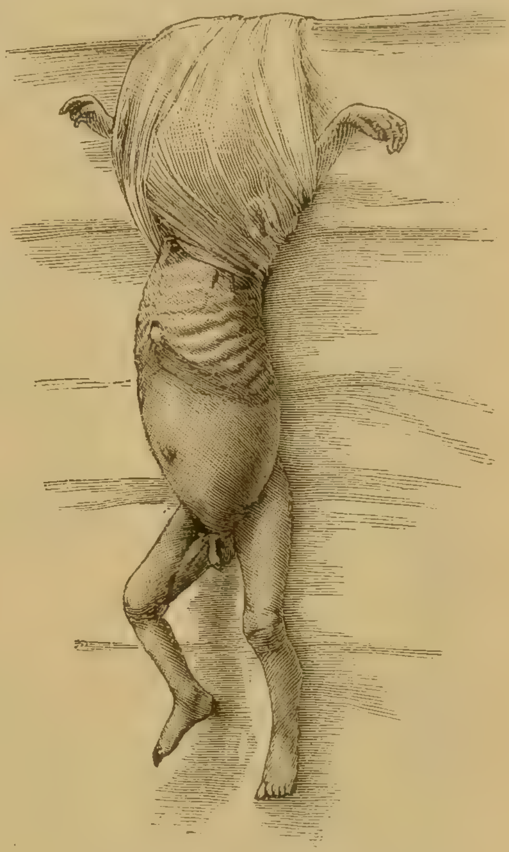
Fig. 440.—Position of Hip Joint when forcibly distended with Water.

A patient in this condition will generally lie upon the affected side, with the leg flexed upon the thigh; but, if placed upright, he will lower the sole of the foot, and rest it on the ground. This necessitates his tilting the pelvis *downwards* and *forwards* on the affected side to make up for raising of the leg, due to the flexion and abduction of the thigh. In this position, if the pain be great, he will probably rest his hand upon the affected thigh just above the knee, with the double object of steadying the limb and of throwing the weight of the body directly upon the leg, without the interposition of the hip. If placed upon his back in bed, he will probably support the limb with his hands in the flexed attitude. From this, where the pain is even slight, no force will move it, and any attempt to bring the knee down to the bed will be followed by much arching of the pelvis, but no alteration of the angle of the latter with the axis of the femur, whether the angle of flexion or abduction. It may, of course, in many cases be brought parallel with its fellow, but only by movement of the pelvis downwards on the affected side. This gives the whole limb the appearance of being lengthened, which is deceptive, but ought never to mislead the experienced surgeon.

The **treatment** of simple acute synovitis has already been considered (page 1034), and need not detain us here. It is only necessary to consider now how it can be best applied to the hip joint.

Rest, which is the great desideratum, is best carried out in this case by confinement to bed, and extension of the limb by weight and pulley. The latter may be attached to the leg, either by means of a well-padded leather collar or, better still, by strips of plaster running from the knee to the ankle. This distributes the strain over the whole leg, and is less likely to be followed by pressure ulcers, etc. The action of continuous extension is to tire out the muscles and steady the limb, and under its influence the flexed and abducted thigh may often be brought into a straight line in a day or two. But it ought to be continued for a much longer time, as a rule, otherwise the old position will be soon resumed. If for any special reason it be considered undesirable to confine such a patient to bed for longer, the weight extension may be replaced by a Thomas's splint, single or double. The latter (Fig. 450) is to be preferred, as giving much more thorough fixation to the joint, and correcting the flexion and abduction much more efficiently. In such a splint a child can be carried about without any disturbance of the limb, and may enjoy all the benefits of fresh air and change. It is also much easier to provide for all the exigencies of defæcation and micturition with such an appliance than with any other known to me.

No attempt forcibly to bring down the limb to a straight line at once under chloroform should ever be made, as the strain on the inflamed parts thus brought about is only productive of evil; it should be gradually straightened.

Local depletion with leeches, and the use of hot fomentations can also be carried out in such early simple cases with much advantage. And it should always be borne in mind that in unhealthy children and adults any attack of simple synovitis, if neglected, is apt to be followed by the tubercular form of disease. In all debilitated individuals, therefore, the constitutional condition should be as carefully attended to as the local. Tonics, cod-liver oil, and good food should be freely given, and every means taken to render the hygienic surroundings of the patient as perfect as possible.

And when all pain and swelling in the joint have passed off, great caution should be used in overcoming the resulting stiffness, lest the inflammatory condition be lighted up again by too early active movement. This should be preceded by massage and gentle passive motion for some time before the patient is allowed to make use of the limb.

Simple subacute synovitis of the hip is often met with as the result of the same influences that have produced the acute form. The symptoms are precisely the same, but less marked; and the treatment is also the same.

Acute septic synovitis of the hip is by no means uncommon as the result of one or other form of pyæmia. The latter condition is met with, as already stated, after absorption from foul wounds, either on the external surface of the body or on some of its internal surfaces in the course of such diseases as scarlatina, measles, typhoid fever, etc.

Symptoms.—In such a case the affection usually runs a rapid course with all the symptoms mentioned above shown out in a very marked degree. The distension of the joint is here so quickly produced that the capsule is apt to be soon burst and the inflammatory matter to be extravasated into the soft parts around. If at the same time the limb have been left to itself, the position of flexion becomes extreme, and the head thus thrown upon the posterior part of the capsule, which is softened by the inflammation, is ready to slip out upon the dorsum ilii, especially as the softening soon extends to the inner and outer aspect of the capsule, and abduction is thus converted into adduction and eversion into inversion. Such a case occurred some years ago in my practice at University College Hospital. A patient had severe typhoid fever, and at the end of some weeks the hip joint assumed the characteristic appearance of acute inflammation with rapid distension. This was followed by flexion, adduction, and inversion, and then by dislocation of the bone during movement in bed. This dislocation "by distension" of Volckmann was followed by rapid subsidence of the inflammation, and when the boy was admitted into the hospital nothing but the dislocation remained. This I reduced with the greatest ease by Bigelow's method, and then found that the surfaces were comparatively intact as indicated by the smooth movements of the head of the femur in the acetabulum. The limb was kept straight, and the head remained in position, the boy making an excellent recovery without any shortening. I saw him many months after with a stiff but otherwise healthy joint.

It is a remarkable fact that in these cases of acute septic synovitis in pyæmia, early evacuation of the inflammatory contents of the joint is often followed by rapid recovery, as in this case, and should always be provided for either by aspiration or free incision and flushing out before the surfaces of the cartilages and synovial membrane have become extensively altered by ulceration.

The **treatment** is early incision and, if necessary, washing out of the joint with sterilised water or with some strong antiseptic, preferably the former. At the same time the limb must be kept in a straight line, either with weight extension or splint. For, if left to itself it will rapidly assume the position which lends itself to dislocation, and this cannot always be so happily replaced as in the case just mentioned. Local antiphlogistics are in this form of disease of but little use, except in relieving pain. But those remedies which maintain the general strength and help the system to combat the poison which is locally affecting the joint surfaces are of the utmost value.

The **subacute and chronic form of septic synovitis** differs only in degree from that first described. Here the distension of the joint is slower, and all the other symptoms are much milder. The disease is, however, equally destructive, and may lead to complete disorganisation of the articulation if not taken in hand soon.

The **treatment** consists in free incision of the joint and washing it out thoroughly. It is remarkable how well such wounds will subsequently heal when we consider that the edges of our incision are bathed in the septic pus which we let out. I have lately had such a case where the joint was completely disorganised by chronic pyæmic disease, the result of some infection before the patient entered the hospital. Here after free incision and removal of much diseased bone, including part of the acetabulum and the head of the femur, which were carious, the wound rapidly healed, and the patient, a man of about thirty, is now walking about with a very useful limb. This is all the more re-assuring, as such operations on adult hips for other forms of disease, such as the tubercular, are usually followed when they heal by very great weakness in the limb.

Syphilitic disease of the hip is not commonly described, except in that form of congenital or infantile disease of the epiphyses already discussed as far as is necessary (page 1065).

Tuberculous disease of the hip.—This is one of the most common affections in surgery. **Points of origin.**—It is most frequently met with at the two extremes of life, among the very young and the very old, though not unknown among those of middle life and young adults. It has its starting-point either in the head of the femur, the acetabulum, or in the synovial membrane. Exact data upon the relative frequency of its appearance in these situations are wanting, but as far as our knowledge in this direction goes, it is believed to be most frequently found to start in the *femur*, next in the *synovial membrane*, and more rarely in the *acetabulum.* In very young patients it is more likely to start in the bones, in adults in whom the epiphysial growth is completed it finds its most frequent starting-point in the synovial membrane. When we speak here of the head of the femur, it should be understood that there are two spots in which the primary infection is likely to take place. The commonest position is undoubtedly in the epiphysial line, *i.e.* at the junction of the head and neck. But it may also start in the growing tissue underlying the encrusting cartilage of the head of the femur. And when it has its origin in the acetabulum, the morbid process begins in the young embryonic tissue at the junction of the three bones in the centre of the hollow. When the synovial membrane is first invaded, it is, frequently at all events, in the loose folds which surround the ligamentum teres. From either of these points the whole of the joint may be invaded, but from each the disease can, on the other hand, spread outwards without invading the surfaces, and may form an abscess outside the capsule, which points externally, in some cases, through the skin, leaving the joint proper practically intact. This is a most important fact to bear in mind, for with improved means of diagnosis such cases can be separated from those in which the joint itself is invaded, and thus many patients may be spared removal of parts of the bone and synovial membrane, who formerly would have had a more or less complete excision performed upon them.

Diagnosis in the early stages.—We must now endeavour to study each of these forms of disease separately, in such a way as to be able, if possible, to diagnose them one from the other.

The *femoral variety* of tuberculous hip disease in the child is first evident to the naked eye in the form of a small focus of inflammatory deposit, situated usually in one or other part of the plane of growing tissue between the neck of the femur and the epiphysis of the head. Sometimes this focus is at the periphery of the plane (Fig. 441), but more usually it is towards the centre. In the middle of this inflammatory patch, as we see it, fatty degeneration has usually advanced to a considerable degree, so that a caseous abscess exists to a greater or less extent. Now, it depends upon the situation of this centre of disease whether the joint will be early infected as a whole or not. If the caseous mass lie towards the periphery of the bone and spread outwards, it very soon involves the periosteum, and on perforating this the tuberculous material gains access to the synovial sac, and the whole inner surface of the cavity is involved in the morbid process. Again, if the caseous focus lie directly in the centre, and grow in all directions evenly, it may eventually separate the head completely from the neck, and the former, lying loose in the joint, cut off from its vascular supply through the neck, plays the part of a sequestrum in a generally inflamed joint, which soon becomes hopelessly disorganised. But when there is a central focus this is not invariably the course of the disease. Sometimes it heals up under appropriate treatment, and the caseous material cretifies or becomes absorbed. But even when the process advances in extent, it does not always do so towards the periphery, but may gradually move outwards down the centre of the neck towards the trochanter, and finally burst through the periosteum somewhere outside the attachment of the capsule. If an abscess be formed in such a case it is clearly extra-articular, and all the time the movements of the joint are intact.

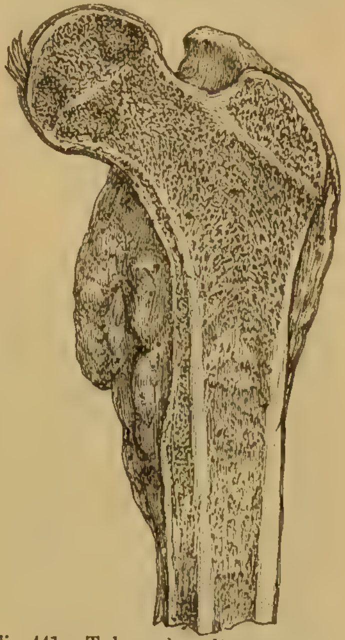
Fig. 441.—Tuberculous Disease commencing in the Head of the Femur at the Epiphisial line (University College Museum).

These forms of femoral disease must be carefully studied, and at all times kept in mind if we are to avoid unnecessary surgical interference with this joint, and to give anything like an accurate prognosis. The diagnosis between these varieties is by no means easy, but we may arrive very near the truth in many cases by observing the following points :—The mode of onset is the first point. In this case it is usually very gradual, coming on in delicate children, as a rule, under eight years of age. The first thing noticed is a slight limp, the child being quite unconscious of pain. This involuntary limp—"unfreiwilliges Hinken"—is doubtless due to the fact that pressure upon the damaged head of the bone at this early stage of the affection produces a sense of uneasiness, hardly amounting to pain, but sufficient to lead the patient to rest as short a time upon the limb as possible; there is, therefore, a short step and a long alternately.

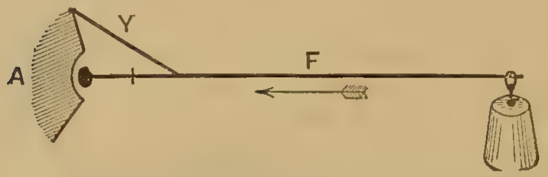

Fig. 442.—A, Acetabulum; Y, ilio-femoral ligament and strong anterior part of the capsule; F, femur.

If the joint be examined at this early stage, while the patient lies on the back, it will probably be found to move in all directions pretty freely without pain, thus indicating that the surfaces that play one upon the other are so far unaffected. It is only when forcible passive movements are made in such a way as to put the ligaments upon the stretch, and so force the head against the acetabulum by leverage, that pain is complained of. Or, again, if the flexed knee is struck in a direction towards the pelvis, or the trochanter pressed inwards towards the acetabulum. The pain, when the limb is over-extended or abducted, is thus explained. The Y-ligament prevents over-extension, and when the femur is brought into this position the ligament acts as the fulcrum of a lever represented by the femur, and when the latter is forced backwards the head is pressed into the acetabulum, and the diseased spot is subjected to strain (Figs. 442 and 443). The same may be said of abduction when carried to an extreme. It is to prevent such pressure that the patient involuntarily flexes, abducts, and everts the limb—*i.e.* to relax the most powerful and tense ligaments (Fig. 443).

Now, if *synovitis* were present, even though we were unable to detect the usual signs of inflammation, we should find the joint fixed, to prevent any rubbing of the inflamed surfaces one upon the other, and any passive movement would be resisted voluntarily, with expressions of acute suffering. The characteristic position of acute synovitis would also be assumed (Fig. 440). As long, therefore, as these two conditions are distinct, these signs will serve to distinguish the one from the other.

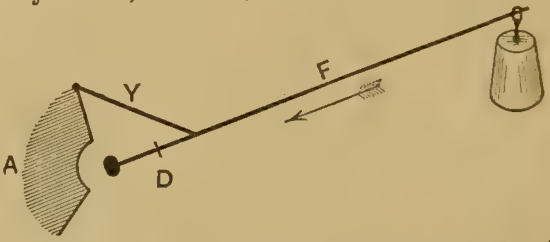

Fig. 443.—A, Acetabulum: Y, ilio-femoral ligament and strong part of the capsule F, femur; D, diseased area.

But to separate the femoral from the *acetabular variety* is quite another matter, and is sometimes impossible. It is, however, not nearly so important to distinguish these varieties from one another as in the last case. Careful palpation of the back of the acetabulum with the finger in the rectum will in some cases aid us here, for we may make out a soft patch or, at all events, a tender spot in the bone. Otherwise, the position of the limb and the other symptoms, objective and subjective, will be the same.

Diagnosis in the advanced stages.—When the disease has begun in the synovial membrane or in either of the bones, and has advanced to any great extent, the appearances will be quite changed, as already stated. Here, in addition to swelling, heat, tenderness, and perhaps redness, there will be increased flexion, in order that the tender capsule may be further relaxed, and as the latter becomes softened, abduction will be converted into adduction, and eversion into inversion. In this latter state the patient will probably lie upon the sound side, with the knee of the affected limb thrown across its fellow, and resting upon the bed. And as he tries to bring the adducted limb parallel with its fellow, the pelvis on the affected side must be carried upwards, inasmuch as the angle which the femur forms with the transverse axis of the acetabulum cannot be altered without painful movement of the surfaces one upon the other. The diagrams (Figs. 444, 445) will make this clear. A B is the axis of the spine; C D that of the pelvis in its broadest measurement from side to side; B C E the angle of the affected adducted joint. If the limb C E is now to be brought parallel to its fellow without altering the degree of the angle B C E, the axis C D must be altered (Fig. 445). This will give the affected limb the appearance of being shorter than its fellow, though really it often remains of the same length to careful measurement. The movement of the transverse axis of the pelvis can only be accomplished by lateral flexion of the spine. But at the same time the curves of the lower part of the spine will be further changed by the efforts of the patient to bring the limb out of the position of extreme flexion into one more in the same plane with its fellow. This is illustrated in Fig. 447, in which S represents the axis of the spine, and O P that of the pelvis antero-posteriorly.

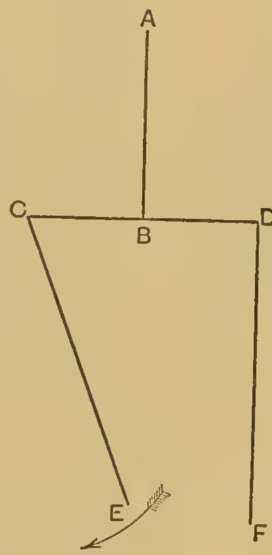

Fig. 444.

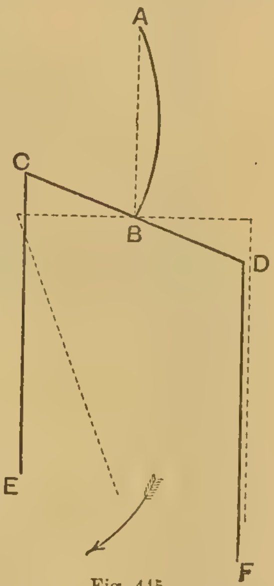

Fig. 445.

Now, if F A P be the angle of flexion of the femur, and the latter be brought into the same plane as its fellow without altering F A P, this can only take place by the bending forwards of the spine, at O, and the consequent production of lordosis, as seen in Fig. 446. (*See* also Fig. 448.)

After these changes in position as the disease goes on, we may have a new factor in the actual loss of substance in the bones where they touch. If this take place to any considerable extent while the limb is flexed, the border of the acetabulum above and behind becomes eroded, and the head of the femur at the same time loses part of its tissue. The two flattened surfaces thus produced are then apt to slide one upon the other, being no longer restrained by the cup and ball arrangement of the joint, and the head of the femur slips on to the dorsum ilii, where it may undergo further destructive changes (Fig. 449).

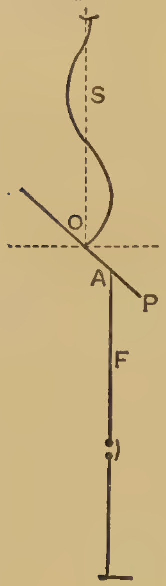

Fig. 446.

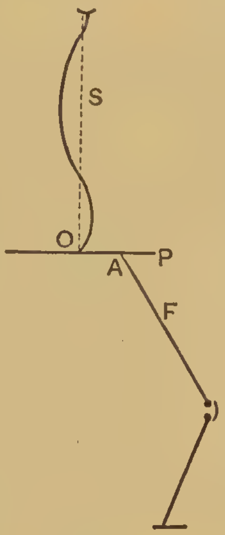

Fig. 447.

The three stages of hip disease. — There are then obviously three different positions of the femur in relation to the pelvis. These are sometimes spoken of as three different stages of the disease, and at all events correspond to important differences in the condition of things within the joint. In the first place we have the position of flexion, abduction, and eversion; secondly, of flexion, adduction, and inversion without real shortening; and thirdly, of flexion, adduction, and inversion, with erosion of bone, dislocation, and real shortening. These stages are sometimes spoken of as the first, second, and third degrees of hip joint tuberculous disease, and there is a certain amount of convenience in employing the terms.

The stages, it will be observed, correspond with—(1) inflammation without softening of the ligaments; (2) inflammation with softening and partial destruction of the ligaments; (3) the same with destruction of bone with or without dislocation, which merely depends upon whether the limb has been kept in the extended position during the destructive changes within the joint or has been allowed to flex, adduct, and invert. As long as it is kept straight, the head is more or less retained by the acetabulum, though the latter may be extensively diseased. But if it have been allowed to assume the flexed and adducted position, in which the head is normally half out of the socket, it requires but little erosion of the brim of the latter behind and above to allow of the head slipping out on the dorsum ilii (Fig. 449). In this last form in which there is actual loss of substance in the femur and acetabulum, we have a variety of "*dislocation by disease*" (Volckmann), which contrasts strikingly with that already described as "*dislocation by distension.*" (*See* pages 954 and 1079.) The variety met with in the cases we are now considering is the "*dislocation by destruction,*" and we can hope for but little benefit from reduction even if it be still possible to effect it. For the flattened head would never rest upon the eroded acetabulum for long; there would be nothing to hold it there, and as long as it did lie in its old position it would be placed in an unsatisfactory condition as regards repair. For the inflammatory products of the bone and synovial disease would be retained between the two surfaces, and if abundant would prevent all repair; and as there are also often sequestra of varying size mixed up with these products, we can well understand the difficulties that exist in the process of healing.

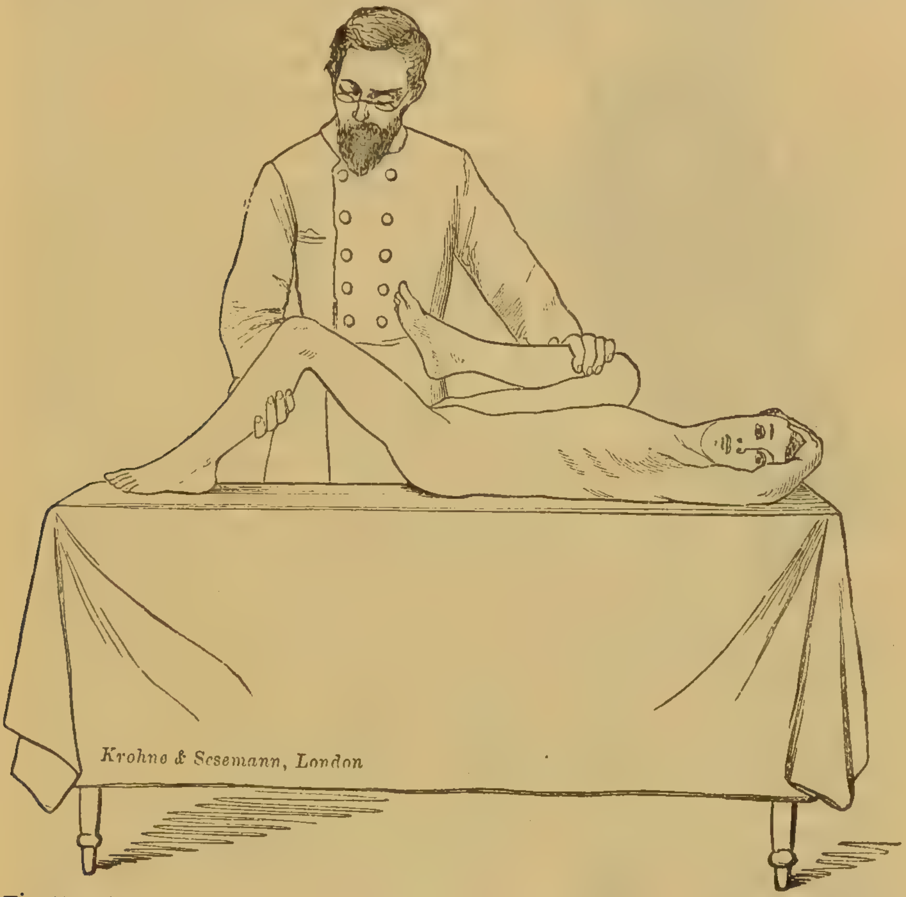

Fig. 448.—Case of left Hip Disease, in which the degree of flexion of the affected thigh is demonstrated by flexing the sound thigh on the abdomen, this movement entirely correcting the lordosis.

Treatment of hip disease.—The treatment of these several varieties of hip disease will vary in many respects according to the stage of the tuberculous process. When the synovial membrane is but lightly affected, and the patient's strength is fairly good, there can be no doubt that a complete *restitutio ad integrum* is possible if proper means have been taken to keep the surfaces at rest, and to improve the general powers by generous diet, fresh air, tonics, and cod-liver oil, as already described (page 359). The best means of combining rest with fresh air is to furnish the patient with some appliance which will fix the joint without confining him to bed or the house. After a long experience with these cases, I am inclined to give the preference in the case of young children to the double Thomas's splint, which is shown in Fig. 450. This secures the limb in the straight position, and immobilises it completely, and in such an appliance the young patient can be carried about easily and enjoy out-of-door life. But where a vicious position has been allowed to occur, it will be best in most cases to confine the patient to bed for a while, and to extend the limb by weight and pulley until it is straight, and then put on the double Thomas's splint. This must be kept in use as long as there is pain and tenderness in the joint, or until other changes have occurred which render operative interference necessary.

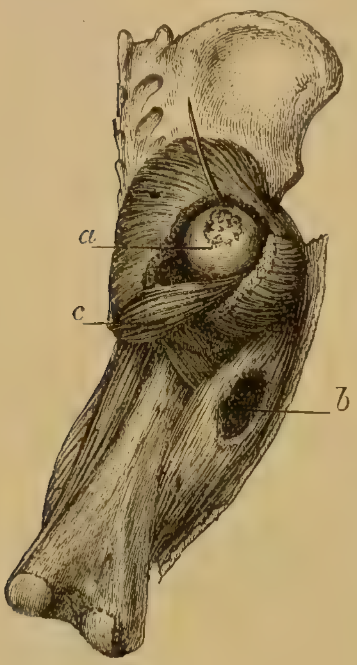

Fig. 449.—Destructive Disease of the Hip Joint.

a, Head of femur dislocated backwards; *b*, abscess extending down the thigh; *c*, obt. internus. (From University College Museum.)

In the case of older children and young adults who can manage to walk with a pair of crutches, the single Thomas's splint, which immobilises the affected limb and yet leaves the sound one free for progression, is an admirable contrivance (Fig. 451). Combined with a patten under the boot of the unaffected foot, it enables the patient to take exercise without injury. It requires, however, to be very carefully applied and to fit perfectly, othewrise it has a tendency to slip to the outer side of the thigh, and in this position fails to counteract the tendency to either flexion, abduction, or adduction. And it very often occurs that if these individuals are left to themselves, as out-patients, they neglect to secure the splint as the bandages loosen, and come back to us with the apparatus on the

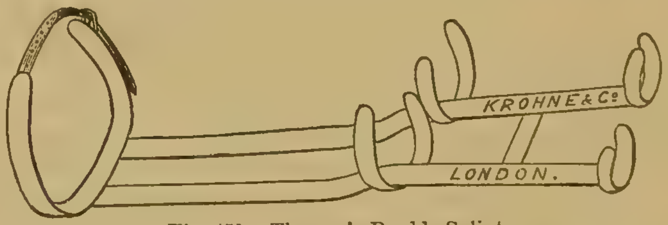

Fig. 450.—Thomas's Double Splint.

outer side of the thigh and perfectly useless. For this reason I have for years been in the habit of employing the double splint which cannot slip laterally, and which has, especially in the case of young children, many advantages, which are a set-off against the drawback that it prevents the use of the sound limb.

When there is formation of abscess.—When synovial disease is running an unfavourable course in the hip, we are not long left in doubt as to the gravity of the condition. The tuberculous process cannot gain much ground here without attacking the cartilaginous surfaces and the bones, and without forming caseous abscesses. When the latter are evident we may almost take it for granted that the bones have suffered, and consequently that whatever the issue of the case may be, the *functions* of the joint will be quite destroyed. Such abscesses when they have attained to any considerable size will generally soon make their way to the surface, and become evident under the skin. The spots at which this pointing is going to take place will vary with the treatment that has been adopted. As a rule in my practice where the joint has been well supported, especially behind by a Thomas's splint, the tuberculous débris finds its readiest way to the surface on the anterior aspect of the joint. It passes, as a rule, between the gluteus minimus and rectus tendon, and then between the tensor vaginæ femoris and sartorius, to form an abscess in front of the thigh. Sometimes, however, though rarely, it works its way through the thin part of the capsule behind and presents somewhere in the gluteal fold, from whence, if neglected, it may burrow in any direction. Again, inflammatory matter pent up in the joint may gradually penetrate into the bursa underneath the psoas muscle as

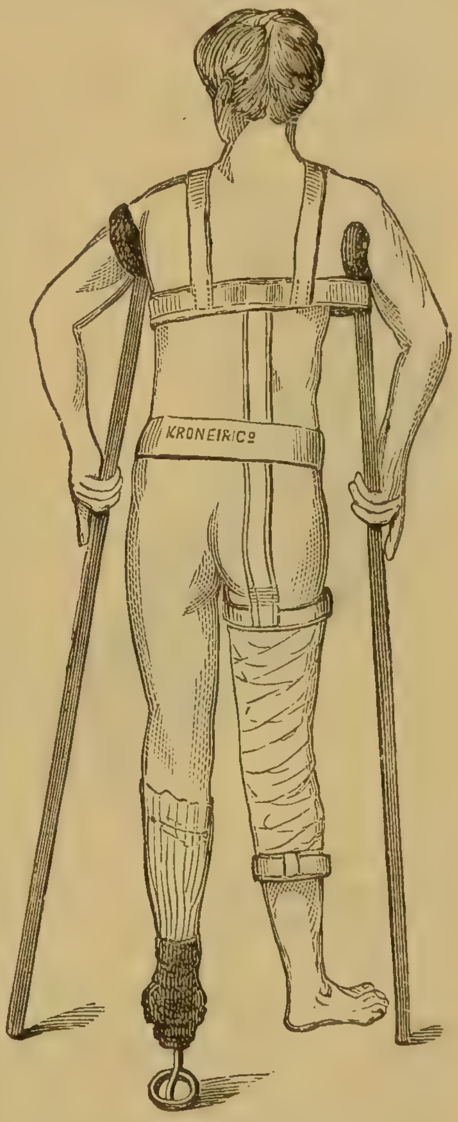

Fig. 451.—Thomas's Single Splint.

it runs over the front of the femur, and may then infect the whole of its synovial lining, forming a large collection, which can be traced into the pelvis under Poupart's ligament. Finally, the contents of the joint may make their way through the anterior part of the capsule, and pass down the inner part of the thigh underneath the adductor muscles, following in some cases the course of the circumflex arteries.

All these abscesses may start originally from primary disease in any of the three situations mentioned—*e.g.* femur, acetabulum, or synovial membrane—but, as a rule, depend upon involvement of all the structures of the joint, and when present may be generally held to indicate the latter. There are, however, abscesses of small size, which may be derived from disease, limited to one or other of these three components of the joint, without involving the others. For instance, a small localised deposit of tubercle in the synovial membrane may caseate slowly and soften the capsule outside it before it has spread to the joint within, and may thus make its way into the parts outside the capsule. Or again, a focus of disease in the head may, as already described, burrow outwards through the centre of the neck, until it finally reaches the soft parts outside the attachment of the capsule round the root of the trochanter. Disease, too, starting in the acetabulum may work its way towards the pelvic aspect of the bone, and form an abscess under the iliacus muscle (Fig. 452). Of course, in any of these cases the general surface of the joint might escape altogether, but as a matter of fact it rarely does so. The abscess which forms within the pelvis is the result of primary acetabular disease, or of the perforation of the acetabulum with general infection of the joint, and may travel towards the surface in several directions. Perhaps the commonest course is to gravitate towards the rectum, into which it may burst or pass along the latter to the perineum. Again, it may ascend under the muscles and present below Poupart's ligament, usually on the outer side of the vessels. Finally, it may enter the bursa under the tendon of the psoas muscle, and so make its way to the inner aspect of the thigh.

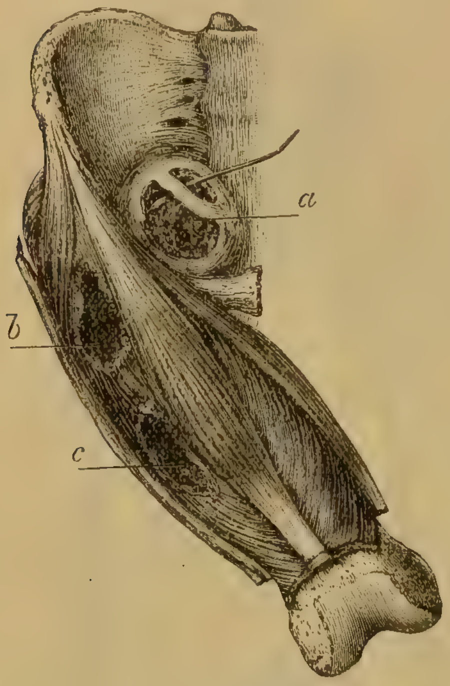

Fig. 452.—Abscesses in connection with Hip Joint Disease.

a, Abscess in the iliac fossa; *b*, abscess under the tensor vaginæ femoris; *c*, abscess extending down the thigh. (From University College Museum.)

When there is much destruction.—Now, although moderate tuberculous synovitis may be recovered from, with little or no damage to the functions of the joint, it is otherwise when the synovial affection is neglected, and the tubercles break down into caseous material and ulcers are formed, from which the disease may spread to all the surfaces of the articulation, or, when the morbid process starts, in the bones themselves. Here a complete restoration of the functions of the part is almost impossible. The surfaces are sure to be destroyed, and, in addition, there are very often portions of necrosed tissue set free in the joint, which will keep up irritation almost indefinitely. It becomes a question, therefore, whether in such cases, inasmuch as the best result which can be expected is a stiff and usually more or less deformed joint, we cannot by operative measures arrest the spread of the disease before it has completely disorganised the part. This is a question of the utmost difficulty, and has been variously answered by different surgeons of large experience. On the one hand, there are those who take a rather gloomy view of the possibilities of operative surgery in this direction, and who persist in the treatment of the affection for years by splints and rest, opening abscesses where necessary, scraping and draining them, but abstaining from every other attempt in the direction of a more radical extirpation of the disease. On the other hand, there is a school of surgeons who, regarding tuberculous disease very much in the same light as they do malignant new growths, attack it as early as possible before it has extensively interfered with the surfaces of the joint, and thus seek to eradicate it at a stage in which there is a prospect of preserving the functions in whole or in part. In the case of the hip I adopt a middle course. I regard tuberculous disease as in many cases purely local and eradicable, but at the same time as curable without operation. And, moreover, I have arrived at the conviction that, when it has to be removed, it should be dealt with by as limited an operative procedure as possible, compatibly with complete evacuation of the locally-diseased tissue.

Briefly stated, my line of action in these cases would be as follows:—Always and in every case to endeavour by rest and other local treatment in the earliest stages, combined with the best constitutional treatment, to arrest the progress of the affection; but when the latter has gone on to undoubted destruction of the surfaces of the joint and abscess within it, to operate for the removal of the contents of the latter before they have made wide ravages in the joints and surrounding structures. At the same time, such operations are to be as limited as possible. For instance, all hip joints which are seen at an early stage of the tuberculous disease are at once treated by rest on splints, etc. And as long as these means appear to keep the morbid process in check they are persevered in. But when it becomes evident that liquefaction of the soft tissues of the joint is taking place, and that the bones are becoming destroyed, I feel justified in assuming that the ultimate results of operation for the removal of the diseased tissue will be at least as good as those arrived at by the expectant mode of treatment, and that they will be, in one respect, very much better—*i.e.* in saving much time to the patient, and in placing him in a position to use the limb often in as many months as years by any other expectant method. Moreover, the amount of joint tissue necessarily removed in operations by this line of treatment I hold to be far less than is usually destroyed where the disease takes its course, even under the best treatment by splints, etc.

Operative measures.—As to the risks of operation for hip disease as at present done, they are very small, and thus one of the objections to this method of dealing with the disease has disappeared. This was formerly a great obstacle to operative interference with hip joint disease, for the mortality was large, having reached as high as 60 per cent. in some of the earlier series recorded. And even later, in 1878, under the antiseptic *régime*, Mr. Croft's statistics showed a death-rate of over 40 per cent. Now we may claim to have reduced the mortality of hip excision to nearly vanishing point. My own rather large series of operations of this kind shows no deaths on a great number of operations, and an ever-increasing improvement as regards the functions of the limb, and the absence of sinuses and general infection of the whole system. My mode of dealing with these cases of destructive tuberculous disease of the joint, whether it arise in the synovial membrane or in either of the bones, provided only that the liquefactive process is well marked and is clearly progressing, is as follows. It is understood that the operative procedure, as now described, is that employed in cases in which no sinuses have as yet formed, no matter how large the abscess.

All care having been taken to prepare the patient for operation by several days or, if possible, a week or two of rest in bed, the joint is opened by a clean cut, all the tuberculous material is cleared out as thoroughly as possible, and the wound is closed from end to end without any drainage. This incision into the joint may be made in several places—*i.e.* posteriorly, externally, or anteriorly. After a large experience of the various incisions, I give the preference decidedly to the anterior incision, first proposed in England by Mr. R. W. Parker, and in Germany at the same time, and quite independently, by the late Prof. Hueter, of Greifswald. This incision has the great advantage of reaching the abscess at the spot where, under the present conditions of previous treatment on the double Thomas's splint, it most frequently presents under the skin. It has, also, the merit that it divides no muscle fibres, no vessels of any importance, and no nerves, and interferes very little with the capsular structures. These last points are of the utmost importance for the nutrition of the parts involved during the process of repair, and for the future strength of the new joint if a movable articulation be the result of the operation. The incision is commenced about half an inch below and external to the anterior superior spine of the ilium, and runs downwards and slightly inwards for two or three inches. The knife, in making it, ought to pass between the borders of the tensor vaginæ femoris externally and the sartorius internally, and as it penetrates deeper, between the gluteus minimus externally and the rectus internally. In this incision the capsule of the joint is reached, in many cases, without the necessity of attending to a single artery, unless the cut be made too far downwards, when the circumflex or its branches may be divided. In any case, these small vessels give no trouble, and, if secured for a minute or two in catch-forceps, will cease to bleed. When the abscess is opened, either before the capsule is divided or after, it is most carefully flushed out with a stream of warm boiled water at high pressure, and scraped clean before going any farther. This flushing is best carried out through a hollow gouge, designed some years ago by the author, by means of which all loose material can be washed away by a powerful voluminous stream as fast as it is loosened or cut by the gouging action of the edge (Fig. 453). When all broken-down matter is scraped and washed away, the capsule is opened by a blunt-pointed bistoury, and the condition of the interior is ascertained with the finger. In the class of cases upon which I am accustomed to operate, the head of the bone will be found to be eroded or completely destroyed, or, on the other hand, it may lie loose within the capsule in the form of one, two, or more sequestra. In either case, it is well to saw the neck across at about its middle with a narrow-bladed saw, so as to obtain a clean surface, unaffected with tuberculous caries. Then the broken-down *débris* of the head is scooped out with the gouge, while the sterilised water is rushing into the cavity and carrying off every trace of dead tissue which is movable. The finger will now feel whether the acetabulum is diseased or not, and if it is the gouge can be used thoroughly to clear out all broken-down cartilage and carious bone, and also to loosen any sequestra which may be present. If it should happen, as is often the case, that the carious process has extended through the centre of the acetabulum into the pelvis, the disease must be followed up with the gouge, until the acetabulum is perforated, and the abscess, which usually in such cases lies on the internal aspect of the bone, is opened up and scraped and flushed clean. I have been able over and over again in this way to remove relatively large sequestra from this situation, and to evacuate large intra-pelvic

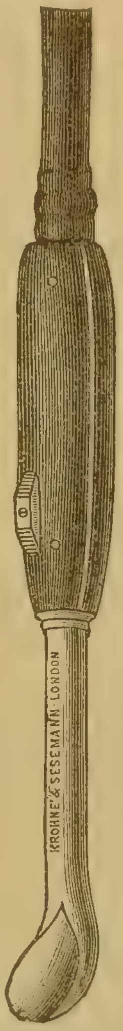

Fig 453.—Flushing Gouge connected with sterilised water tank by flexible rubber tube.

abscesses. When this has been done thoroughly, the whole cavity of the joint is scraped and flushed clean, and filled with a sponge. Stitches are then inserted in the skin wound at short intervals, but not yet tied. When all is ready for the final closure of the wound, the sponge is removed and the cavity of the joint is filled with iodoform emulsion, and then the latter is squeezed out as far as possible. The stitches are then tied, and the whole wound is accurately closed without any drain. It is then dressed with dry salicylic wool, put on in large quantity, so that, when bandaged, even elastic pressure may be brought to bear on all parts of the cavity. At the same time, the remains of the neck are forced into the socket by strong abduction, and retained in this position by the bandage which passes round the pelvis, and by the Thomas's splint, which is at once applied. Over this dressing it is well to apply a covering of some waterproof material, to protect it from accidental wetting with urine, etc.

After treatment.—Almost all the cases thus treated may be thus left undisturbed for nine or ten days, and when the first dressing is removed, it will usually be found that the wound is healed *per primam.* The stitches are then taken out, and a strip of wool is secured over the scar by means of collodion. In some cases, where a little of the tuberculous matter has been left behind, or where the edges of the incision have been inoculated by the tuberculous *débris* passing over them, there will be evidence of the formation of tubercles in the scar at about the end of the third week. If this is the case, and nodules form in the scar, the whole of the latter should be included between two elliptical incisions, and excised; and if there is any of the abscess remaining, it should at the same time be scraped and flushed clean, as before. Then the wound is carefully sutured once more without drainage. The same procedure may in rare cases require to be repeated at the end of another three weeks, but in every case where recurrence is evident the method employed should be that just described, until, finally, sound healing is the result. On no account should a recurrence be allowed to burst through the skin and form a sinus in any case. All this time the patient may be carried about in the open air on the double iron splint, and, as there is no fever, the recovery of general health is usually rapid. At the end of three months or so he may be allowed to walk without the splint, but the latter should be worn always at night to counteract the tendency to flexion, which lasts for a long time.

This method achieves the most thorough eradication of the disease with a minimum of removal of bone or interference with the capsular structures of the joint, and when the union has taken place satisfactorily in the wound, the limb is firm and useful. It is, of course, shorter than its fellow, but this not necessarily because much bone has been taken away, but because of the atrophic changes due to its not being used so much as its fellow. Indeed, if compared with many cases treated without excision that have recovered well and without even sinuses, the amount of shortening and deformity is much less.

The results of operation.—The question whether those joints which have been treated without operation give as good results in respect of their functions as those which have been operated on in the manner described is not yet to be settled by figures. But since this method has been employed by myself and others, a very marked improvement has been noticed in the functions as well as in the permanent local condition. Children are much sooner up and about, and if the tendency to flexion is counteracted for a time by the use of a splint at night, the limb remains in good position. Of course the joint has been destroyed—not by the operation, however, which has only cut a few of the anterior fibres of the capsule and cleaned out carious bone, but by the antecedent disease; and having been destroyed, the resulting condition can never be as good as the original articulation. But with early progression the general nutrition of the limb improves, and the atrophy which invariably results from long immobilisation is prevented. The movements of the articulation are, of course, more or less limited after such an operation, just as they are after recovery from destructive disease, where there has been no interference with the joint. But this has its advantage, nevertheless. For if movement after destructive disease with or without operation be free, it will mean that there is a loose bond of union between the two bones; and if this is the case, the weight of the body thrown upon it must stretch it and lead to much shortening. But if by operation before the whole of the neck of the femur has been destroyed, and the ring of the acetabulum obliterated, the sound remains of the neck can be thrust into a clean gouged acetabulum, and maintained in this position until the two bones are more or less united, the result will of course be much better than in cases where the disease has been allowed to destroy the head and acetabulum completely, and has led to more or less dislocation of the femur upon the dorsum ilii.

But there is an idea that the result of excision for caries of the hip ought to be a *freely* movable joint. This is surely a mistake, for movement in all directions means general looseness of the bond of union, and this must mean slipping of the femur upwards upon the ilium under the weight of the body. My aim in all cases in recent years has been to secure as firm a joint as possible, admitting of little or no movement, but with the remains of the neck firmly soldered into the acetabulum; this reduces the tendency to shortening to a minimum, and also reduces the tendency to flexion at the hip. And even if bony anchylosis were the result (which is extremely rare in young children), if the synostosis took place in good position, it would be better than a very loose joint, which must be more or less weak when weight is thrown upon it.

If there were much risk in the operation as above described, it might be a reason for relying on prolonged rest and the draining of abscesses as they arise rather than face it. But the risk must be very small now. As a matter of fact, I have now done some scores of these limited excisions of the hip for extensive disease, and have so far had no fatalities. Of course the statistics of hip excision in the old septic days were far otherwise, and one of the first series published showed 60 per cent. of deaths. And even later in the Listerian days they were set down at 40 to 45 per cent.; but it is not so now. And not only are the immediate results of the operation immeasurably better than before, but the fact of having got rid early of a focus of tuberculous disease, liable at any time to infect the general system, is a great gain. Further, to put a patient soon into a position to move about and get fresh air and exercise has a most important influence upon his future prospects as regards the recurrence of the disease locally or generally. And as a matter of fact, the amount of general tuberculous disease following upon hip disease in my series has so far been comparatively small.

I am entirely in accord with those who maintain that it is better not to excise those cases in which there is very extensive caries with putrid sinuses, and that their chances as regards life and limb are better when treated by rest, drainage, and local cleansing. But so long as no sinuses are present, we ought to be able to remove all diseased tissue, without allowing the resulting wound to become septic; and if this is possible, the latter ought to heal throughout by first intention.

But there are cases, of course, in which recurrence will take place. These must be treated as was the original disease, by excision of the focus and suture of the clean-cut edges with a view to primary union.

The question of early operation.—There are surgeons of undoubted eminence, however, who hold the opinion that disease of the hip joint ought to be treated at a much earlier stage by operation. Those who adopt this view think that by opening up the joint and removing diseased synovial membrane or bone before the morbid process has attacked the joint generally, that the latter may be spared. This is, however, an opinion which is not largely shared, and which is not generally acted on. The reasons which are given against the practice are briefly these, that inasmuch as the most likely spot for synovial tubercle is around the ligamentum teres, where the synovial tissue is most abundant, the head of the bone would have to be turned out of the acetabulum in order to reach the diseased material, and this in itself would be a great difficulty; moreover, when the affection is concentrated upon this spot, it is very likely to have attacked the bones secondarily if they have not been already primarily affected. If this is so, nothing but the sacrifice of a considerable quantity of the latter would have any prospect of eradicating the disease, and to get at the bones thoroughly would necessitate such a wide dissection of the joint as to imperil its functions very considerably. Finally, experience has taught us that such operations do not, as a rule, secure the patient against recurrence. In my experience of the few cases in which I have attempted thus to resect the hip joint partially at a very early stage of the disease, little, if anything, has been gained.

When this measure is regarded as proper, the first incision is usually made at the back of the joint, curving round the trochanter, and dividing the small rotators. In this way access to the head is secured, and by strong adduction and inversion the head is thrown out of the socket posteriorly, and the acetabulum is exposed. Then all diseased tissue is removed, and the head is replaced, the wound being sewn up completely or drained, according to the view of the operator as to the possibility of obtaining primary union.

Another incision (Langenbeck), sometimes practised with the same object in view, is a vertical one in the axis of the neck of the femur down to the upper border of the trochanter. This gives access to the joint between the fibres of the muscles covering it, and the head of the bone can be forced out by strong adduction when the ligamentum teres is divided. My opinion, however, of these measures, which aim at eradicating the disease in its very early stages, is that they are not likely to be followed by the success hoped for, and that the cases considered suitable for these operations can be better treated by the expectant methods, holding the operative treatment in reserve for such as, in spite of the latter, pass on to the stage of destruction of the head and acetabulum, and the formation of abscess within the joint.

In those cases, however, in which a focus of tuberculous disease which has not yet spread to the surfaces of the joint can be diagnosed in the head of the femur (see above), an operation has more than once been done lately far short of excision, which has given excellent results. This is the trephining through the trochanter, right down the centre of the neck until the focus has been reached, and scraped out clear. All this can be done without opening the joint at all, and if practised at an early stage may save the patient from an excision of the head later and leave all the functions of the joint intact. Cases of this form of disease, however, present themselves but rarely to the surgeon early enough for this method to be followed, but should always be looked out for.

When the disease starts in the centre of the acetabulum it almost invariably spreads rapidly to the surface of the head as well as to the synovial membrane round the ligamentum teres, and this being so the prospect of saving the joint from utter destruction, otherwise than by excision of the head, is very remote. The head in this case acts as a barrier to prevent the products of liquefaction from working outwards, and the consequence usually is that the latter spread towards the pelvic aspect of the bone, and form an abscess internally within the pelvis. To prevent this when the condition is recognised, nothing can be done but to excise the head of the femur and treat the whole condition as described above. Only when the head has been removed can the acetabular disease be satisfactorily dealt with.

Amputation at the hip joint.—The question whether amputation at the hip joint for tuberculous disease should be performed must be answered in each particular case for itself. Undoubtedly, there have been cases in the past in which this measure was proper, and has saved life at the expense of the limb. But such cases are becoming rarer and rarer. My own opinion is that in instances of very extensive hip joint disease the patient usually stands as good a chance of his life by waiting and careful evacuation of morbid products as he does from amputation at the joint. In my own experience, which has been large both of cases treated without and with excision, it has never been necessary to sacrifice the limb, and I have never had occasion to regret not having done this operation. In one case it was proposed because the patient, a boy of about ten, had run down with chronic suppuration, and was extensively affected with amyloid disease; but the parents decided against removal of the limb. This boy ultimately recovered wonderfully, and years after had a sound and useful limb and healthy body. Indeed, so much is now accomplished by early constitutional treatment and immobilisation in preventing destructive change in the hip joint—and, when this latter has begun, in arresting it by timely limited operation—that the need for amputation at the hip joint has almost disappeared, and ought before long to be quite a thing of the past. I have quite lately had under my care a boy of about ten, whose limb had been thus removed for tubercle of the hip by some other surgeon, and who came to me for tuberculous disease of the remaining knee, but though he was alive, the result was anything but encouraging.

Chronic osteo-arthritis of the hip.—This is a very common affection in patients past middle life in both sexes. Its general pathology has already been described (page 1052), and we are here only concerned in its clinical features.

The **symptoms** are at first usually stiffness and a certain amount of "tearing" pain on rising in the morning, and especially in bad heavy weather, or when the patient is run down and debilitated. This is followed by a sensation of grating in the joint on use, and more and more limitation of movement, even to complete fixation. Sometimes there is effusion into the capsule in this stage, especially after over-use; and if this condition is present, it will indicate an inflammatory process grafted on the degenerative, as the result of the friction of the roughened surfaces. But as the altered head of the femur becomes further and further buried in the osteophytes of the acetabulum, the liability to this inflammation will grow less.

The **treatment** consists in improvement of the powers of assimilation and elimination by attention to diet, and the moderate use of the more digestible stimulants and the alkaline mineral waters. The clothing must also be abundant, and the patient should take regular exercise. The warm baths of Wildbad appear to exercise a most favourable influence upon this disease, and if combined with regular massage even to arrest its progress in the early stages.

When locking of the bones has taken place, as the result of the growth of osteophytes and the alteration in the shape of the head of the femur and acetabulum, nothing further can be done. All operative interference is contra-indicated on account of the degenerative nature of the initial disease.

Faulty anchylosis of the hip.—In cases in which destructive disease of the hip has been followed by recovery without operation, the position of the joint is often extremely faulty. Most usually it is strongly flexed, adducted, and inverted. The reasons for this malposition have been given above (page 1083), and we need only add that in addition to what may be called the pathological grounds for the displacement, there are also natural reasons. In the first place a child who has had disease in the hip, even after all active change has ceased, will find the most comfortable position one of flexion. For instance, most children, and, indeed, adults, lie in bed with the thighs flexed—few of us lie straight out in bed all night. That is to say, that for eight or nine hours out of the twenty-four the joint is flexed. If consolidation be going on at this time the fibrous tissue is likely to adapt itself to this position. And during the waking hours it will be always irksome, to say the least, to bring the joint into a straight line again. It therefore tends to get a "set" in the flexed position, and to become firm in it. Again, during the day such patients will remain seated for a considerable time, and they can only do so with the thighs partly flexed. These two reasons for the assumption of the position of flexion are constantly at work, and it is not to be wondered at that the patient recovering from hip joint disease with anchylosis is frequently found to have the limb in a very faulty position for progression. It is usually flexed and adducted.

Treatment.—The question now arises, can anything be done to improve this condition; and if so, ought it to be undertaken, and how? The answer to this is that we should only make the attempt to remedy the anchylosis when the displacement is extreme, and when all traces of active disease have disappeared. Further, unless we have a reasonable prospect of securing a movable false joint by our operation, we had better abstain from all interference, except in extreme cases. For to obtain a fully extended hip which is quite rigid is a doubtful benefit. For with it the patient cannot sit down with comfort, and this has many inconveniences, *e.g.* during defæcation, etc., and these will more than counterbalance the advantages of being able to walk somewhat better. We should, therefore, in every case aim at producing a movable joint, and only operate in extreme cases.

Several modes of *operation for bony anchylosis* have been proposed and practised, differing principally in the position in which the bone is divided. Probably the best is that in which the neck of the femur is sawn across as near the acetabulum as possible. This is effected with a narrow-bladed saw, introduced to the front of the neck through a small incision made above the trochanter. When the bone is divided, it may be well to dig out a hollow representing the acetabulum into which the stump of the neck can be laid and turn in between the cut surfaces a flap of muscle or tendon in order to prevent the union of the surfaces by bony tissue. In any case every effort should be made by passive motion and by extension to hinder the union of the cut surfaces. This may be done in some cases, but is a matter of great difficulty.

In other instances it is more desirable to divide the femur below the lesser trochanter either with saw or chisel. If bony anchylosis after this is inevitable, the limb should be allowed to become stiff in a slightly flexed position.

The general rules for the treatment of other forms of limitation of movement following upon disease of the hip joint will be found under the heading "Anchylosis of Joints," page 1067.

Neoplasms about the hip.—These are not common. When present they are most usually chondromata or osteo-sarcomata of the end of the femur or periosteum. The joint is only secondarily invaded, and is for a long time uninfluenced except in its movements.

The considerations arising out of the presence of neoplasms about the hip turn entirely upon the question of the propriety or the reverse of amputation through the joint. When such a growth has been diagnosed near or in the hip, the question of operation or not depends upon the capability of the patient to bear the shock of such a severe procedure. But as the outcome will eventually be death if left untreated, amputation is justifiable in most cases.

Cysts about the hip.—These are, on the whole, rarely seen. When present they are usually due to the distension of one of the synovial bursæ of the part, and more rarely of pouches of the proper synovial membrane of the joint. The cause is chronic inflammation, usually of the tuberculous type. Thus the bursa of the psoas muscle may be distended, as in a case lately under my care. Treatment of these conditions is not satisfactory, but repeated aspirations and injections of the iodoform emulsion may be tried with advantage in some cases. In other instances, the greater part of the diseased synovial membrane may be cautiously dissected out.

Loose bodies in the hip joint are very rarely seen, and when present require the ordinary treatment of incision and extraction from behind. I have only met with such a condition once, and then the case required no treatment.

AFFECTIONS OF THE KNEE JOINT.

In considering the affections of the knee, we have before us the same diseases as those we have just been considering in the hip joint, modified, of course, by the special conditions which surround the part.

Simple acute synovitis of the knee.—This is a common affection, owing to the exposed position of the joint, which subjects it to extremes of heat and cold and to injuries of all kinds.

Symptoms.—However caused, the symptoms are much the same, both subjectively and objectively, as in the case of the hip, just considered.

The first thing noticed is stiffness, then heat, and later, general pain and throbbing in the joint, which will be also tender to the touch, and on movement. There will also be a general rise of the body temperature in all probability. Objectively, there will be swelling and possibly redness, and a characteristic attitude. The swelling will be due to two causes: first to effusion of synovium into the cavity, and next, in severe cases, to a certain amount of œdema of the soft parts surrounding the joint. It will be most noticed above and below the patella at either side, and where there is much effusion, the patella may be raised from the anterior surface of the condyles, and "float" as it is said. When pressed upon it can be made to touch the femur again, but immediately rises owing to the distension of the capsule. Fluctuation may be also felt from one part of the capsule to the other. The redness and œdema in ordinary cases are but slight; but where suppuration is present, they are a marked feature.

The limb is always more or less flexed in acute synovitis of the knee, and can only be straightened with difficulty by the patient or surgeon. This position is assumed in order to relax the posterior parts of the capsule, which with the limb extended is tightly stretched, and in this state more tender than if relaxed. The patient will resist all attempts to move the joint, but if placed under chloroform the movements will be free and extension perfect It is plainly, then, a position assumed to relieve tension and pain, and is no deformity in the usual sense of the word.

The **diagnosis** of this affection, as a rule, presents no difficulties. The history and the rapid onset with the symptoms of acute inflammation serve to distinguish it from all other swellings except one: this is hæmatoma or hæmarthrus of the joint (page 1034). Sometimes, owing to a slight blow or strain of the knee, some small vessel in the interior of the joint gives way, and the cavity rapidly fills up with blood. Here the general appearance of the joint very much resembles that seen with synovitis. The pouches above and below the patella on either side are distended with fluid, and fluctuate, and the latter bone "floats;" the knee is slightly flexed, and is, perhaps, a little tender. But the condition can usually be distinguished from synovitis, by accurately noting the time at which the swelling sets in. With hæmarthrus the effusion takes place immediately on receipt of the injury, and the capsule will be tense within an hour of its occurrence. Synovial effusion does not set in, as a rule, for some hours after the exciting cause has commenced to operate. Again, there will be little or no heat in the joint in the case of the blood effusion and in general fever, while in synovitis the joint will feel hot to the patient and to the surgeon's hand, and the body heat will be increased. In some cases, of course, the injury which gave rise to the hæmarthrus will also set up synovitis; but in that case, the symptoms of inflammation will not commence until some time after the swelling of the blood effusion has been well established.

The **treatment** of simple acute synovitis of the knee has the same general principles as its basis as in dealing with acute inflammation anywhere else. General vascular tension is relieved by laxatives or brisk purges, according to the patient's condition. To this may be added the administration of tartarised antimony in doses of about one-sixteenth of a grain every three hours *ad nauseam* in sthenic cases. Locally, the joint must be treated by absolute rest in the first place. This is best secured by a back splint of a kind to fix the limb in the extended position, and at the same time to leave the knee free for the application of various antiphlogistics. The Macintyre splint fulfils all these conditions; but where it is not at hand, a straight back splint of wood will serve all the purposes required. It is well, however, not forcibly to extend the joint in the first instance, but to bring it down to a straight line gradually.

For the relief of local congestion leeches are in some cases an excellent remedy, half a dozen being applied in various parts of the front of the joint, followed by hot fomentations. In milder attacks an ice-bag laid on the front and sides of the knee at the outset will check the local inflammation, and give relief from pain sooner than anything else. In some cases without using leeches great relief is given by the application of belladonna and glycerine and hot fomentations.

By one or more of these means the inflammation is soon brought to a standstill, and nothing is left but stiffness and some tenderness. This is best relieved by massage and gentle passive motion. But in the case of unhealthy young individuals we must exercise the greatest caution in allowing the limb to be used before all congestion is relieved, remembering how readily tuberculous disease is grafted on a part damaged by inflammation. If there be any doubt as to this infection the limb should be immobilised for long periods, and the patient should be given all the benefits of fresh air at the seaside, with tonics, cod-liver oil, and all the other general and local remedies mentioned above (page 1049).

Acute suppurative synovitis is best treated by free incision and washing-out of the joint with warm sterilised water or one of the numerous antiseptic lotions, care being taken that the germicide employed be not strong enough to irritate the surfaces of the joint. The incisions in such a case should be placed on either side of the joint and towards the posterior aspect of the lateral pockets of the synovial membrane. These incisions should be carefully kept open with some form of drainage apparatus, not necessarily a tube, but often a strip of gauze.

It is remarkable what excellent results as regards the functions of the joints are obtained in such cases, provided the part be opened early and freely even when the cause has been some form of septic infection, such as pyæmia in one of its milder forms.

I have lately heard from a gentleman in America, upon whom I performed this operation some years ago for extensive septic suppuration of the knee. The joint is now perfectly restored, and is as good as its fellow by the patient's account.

In the more severe attacks of pyæmic inflammation, the general condition has so lowered the nutrition of all the tissues that repair is usually very imperfect.

Simple subacute synovitis.—In this form the same symptoms will be present in a modified degree. The same treatment, carried out in a less active way, is suitable. But even these cases of subacute synovitis, if septic, are best treated by early incision and drainage, and are thus saved in many cases from adhesions and stiffness, which would seriously interfere with the functions of the joint if the incision were delayed.

Simple chronic synovitis, independent of tubercle, and except as the outcome of acute attacks, is a comparatively rare disease. It usually appears in one of two forms. Either there is a chronic hyper-secretion into the joint, unaccompanied by thickening of the capsular structures, or the latter is present without much effusion. The first form is due to chronic congestion of the synovial membrane, destroying the balance between secretion and absorption, and large quantities of fluid are poured out into the joint, distending the lateral pouches and to all appearances thinning them. This *hydrops articuli*, as it is sometimes called, produces great weakness, but little pain. If left unrelieved long, it stretches the capsule and the ligaments, and the whole joint is thus rendered insecure.

The **treatment** in milder cases consists in free blistering of the joint at either side, with immobilisation. After the blisters have healed, the part should be firmly bandaged during the day and massaged morning and evening. When the fluid has been removed by these means, an elastic knee-cap should be worn continuously for some months. There is in some of these cases unfortunately a great tendency to reaccumulation of fluid in the joint as soon as patients begin to use the limb. In such cases blistering may fail to remove it permanently, and it may be necessary to aspirate the joint repeatedly. This is certainly better than to allow the continuous distension to weaken the whole apparatus of the ligaments and capsule. But this, again, may fail, and then it is well to combine the aspirations with washing-out of the joint with some antiseptic and at the same time stimulating fluid. For this purpose a lotion of perchloride of mercury 1 to 1,000 may be used, or tinctura iodi ʒj to the pint of sterilised water, or carbolic lotion 1 to 20. With this treatment many cases are cured after the other simpler modes of treatment have failed.

Stubborn forms of hydrops articuli.—But there is a form of dropsy of the joint which is particularly stubborn, which is combined with a peculiar hypertrophy of the synovial fringes, giving to them the appearance of polypoid masses. There can be no doubt that in some cases the cause of this change is tuberculous irritation, and that the most marked examples are due to this cause. But it is also equally certain that a hyperplasia of the synovial fringes, leading to the formation of pendulous folds, may occur quite independently of tuberculous irritation as far as the evidence to be obtained from the microscope goes. What the exact

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cause is, is not in all cases clear, but the visible effect upon the synovial structures is plain enough. There is a chronic congestion, and the result is hyperplasia taking the form of hypertrophy of the synovial membrane, which is thrown into ridges and folds, gradually elongating into more or less pendulous masses. In a case I operated on within the last year on account of chronic synovitis with recurring effusion in an otherwise healthy young man, this was exceedingly well seen on opening the joint. The lining was deep pink and irregular and rugose, with fringes as described. After removal of the latter, they were found to consist solely of inflammatory tissue in various degrees of organisation, but without a trace of tubercular structure. The removal of a quantity of this altered synovial membrane from both sides of the joint was followed by complete recovery of its powers.

Besides this variety of chronic synovitis, accompanied by *hydrops articuli*, there is also, as already mentioned, that form in which all the synovial membrane undergoes a general hyperplasia without any particular hyper-secretion. In this form there is not necessarily any formation of fringes, but the thickening may give rise to much general enlargement of the joint and a pulpiness appreciable to the fingers. It is a matter of the very greatest difficulty to distinguish this form, in which tubercle plays no part, from that in which this infection is the cause. And in any case in which there is doubt, the disease should be treated as though it were tuberculous.

But where there is no evidence of the latter disease, we can do a good deal short of operative measures for its relief. Frequent counter-irritation, combined with rest, will often bring about a return to the normal condition. In addition to this, careful and continuous bandaging and strapping may relieve the congestion and promote absorption of inflammatory matter.

The patient's general health, of course, must be attended to at the same time, and in the later stages and where only stiffness and thickening remain, much benefit will result from massage carried out methodically and continuously. Where these means fail, the joint may be opened and washed out; and if this likewise is without beneficial result, recovery is much hastened by excision of considerable portions of the thickened synovial membrane on both sides of the joint. This is best carried out by lateral incision curved from either side of the ligamentum patellæ upwards and outwards for about three inches. When the capsule has thus been laid open the synovial membrane is dissected away from the sides and front of the bones as far as possible. It is needless to say that this can only be safely done where perfect asepsis can be compassed. When as much as is considered necessary has been dissected away, and all bleeding has been checked, the capsule is completely closed all round, and then the skin wound. In my opinion drainage is in these cases quite unnecessary. When sound union has taken place, passive movement and massage should be regularly carried out, and as soon as tenderness has passed off, cautious active movement must be begun, and should not be put off too long.

When some of the pendulous masses just referred to become detached, being torn off during the movements of the joint, one of the many forms of "loose bodies," or "joint mice" as they are called in Germany, is produced, to which reference has already been made (page 1070).

Tuberculous disease of the knee. **Pathology.**—This form of disease has much in common with that described as affecting the hip joint. It is found mostly among the very young and also among the very old, while middle-age is almost exempted from it. The infection is met with primarily either in the synovial membrane, or in either the femur, the tibia, or the patella. In the very young the femur is most frequently the starting-point, next the tibia, then the synovial membrane, lastly the patella. The older the patient the greater the probability of the synovial membrane being first affected, inasmuch as the growth of the bones is less active as time goes on, while the functions of the synovial membrane remain during life. But starting in any of the situations mentioned, the disease may spread to any of the other structures.

When the tubercle is primary in the synovial membrane it shows itself first in the subendothelial structures, as a rule (Fig. 431), and from this spreads either towards the surface of the joint or towards the external parts. Commencing in the bones, it is found either under the encrusting cartilage or at the junction of epiphysis and diaphysis, where growth is most active. From this it may spread to any or all the surfaces of the joint, or work its way to the skin, without in any way involving the articulation. This latter point should never be lost sight of, for it is a grave mistake to conclude that because there are extensive tuberculous abscesses round the knee, with perhaps several sinuses, the surfaces of the joint are necessarily involved. If this conclusion be come to in any given case, the joint will probably be opened freely as though for excision, and if it then be found that the abscesses could have been cleaned out and the foci of disease removed without interfering with any of the surfaces or functions of the part, it will be seen that an unnecessary and possibly injurious operation has been done.

Indications.—In all cases, then, it is a matter of the greatest importance to determine at what spot the tuberculous disease has started, and in what direction it is extending. The question is, can this be done? In some measure it can, but only to a limited extent at present.

In the first place it must be remembered that disease commencing in the growing parts of the bones is an affection of early life. The older the patient the less probable is it that the tuberculous infection has begun in the bones. Again, disease of the synovial membrane is more likely to be found among the more mature patients, though by no means limited to them.

Bearing these points in mind, we may study the symptoms of tuberculous synovial disease and of bone disease of the knee joint. (*See* Fig. 455.)

In the former the infection commencing in the subendothelial tissue is almost certain very soon to affect either the surface of the joint or the capsular structures. In the first case the movements of the articulation are early impaired, the patient being anxious to avoid the rubbing of the affected surfaces one over the other. He will, therefore, keep the joint fixed, though he may be able to bear his whole weight on it without pain. Moreover, he will place it in the attitude of greatest ease, namely, of slight flexion. The same may be said of that form of disease in which the infection progresses outwards through the capsule. Here the necessity of relaxing the latter will be felt also, and this will be best secured by flexion. In the case in which the surface is affected, there will also probably be a certain amount of effusion of synovium into the joint with all the usual symptoms. (*See* page 1034.) The subjective symptoms will be pain on movement and slight tenderness, but probably no sense of heat in many cases.

When the epiphysis of the femur or tibia is alone affected, there will be little or no alteration in the attitude of the limb, inasmuch as the ligaments may be wholly unaffected, and consequently do not need relaxation. The movements again may be free in so far as the surfaces are not involved, and there will be no effusion for the same reason. On the other hand, the end of the affected bone will be thickened and tender, and when the weight of the body is thrown upon it, there will be decided pain. The same may be said of jars given to the limb in its long axis by blows with the hand on the heel and also of hyper-extension. Subjectively, a sense of heat and throbbing will probably be felt in the end of the bone, especially at night, and one or more especially tender spots may be felt over the epiphysial line. For those cases in which the disease starts under the encrusting cartilage, no special guiding symptoms can be given, and in most cases evidence of the surfaces being involved will soon be forthcoming.

The **treatment** in all three forms is that already laid down for tuberculous joint disease wherever it occurs. It consists chiefly in absolute rest with counter-irritation in the early stages, combined with tonics, fresh air, and good food. The completest rest can be effectually obtained by the use of a Thomas's knee splint (Fig. 454) or by a plaster-of-Paris case carried from the fold of the buttock to the sole of the foot. With either of these appliances, which effectually immobilise the joint, the patient can walk about, but will require crutches for the last named and a thick-soled boot for the sound foot. For this reason, and from the fact that with a Thomas's splint the knee not being covered in can be treated by external applications, the latter form of appliance is preferable to any, and the patient can walk about without crutches, resting the whole weight of his body upon the splint on the affected side, and on the sound side upon an iron patten screwed on to the sole of a strong boot.

In this case, too, the method of treatment by passive hyperæmia, formulated by Dr. Bier of Kiel (*see* page 1050), can be carried out without confining the patient to bed or the house; and to this may be added injection of the diseased area with iodoform emulsion, repeated as often as is called for. So long as there is no evidence that the disease is running on into caseous abscess, this treatment should be persisted in, especially as it can be carried out without confining the patient to bed or even to the house. And this is especially true of very young children, who have a wonderful power of battling with the disease in many cases, while at the same time the best results of operation are not likely to be attained in very young individuals. But as soon as the surgeon is convinced that the disease is progressing to the stage of caseous abscess, whether in the bone or synovial membrane, operative interference is called for.

Operative measures. — I am not in favour of operations for the removal of tuberculous disease of the synovial membrane of the knee in its very earliest stages, and this for many reasons. In the first place a large number of patients with this form of disease may be restored to health by the ordinary modes of treatment, local and general. Secondly, though the risks of the operation are slight, they have to be taken into account; and if the disease can be brought to a standstill without them, it is plainly right to avoid them. Again, it is difficult at first to define how far the disease has invaded the synovial membrane, and, consequently, how much ought to be taken away. Finally, if we are likely to have to remove the ligaments of the knee, the result so far as the preservation of the functions of the joint is concerned will not be much the worse for waiting a little. When the ligamentous structures are destroyed or cut away the difficulty of securing a firm joint is very great in young patients. They may have to keep the part immobilised for years after in either case. And even where partial resection of the superficial layers of the bones has to be undertaken, on account of the extension of the disease from the synovial membrane to them, the difficulties of

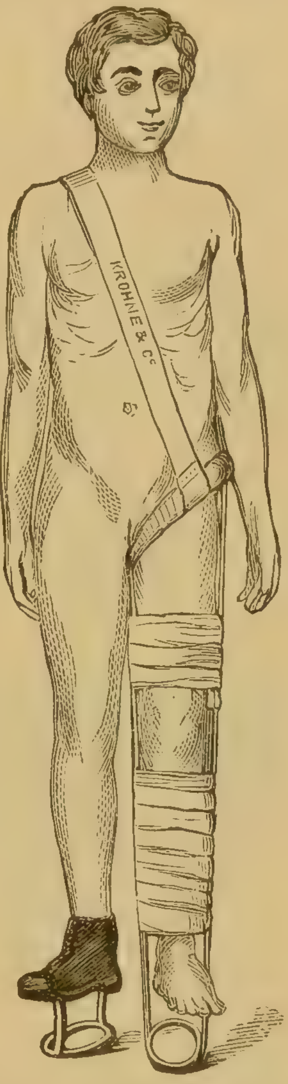

Fig. 454.—Thomas's Knee Splint applied.

securing a firm union of the solid parts with one another, so that they will not subsequently yield to the weight of the body, are very great indeed in very young individuals.

But, where the disease is manifestly advancing, and caseous foci are forming and are developing into abscesses, it is plain that a state of things exists which indicates a low degree of resisting power in the tissues, and that we may expect further destructive change in the joint, unless some special measures are taken to arrest the local process. Perhaps even at this stage the local injection of iodoform emulsion, as already described, should be of some use, and certainly should be tried in suitable cases. Or Bier's method of treatment by "passive hyperæmia" may be employed tentatively for a time. But supposing these means to have failed, and caseous abscesses to be in process of formation, what is to be done? The answer to this is that complete excision of the diseased material in the joint ought to be undertaken.

In using the term "excision" it is necessary to say that it has a very different signification at the present time to that it was meant to imply in former days. Then it meant very free removal of much of the structures essential to the functions of the articulation, while at the same time it was far from meaning complete removal of all morbid tissue; now it means in many cases very partial interference with the structures essential to the joint, while it aims in every case at the complete removal of the diseased tissues.

For such a condition, for instance, as that figured (Fig. 455), it would obviously be unnecessary now to do more than dig out the sequestra from the surfaces of the bones and scrape the latter, which would not shorten the limb appreciably. Formerly the ends of the bones would have been sawn off without any extra gain.

It has become the fashion of late in some quarters to speak of partial excision of the joint, where the diseased synovial membrane is chiefly, or solely, removed, as "arthrectomy," and to reserve the term "excision" for that procedure involving the removal of the ends of the two bones completely. This nomenclature, however, appears to be attended with inconvenience, inasmuch as it is very rare, on the one hand, to meet with a case of disease of the knee in which the whole of the synovial membrane requires extirpation, and yet none of the bone need be removed; and, on the other hand, it is equally rare to come upon a case which requires free removal of the ends of the bones, and yet does not call for extirpation of all the synovial structures. For these reasons I would suggest the use of the terms "partial" and "complete" excision of the knee as preferable and more accurate. The terms "arthrectomy" and "excision" overlap too much to be used with much advantage.

Nowadays complete excision of the knee is a comparatively rare operation—that is to say, it is uncommon to see the knee joint opened and the ends of both bones sawn off and fitted together, as was formerly so often done. The reasons for this are twofold. In the first place the recognition of the importance of early and vigorous, as well as patient, treatment of tuberculous disease has led to the arrest of much disease which would formerly have run on to complete destruction of the ends of the femur and tibia; and, on the other hand, when operative treatment is decided on, it is now usually at a stage at which the removal of the diseased parts of the bones can be accomplished without complete removal of the ends. But at the same time, though the bones are treated in a much more conservative way than formerly, the removal of the synovial membrane is now particularly free, and sometimes amounts to its entire extirpation. Indeed, in those cases in which a movable joint is not to be expected so great is the amount of the disease, we cannot do better than dissect out every fragment we can trace of synovial membrane, whether visibly infected or not.

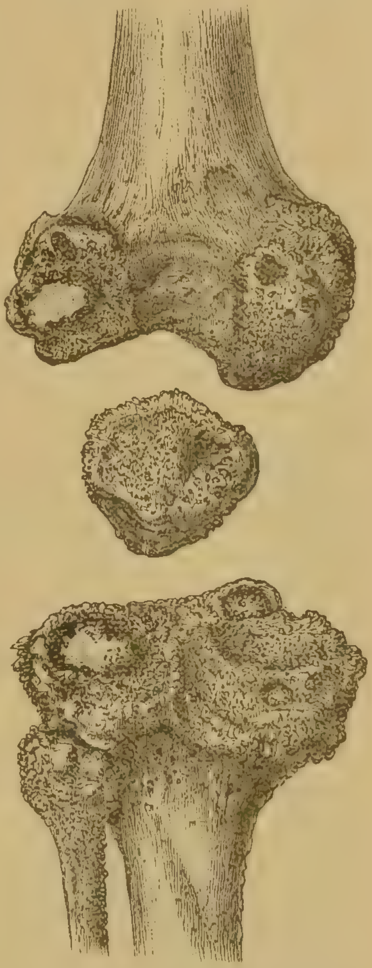

Fig. 455.—Tuberculous Disease of the Knee Joint starting in the Synovial Membrane, and producing superficial Necrosis of Bone. (From University College Museum.)

And now let us take a case of tuberculous disease of the knee sufficiently advanced to call for operation, and consider how we may best secure the removal of the diseased tissue, whether it lie in the bone or in the synovial structures (Fig. 455). Of course, if it have invaded the soft parts external to the capsule, and have formed abscesses in them, the same principles of removal guide us as in the other instances. Now in such a case the first principle which is paramount is the eradication of all tissue in which we believe the tubercle to have been deposited; the next is to spare all structures, except the synovial membrane, which are not infected with the parasite. This sounds a fairly simple problem, but as a matter of fact is far from being so in most cases.

The first incision into the knee joint may be made in a variety of ways, so as to gain access to the whole of the diseased tissue within. In the older operations, in which an absolutely stiff joint was the object aimed at, it was immaterial whether the ligamentum patellæ was divided or not, inasmuch as the action of the extensor muscles was not of importance. In some of the newer operations, in which an attempt is made to preserve some or all the movements, the ligament is preserved intact.

In the first instance an oval flap is formed, the lower curved end of which crosses the middle of the patellar ligament, the two lateral incisions being placed well back, and running to a variable height upwards.

But in those cases in which only a moderate amount of synovial disease is present, two curved incisions reaching from the edge of the patellar ligament on either side upwards on the sides of the joint will give very free access to the synovial pouches, without dividing the ligamentum patellæ or the lateral ligaments. And should it prove possible in some cases thus to remove all the diseased synovial membrane through these lateral openings, there is nothing to prevent the joint being as strong as before. Only the capsule has been divided.

Again, it may be found necessary in some cases to secure very free access to all parts of the articulation, while aiming at the preservation of all its movements. Perhaps the best way of achieving this object is to make a transverse incision through the soft parts across the centre of the patella, and then saw through the latter in the same line. If the knee is now flexed it is very widely exposed, but if there is any difficulty in seeing its deeper parts, the single transverse cut may be combined with two vertical lateral ones, forming an H-shaped opening. In this way two quadrilateral flaps are formed, one of which can be turned up, and the other down, thus exposing the whole joint in the fullest manner. When the removal of the diseased tissue is complete, the two flaps are replaced, and the surfaces of the divided patella are brought into accurate apposition and sutured firmly with wire. In this case, if the whole operation has been perfectly aseptic, and the wound heals by first intention, the strength and movements of the joint are left unimpaired.

By any of these means access to the joint sufficiently free for the removal of any or all diseased tissue can be secured.

The general measures adopted by the author in carrying out such operations are practically the same as those described in dealing with the hip. From the time the joint is opened until all diseased material is removed, and it is closed again, a stream of sterilised hot water is kept playing over the field of action at high pressure. This carries away all infective *débris*, checks oozing, and enables the surgeon to see clearly what is diseased tissue and what is sound. When all morbid material—whether synovial or osseous—has been removed by careful dissection with the knife, assisted by the scissors and flushing gouge (Fig. 453), the cavity is filled with small sponges, the stitches are inserted in the skin and soft parts with the greatest care as to accuracy of adjustment. Then, before they are tied the sponges are removed, and the joint receives a final washing out with hot water, all clots being thus cleared out. The surfaces are then dried carefully and covered with iodoform emulsion, and then the stitches are secured without leaving any room for drainage. An antiseptic wool dressing put on very thick, and firmly bandaged to prevent oozing internally, completes the operation. The limb should then be secured upon a straight splint, and be kept in an elevated position for several hours, also to check oozing. The result is almost invariably primary union, without a trace of suppuration. The stitches are taken out on the ninth or tenth day, and all is sound. Of course a careful watch is kept upon the temperature all this time, and if this rises and remains elevated and the patient complains of pain and throbbing in the joint with malaise and anorexia, the dressings must be removed and the joint inspected. If it be found distended with either blood or pus, a tube must be inserted and thorough drainage provided for. In dissecting away the synovial membrane I always commence methodically at the tip of the oval flap when it is turned up, and work upwards, peeling it from the posterior surface of the quadriceps, then peel it off from the point of the femur and at the sides, then from about the crucial ligaments, and, finally, off the head and borders of the tibia. In many cases it is possible in this way to remove the whole synovial membrane almost in a continuous sheet.

If the patellar ligament has been divided I always stitch it together again with silk, which remains buried. It is well in these operations not to include the capsule of the joint in the stitches which unite the skin wound. If the capsule and expansion of the quadriceps be included, the skin is apt to be dragged in and the edges displaced, which may interfere with primary union. Indeed, it is probably better, for several reasons, not to unite the edges of the capsule and the fibrous expansion of the muscle at all. One advantage of not doing so is that, if there be any effusion into the joint cavity after the operation, the fluid is not pent up, but escapes into the areolar planes around the knee, and is more rapidly absorbed than if it remained in the joint. And should the deeper parts unhappily suppurate from any mischance, the pus would at once appear under the skin, and be easily evacuated, which would not be the case were the divided capsule closely sutured.

When the wound is perfectly healed, which, as a rule, is the case at the end of ten days, I find it best to put up the whole limb, from the gluteal fold to the toes, in a firm plaster-of-Paris case, applied closely over a thin layer of cotton wadding. This splint can be removed at the end of six weeks, and renewed if the joint is not yet firm enough to walk upon. As a rule, the limb may be used in the eighth week, but not without the support of the splint. In some cases, however, where only a very limited removal of synovial membrane has been undertaken, and where all the more important ligaments have been spared, a movable joint may be aimed at, and here it will be necessary to leave off the splints during the day, and to encourage passive and active movement. Massage, too, should be employed to get rid of the stiffness which remains. But in these cases it will be very desirable to keep the limb on a straight splint at night, lest angular displacement take place, which would be difficult to correct during the day. But, hitherto, the author has only operated, as a rule, upon such cases as were affected with too much disease to justify the hope that after operation the movements could ever be restored, and a stiff, firm joint has been purposely aimed at. The knee has been put up in very slight flexion, so that the front of the foot should reach the ground first in walking. Moreover, this is the most comfortable position for the limb when stiff if the patient is sitting down. And in those cases where, either from atrophy from disuse, or from removal of diseased parts of the femur or tibia, the limb is shortened, the pointing of the toes will make up for the slight shortening.

This atrophy from disuse must never be overlooked. It is not uncommon in cases where disease of the knee joint has existed, without any operative interference, perhaps, for years, to find that the tibia is shortened to the extent of an inch or so, and the femur as well. If we have not made careful measurement of the limb before operation in such cases, we may be inclined to set down the loss of length of the leg to our interference, where really the latter has had nothing to do with the condition.

But if the knee has been put up in a slightly bent position, it is necessary to guard against this flexion becoming greater, and this can only be done by the use of splints for months or, in some cases, years. This is always necessary in young children under ten years of age. Here, if a limited excision be done at all, and the cartilage on femur and tibia be left intact, or nearly so, the union between these surfaces must be fibrous. And if this be so, the soft bond of union is capable of being stretched to a considerable extent. Now, children spend a great part of the twenty-four hours in bed or in a sitting position. As a rule, when thus in bed or seated, there is a great tendency to flex the knee into the position of greatest ease; and if this position be maintained for many hours daily, there is always a difficulty in restoring it to a straight position. Indeed, it has a tendency to grow more and more flexed, on account of the preponderating action of the hamstring muscles. For this reason, in all young cases I insist upon the use of a straight splint, at all events at night, for months or years it may be after all trouble has been overcome in the knee. And inasmuch as among the poor and ill-educated a back splint is rarely properly attended to, I find it better to keep the limb in plaster-of-Paris for long periods. In this the patient can be allowed to walk about, and gradually the habit of keeping the limb straight becomes fixed, and it can be ultimately left off when all resiliency has ceased.

In those cases, however, in which the cartilages have been removed by the knife or destroyed by disease, and where bony surfaces are brought in contact, the union is by bone, and the need of splints is not felt after two or three months. It must not be forgotten, however, that the ends of both femur and tibia in such cases have been softened by atrophy, the result of disuse. If under such conditions too much weight is thrown upon the bones before they have quite recovered their firmness, they may actually bend above or below the joint, as in rickets, and antero-posterior or lateral flexion be the result. This is due to no fault in the operation, but is due to the atrophic state of the ends of the bones and their over-use too early.

When these pathological facts are overlooked, much disappointment is often experienced. The child comes back after some months with a sound knee, but with flexion often to a high angle, requiring much trouble to correct, even if it can be corrected at all.

Syphilitic disease of the knee.—This is a comparatively rare disease, if we except those cases in which gummata form on the surfaces of the joint. Such are not uncommon, and may be mistaken for bursal or other tumours. The patient's history and the chronic nature of the ailment, together with the early discoloration of the skin, will generally leave but little doubt as to the condition.

Another form of syphilitic disease, too, is not very uncommon in the knee, namely, gumma of the head of the tibia or end of the femur, which works its way into the joint and seriously damages the latter.

But syphilitic synovitis, meaning by this the deposit of granuloma or gummata in the synovial membrane of the joint, is not common. The cases I have seen have all been congenital in young patients, and have been associated with other evidences of the hereditary disorder, such as pegged teeth, interstitial keratitis, etc. It is, as a rule, an almost painless affection, and only troubles the patient by producing a weakness and stiffness of the joint. Progression is not interfered with. The part is swollen but not tender, and the sense to the finger is as of a doughy mass without any fluid effusion.

The *diagnosis* is not difficult in view of the concomitant evidences of syphilis and the absences of any signs of tubercle, the only disease it is at all likely to be confounded with.

The *treatment* is that of congenital syphilis generally. The limb may be used as much as is reasonable, and requires, therefore, no restraint.

Local applications appear to have no influence upon the course of the disease, which, on the whole, is amenable to the general remedies.

In cases, however, in which we suspect a tuberculous as well as a syphilitic taint we must be careful, and treat the joint rather as a tuberculous one than as a syphilitic one.

Chronic osteo-arthritis of the knee.—This is one of the commonest affections of the joints, and one of the most troublesome. Its general pathology has already been described (page 1052), and we need only here allude to its clinical features. It is rarely seen before forty years of age, and seems to affect both sexes in about equal degrees.

The **symptoms** at first are usually ill-defined pain and stiffness, most marked in the morning and going off after exercise. There is no heat, redness, or swelling at first, but after a time the outline of the bones becomes irregular, and the joint looks and feels nodular. The movements, too, become uneven, and a creaking o grating may be felt in the joint with the hand placed over the front. After much exertion at this stage there may be some effusion, and the temperature will be increased.

The diagnosis usually offers little difficulty in view of the patient's age, and the above changes and the probable affection of other joints at the same time.

The **treatment** in the early stages consists in keeping the part warm and in proper exercise, with regulation of the diet. As regards the latter, everything which will promote healthy digestion and elimination must be attended to. Massage, too, will be found of great benefit if carried out regularly. Alkaline mineral waters are also indicated, and a course of warm baths, such as those of Wildbad, is frequently followed by much benefit.

In the later stages very little can be done to cure the disease, but the measures just recommended will retard its progress and diminish the suffering caused by it.

Ultimately great deformity is often caused by this disease, and a locking of the femur against the tibia, owing to the osteophytic outgrowths from both bones. Sometimes one or other of the latter become detached and loose in the joint, giving rise to all the symptoms, and requiring all the treatment, of loose bodies (page 1070).

But though the locking of the two bones may ultimately almost completely suspend its movements, true synostosis is exceedingly rare.

Anchylosis of the knee.—Any of the forms of limitation of movement, partial or complete, affecting joints as described under the heading of "Anchylosis" (page 1067) may affect the knee, and the general principles of their treatment were laid down there. But in the case of the knee it must be remembered, in the first place, that the primary function of the part is to support the body in progression; and in the second, that unless there is a good prospect of obtaining voluntary movement in both directions of flexion and extension it is better to abstain from all attempts to restore motion. For there is a strong tendency in this joint to assume a position of flexion whenever in pain; and if our passive or its own active movements produce pain, it will be kept flexed as far as possible. In this way a limb which has been straight and stiff after inflammation, but useful for progression, may be rendered useless by injudicious attempts to restore its movements; for as soon as the patient can move it he will place it and maintain it involuntarily in a state of flexion. Unless, then, the condition be completely relieved and free movement without suffering be restored, a perverted position is the result in many cases, and when strongly flexed the joint is not as useful as before, and even tends to grow worse, there being always a tendency to flex it involuntarily, and none to extend it again. This may be counteracted to some extent by splints, but, on the other hand, the latter prevent the very thing we are aiming at—namely, free movement—and favour the production of stiffness again. Occasionally anchylosis with flexion is combined with great rotation of the tibia on the femur (Fig. 456), and in some very rare cases of disorganisation of the knee the joint becomes anchylosed in the position of hyper-extension as seen in Fig. 457.

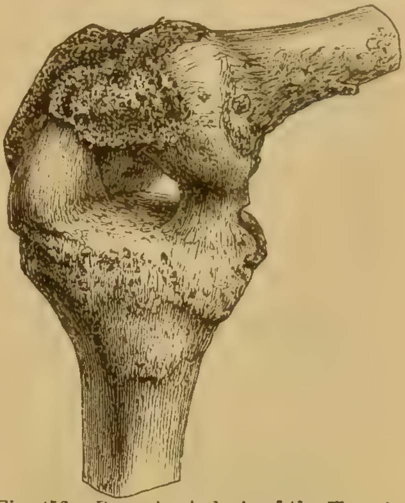

Fig. 456.—Bony Anchylosis of the Knee in the Position of extreme Flexion and Rotation of the Tibia outwards. (From University College Museum.)

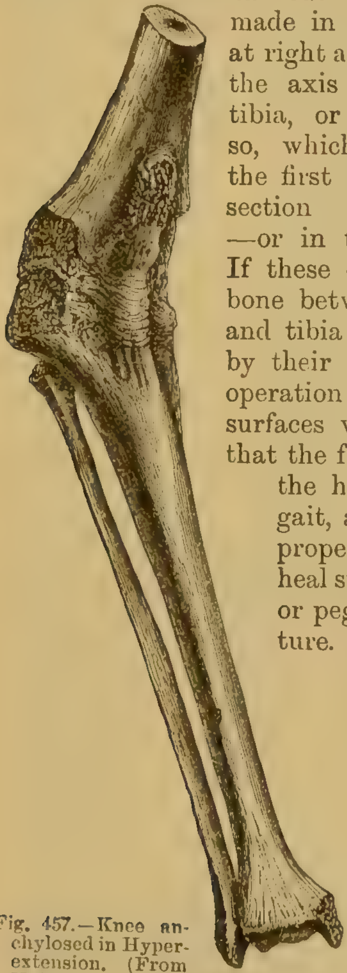

Fig. 457.—Knee anchylosed in Hyper-extension. (From University College Museum.)

Treatment.—For bad fibrous or bony anchylosis with great displacement, whether of flexion, extension, or rotation, the only thing to be done is either osteotomy of the femur above the joint, or the more or less complete resection of the joint. This is best done by the anterior flap method, by which the articulation is opened, and then the bond of union between the bones is divided by the saw in a plane at right angles to the axis of the femur. A second saw-cut is then made in a plane at right angles to the axis of the tibia, or nearly so, which joins the first line of section behind —or in the case of hyper-extension—in front. If these cuts be made true, and the fragment of bone between the two be now removed, the femur and tibia must come together in a straight line by their sawn surfaces. My aim in doing this operation has always been to adapt these fresh-cut surfaces with the knee in very slight flexion, so that the front of the foot reaches the ground before the heel, thus giving more springiness to the gait, and greater length to the limb. With a properly-applied plaster-of-Paris splint such cases heal straight off, and there is no need to suture or peg the bones. They heal like a simple fracture. But whatever is done, great care should be taken not to put undue strain upon the structures behind the joint which have been long contracted. If this be done, there is danger of gangrene of the foot from stretching of the arteries and nerves. To guard against this, enough bone must be removed to enable the limb to be brought nearly to a straight line, without any drag upon the popliteal structures, and the result must be carefully watched, the circulation and sensation in the toes being our guide. As long as these are natural we may be satisfied; but if impaired, the limb must be placed in a flexed position again at once, and a second operation removing more bone be done. Of course, in such a case firm, bony anchylosis is the aim we have in view.

But even in cases where the bone is freely removed, and there is no strain put upon the arteries from the straightening of the limb, the old cicatricial tissue dating from the original disease may press upon the vessels in the new position, and prevent the circulation. Such a case has recently occurred in my own practice, and resulted in gangrene of the toes. The limb was ultimately amputated above the knee, and the vessels were found on dissection to be normal, but had probably been pressed upon by the old cicatricial tissue when the limb was put in a straight line.

Hæmophilia affecting the knee.—This joint is probably more frequently the seat of hæmorrhage among "bleeders" than any other. It is so slightly covered by soft parts, and so frequently affected by blows and strains, that this is not to be wondered at. The symptoms and treatment have already been considered (page 376).

Neuralgia of the knee is frequently met with after inflammatory affections of the knee. The symptoms and general treatment have been already alluded to (page 1074).

Neoplasms about the knee belong almost exclusively to the chondroma and sarcoma groups. As a rule, once a diagnosis of such an affection has been made nothing is left but amputation well above the growth through the thigh. If there be any doubt as to diagnosis, the tumour may be explored easily by an aseptic incision, and in some few cases perhaps it is safe to remove chondromata which do not too widely involve the joint surfaces.

Cystic swellings about the knee.—These are not infrequently met with in the course of Charcot's disease, and of tuberculous synovitis in adults. In either case they only represent expansion of the true capsule of the joint distended with the fluid produced by the original disease, or they may be dilatations of the various bursæ round the joint, with or without a communication with the latter. When due to Charcot's disease, but little can be done for them; but when the result of either tuberculous or other chronic irritation, I have frequently dissected them out in whole or in part with the best results.

Loose bodies are commoner in the knee than in any other joint. Their pathology, symptoms, and treatment have already been discussed (page 1070).

AFFECTIONS OF THE ANKLE JOINT.

Simple acute synovitis of the ankle joint.—This is, on the whole, a comparatively rare condition. And when one considers the exposed position of the ankle, and the consequent blows, twists, and strains to which it is subjected, this is a matter of some wonder.

When it is met with, the usual signs and symptoms of synovitis are observed —*i.e.* pain and limitation of movement, swelling, fluctuation, and occasionally some redness. The swelling and fluctuation are usually most noticeable in front at either side of the extensor tendons, but also to some extent behind at either side of the tendo Achillis.

The diagnosis is based upon the history of injury or exposure and the signs just alluded to, and usually gives rise to no difficulty.

The **treatment** is that of inflammation generally, and consists in absolute rest for the part in the elevated position, and the application of leeches at the outset, followed by ice-bags or evaporating lotions, and later by hot fomentations with belladonna. At the same time, the bowels are to be kept open, and vascular tension is to be relieved by saline purges, with a small quantity of tartarised antimony pushed *ad nauseam.* When, by these means, the general vascular tension and the effusion have been checked, blisters may be employed to get rid of the fluid. This is usually easily done in simple cases, and then nothing remains but to support the joint for a time in either a plaster-of-Paris case or such a splint as that figured in Fig. 458. As the pain and stiffness subside, the patient may be encouraged to use the joint for walking, first with the splint still on, and later without it. Care should always be taken not to allow the joint to remain too long without movement, lest the stiffness become permanent.

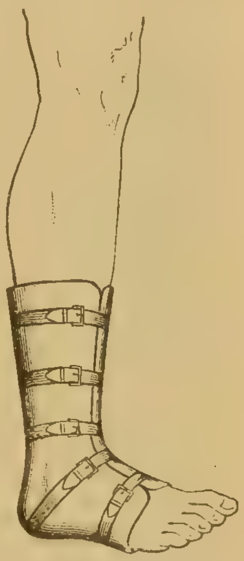
Fig. 458.—Leather Splint for the Ankle.

The **subacute form** of this affection is, perhaps, less uncommon than the acute, and is recognised by the same signs and symptoms present in a less marked degree.

The *treatment* of this will also be identical generally as in the last instance, but here we may resort to blistering earlier, and follow this up by rubefacients such as tr. iodi. If by these means the swelling and effusion be relieved, passive movement and massage at the same time must be employed to overcome stiffness. Then active movement with caution, lest the inflammation be set up again.

It is well in this, as in all other forms of synovitis in the joints, after the process has subsided to support the latter by means of strapping or elastic webbing for a time, to prevent passive effusion. (*See* page 1038.)

The **chronic form** of simple synovitis of the ankle not infrequently springs from one or other of the two previous affections. Here the tendency is to great thickening of the synovial membrane, with less effusion than in the acute or subacute forms, as a rule. But the swelling manifests itself in precisely the same position as the effusion (page 1115). The great difficulty with such cases is to diagnose them from the tuberculous form of the disease, and, indeed, among young patients this is often impossible. The same parts of the joint are affected, and the same general symptoms are present.

Fortunately, the *treatment* for both these forms of disease is the same. They are each varieties of chronic inflammation of the synovial membrane, but with different exciting causes. This treatment will consist in rest of the part and depletion in the earlier stages. Then blisters and rubefacients follow, combined, of course, with tonics, good food, and fresh air. Later, if the synovial membrane remain swollen and pulpy, and the joint tender, it will be just as proper to remove it in the case of the simple synovial disease as in the tubercular. The beneficial results will be the same, and the methods employed also (page 1117).

Syphilitic disease of the ankle joint, whether commencing in the bones or synovial membrane, is very common. It needs no special description either of its pathology, symptoms, or treatment, enough having been already said of the condition as seen in other joints generally (page 1044).

Tuberculous disease of the ankle appears under two forms, both being, as a rule, chronic. We find it commencing primarily, either in the bones or the synovial membrane. In either case the affection is a grave one, for it rarely remains long limited to either structure, but has a great tendency to spread to all the tissues of the joint. It is mostly met with in children and young adults, and is particularly destructive to the joint in the former. In the latter, however, it is probably a graver disease, for when tuberculous infection begins to spread in a young adult who ought to be in a state of mature vitality, it either means that the dose of the poison is particularly virulent, or that the soil has become unusually receptive, owing to advanced debility. In either case the chances of an arrest of the disease are small, while in the child, who with every year is acquiring increased power of resistance against infective disease, the prospect is better.

The affection in the ankle joint is also particularly grave, from the ease with which it can spread to all the aspects of the astragalus, until the latter bone is literally surrounded by tuberculous inflammation, and is little more than a sequestrum in the midst of a tuberculous abscess. And, again, the latter is prone to invade the other neighbouring articulations of the foot.

The **symptoms** of tuberculous disease of the synovial membrane of the ankle joint in its earlier stages are those, as a rule, of subacute synovitis (page 1115). Later, the usual pulpy swelling will be found in the spots indicated above, together with much stiffness and weakness of the foot, and some pain, though not much. The patient's general history will help much in a diagnosis. If the infection has spread to the bones or has commenced there, the latter will be expanded, and the pain on treading with the foot will be greater.

As a rule, the bones and synovial membrane are early involved in the same disease.

The **treatment** will consist, in the first place, of rest for the joint. This may often be provided for without condemning the patient to inactivity, by either employing a Thomas's knee splint or by supporting the knee in the flexed position in a bucket and wooden pin, upon which the patient can walk without using the ankle. This joint also lends itself very well to the treatment by passive hyperæmia (Bier, page 1050), the constricting bandages being easily controlled on the part. It may also be treated by the injection of iodoform and glycerine, either alone or combined with the last procedure.

These measures are often sufficient to arrest the disease in its earliest stages if carried out effectually and for long enough. But where, in spite of all constitutional and local treatment, the process is advancing, it becomes a question whether the foot ought to be amputated, or an endeavour be made to save it by some form of excision. Of course, where the latter is thought feasible, it ought to be tried, but, it must be confessed, that until quite recently excision, partial or complete, of the ankle joint has not proved a very satisfactory procedure. The chief reason for this is to be found in the difficulty of eradicating the whole of the diseased tissue from the joint. Here the astragalus has a disproportionately large articular surface, which extends over its lateral aspects nearly down to its articulation with the os calcis on the one hand, and to the scaphoid on the other. The synovial pouches extend even beyond the cartilaginous borders. Any disease, then, starting either under the encrusting cartilage or in the synovial membrane is very apt to advance to the other articulations, and from them to the adjacent bones. Under these conditions, mere removal of the articular disease of the ankle joint would be inadequate, the residue in the neighbouring articulations being capable of perpetuating the process indefinitely.

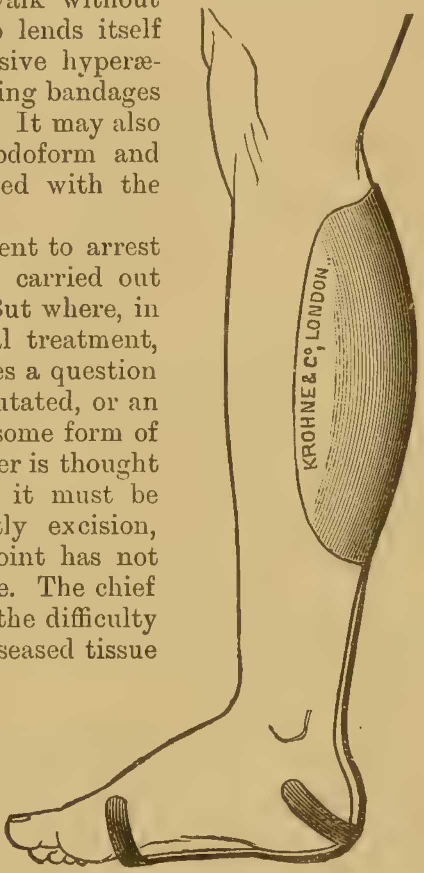

Fig. 459.—Splint for fixing the Ankle with flexible Rod connecting Leg and Foot Pieces.

Various formal excisions have, however, been formulated for the ankle joint. But in my opinion, here everything that can be done by a formal excision can be achieved by the free opening of the joint by longitudinal incisions at either side and behind, and the removal through these of all evidently diseased tissue by knife, gouge, and scissors, with free flushing-out of the cavity with warm water as described in reference to the hip and knee (page 1091). These incisions can then be closed without drainage, and the foot put up at a right angle in plaster-of-Paris or such a splint as that figured in Fig. 459.

In children, where the astragalus, as a whole, is very prone to be affected by tuberculous osteo-myelitis in addition to the disease actually present in the ankle joint, I have frequently found it necessary and exceedingly advantageous to remove the whole bone. This is best done through an incision running along the outer border of the common extensor tendons. Through this the astragalus can be extracted in whole or in pieces. The cavity can then be cut and scraped clean, and the ends of the tibia and fibula be gouged away until sound bone is reached. The surfaces of the os calcis and scaphoid must be similarly treated until all diseased tissue is eradicated. Then the usual flushing is carried out until the whole field of operation is cleared of tuberculous débris, and a sponge is thrust into the cavity. The stitches for the closure of the wound are then inserted, but before they are knotted the sponge is removed and the cavity is filled with iodoform emulsion. Any excess of this is finally squeezed out and the stitches are tied, a voluminous salicylic wool dressing being applied over all. In some cases it may be necessary in order to reach all aspects of the disease to make more than one incision into the cavity, and these must be placed with due regard to vessels and nerves.

The results after such informal excisions and removal of the astragalus are excellent in many cases, and fully as good as any claimed for the more classical excisions. Indeed, a foot which has completely lost its astragalus, appears to be but very little the worse for its removal.

Amputation for tuberculous ankle.—The question of the removal of the foot for tuberculous disease of the ankle is usually determined by the history of the patient's general health in the first place, and by the local condition in the second. Many cases of very advanced disorganisation are curable by careful general and local treatment in the direction of removing the tuberculous material by more or less perfect excision, provided the general vitality of the patient is fairly good. But in view of the possibility of general infection of the system from the local foci in the ankle, if the patient become debilitated by pain, want of outdoor exercise, and prolonged suppuration, the surgeon should be on his guard against pushing the attempt at preserving the foot too far, and thereby risking the patient's general well-being from the spread of the disease to internal organs. No hard-and-fast rule can be laid down to meet these cases, but it may be said generally that in young children conservatism may be persisted in longer than in young adults, and that among the latter, where the disease is stubborn and extending in spite of careful treatment, and where the general health is distinctly running down and loss of time is of great importance, amputation above the ankle, or by Syme's method if possible, should not be too long postponed.

Neuralgia of the ankle.—This is very prone to follow the ordinary inflammatory diseases of the part, and is often a source of much trouble to the patient. Indeed, the ankle like the wrist requires frequently a long time to recover from the painful effects of disease. The diagnosis will be based upon those general considerations already alluded to (page 1074), and upon the absence of any objective evidence of mischief.

The **treatment** is that for neuralgia, as it occurs anywhere in the body; the local will consist in blistering, repeated in different spots along the painful area. In addition, passive movement and massage are necessary in many cases to lead up to active movement. This must be encouraged as much as possible, both on account of the local condition and of the general health. Patients are only too prone to spare the painful joint, and in doing so injure their general health by want of exercise, while at the same time the nutrition of the parts round the ankle remains defective, and the morbid change inducing the neuralgia is not shaken off. These patients, therefore, should be encouraged to walk and run about in the fresh air, to an extent sufficient to secure abundant bodily exercise without over-fatigue. Then when in the house the ankle should be rested on a chair. Good rubbing before going to bed at night, and the wearing of thick woollen stockings in bed if the foot is inclined to be cold, will also materially assist the cure.

Neoplasms about the ankle.—These cannot be said to be common, but are occasionally met with starting from the bones of the leg or tarsus, or under the periosteum. They are usually either of the chondroma type, or belong to the varieties of sarcoma. They are likely to be mistaken in their early stages for tuberculous swellings connected with the joint or tendon sheaths. An exploration will very easily settle the question of their existence or not. When present nothing can be done but amputation well above the growth.

Cysts of the ankle.—These are occasionally met with in the form of distended synovial bursæ or tendon sheaths, or offshoots of either. Remembering that the pathological changes of synovial bursæ and tendon coverings are the same as those of the joints practically, we need not describe them more minutely here, as they have been already dealt with. (*See* page 1069.)

The *treatment* is also, on the whole, the same, except that these synovial cysts can be very easily dissected out from the parts round the ankle, and this should be done fairly early, before the effects of their pressure have gone too far.

Loose bodies in the ankle.—These are very uncommon, and are rarely the cause of inconvenience. Their pathology and treatment is the same as that suitable when they appear in the knee (page 1070).

Osteo-arthritis of the ankle.—This is not as common an affection as that of the hip and knee, but may be met with from time to time. There will, as a rule, be but slight difficulty in the diagnosis, inasmuch as the affection in the ankle is usually seen after other joints, such as the hip and knee, have been extensively affected, and the symptoms are the same as in the larger joints.

The *treatment* is also the same.

AFFECTIONS OF THE TARSAL JOINTS.

These joints are subject to the same diseases as the larger articulations, but no formal description is required for their recognition. The various forms of synovitis, from the simple to the tuberculous, are met with in every degree, and their treatment is conducted upon precisely the same principles that guide us in dealing with the larger articulations. Beyond all question the most important of all are the tuberculous affections. For these rest is the first consideration, and is best secured by disuse of the foot altogether as an agent of progression. To this end the patient should be furnished with a bucket and pin leg, upon which he can walk instead of the foot. But if this rest with or without Bier's treatment (which is peculiarly suitable to the tarsal affections) and the iodoform injections have failed, the tuberculous foci must be removed by operation. No hard-and-fast rules can be laid down for this removal, except that the contiguous bones are almost always involved, and require resection, and that this must be thorough, and must be undertaken through adequate incisions. These may be placed over the bones affected, and the latter can be often shelled out of their periosteum cleanly with great ease and advantage, more or less reproduction of bone taking place. The after-treatment is the same as in other cases.

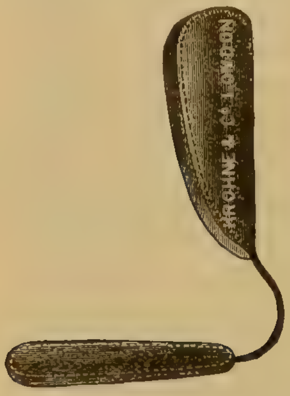

Fig. 460.—Splint for fixing the Ankle Joint with Copper connecting Rod between the two portions admitting of change of angle.

In all such cases I prefer the bucket splint and wooden pin or plaster-of-Paris splints to every other, and these latter should be made thick enough (with or without windows for drainage) to bear the weight of the body when the patient begins to walk about. When windows are necessary to admit of access to wounds or sinuses, the plaster should be varnished over to prevent absorption of discharges. The best varnish for this purpose perhaps is Friar's balsam (Tr. benzoin co.), which may be painted freely over the edges of the window, and on the wound or sinus as well, as it is an excellent antiseptic. But, of course, other forms of fixing apparatus may be desirable in special cases, and such a pair of moulded leather splints as those figured in Fig. 458 will in many cases answer admirably. Metal splints (Fig. 460), consisting of a flat foot-piece of tin united to a grooved leg-piece by a stout copper rod, are also excellent. The rod in these splints is soft enough to be bent in any desired direction to suit the shape of the limb. These splints have also the great advantage that they can be sterilised by heat.

AFFECTIONS OF THE STERNO-CLAVICULAR JOINT.

The ordinary forms of simple synovitis of the articulations of the clavicle are very rare, but this joint is occasionally the seat of acute septic inflammation in the course of pyæmia or septicæmia. In such a case a swelling rapidly forms over the front of the articulation, and fluctuation and œdema soon follow, with pain and inability to raise the arm to the horizontal position. The general condition leaves no uncertainty as to the diagnosis and the mode of treatment. There can be no doubt that here the proper measure is early and free evacuation of the effusion with thorough drainage. If this is not done, there is great danger of rupture of the abscess on its posterior aspect, and extravasation of the pus into the root of the neck and mediastinum, where it will run riot, and cannot be drained easily.

Tuberculous disease is also seen in rare instances in this joint, and presents the usual characters of this affection, attacking both the synovial membrane and the bones on each side. In a case recently under my care it was necessary to open the joint freely, when a large sequestrum was removed from the end of the clavicle, and the cavity of the bone was gouged clean. The result was most satisfactory.

Chronic rheumatic arthritis is also frequently seen to affect the sterno-clavicular joint, and has the usual characteristics seen elsewhere. The joint becomes nodular and tender, and movements of the shoulder are felt in it painfully. The presence of the same disease elsewhere helps materially in a diagnosis. The treatment is the same as that described already (page 1055).

AFFECTIONS OF THE SHOULDER JOINT.

This joint is comparatively rarely the seat of the affections which ordinarily attack the other articulations. The reasons for this are probably to be found in the fact that it is well covered by muscles, and is thus sheltered from the vicissitudes of temperature and weather, and besides is protected to a great measure from the effects of injury by the great mobility of the scapula. Nevertheless, we do occasionally meet with simple synovitis in its three degrees—acute, subacute, and chronic—and the septic forms of the same disease. Besides these, tubercular conditions may be met with and rheumatoid arthritis.

Simple acute synovitis of the shoulder.—This, when seen, is usually the result either of exposure, injury, over-use, or strain. It generally manifests itself by a sense of stiffness and weakness in the part, followed by pain and swelling. The latter is best felt anteriorly over the bicipital groove and on the axillary aspect of the capsule.

Here in most cases distinct fluctuation can be felt. Movements of the joint, whether active or passive, are productive of pain, and are in the latter case strongly resisted by the muscles which fix the humerus to the scapula, and cause the two bones to move as a whole. Together with this there will be increased heat in the part.

These symptoms will be sufficient for a diagnosis in most cases, the rapid onset and general acuteness serving to distinguish the condition from tuberculous invasion. The absence of general constitutional disturbance distinguishes it from the septic form of synovitis.

The **treatment** consists in fixing the arm so as to prevent movement in the joint; and here nothing is better than strapping applied as for a fractured clavicle by Sayre's method (page 804). But the same immobilisation may be brought about by putting the arm in a sling and securing it to the side by bandages encircling the body.

In addition to this, hot fomentations must be applied, combined with belladonna and glycerine. When there is much distension of the capsule, the joint should be aspirated. Leeches may also reduce the tension, and should be placed over the front and inner aspect of the capsule. When the inflammation has been by these measures allayed, blisters and rubefacients are desirable, and, later, massage and douching with hot salt water, together with regulated passive movement. In certain cases where much stiffness remains after an attack of simple acute synovitis it may be well to put the patient under an anæsthetic, and forcibly move the joint in all directions, the scapula being fixed by an assistant standing on the opposite side and clasping his hands over the clavicle. This measure, however, should only be adopted with great caution, and when all traces of the original inflammation have disappeared. On the other hand, such stiffness should not be allowed to remain too long untreated; for muscular atrophy sets in early, and the pain associated with the stiffness is often completely relieved as soon as adhesions are broken down and the movements of the joint are restored.

Simple subacute and **chronic forms of synovitis of the shoulder** joint only differ from the acute in intensity and duration, and need no special description here. They call for the same treatment in the earlier stages as the same affections in other joints (page 1034). The same rules, too, as regards forcible passive movement apply here as in the case of the acute affection.

Acute, subacute, and **chronic septic synovitis of the shoulder** may be said to be uncommon. When met with, the same treatment must be employed as for other joints (page 1042). When incisions have to be made to evacuate the effused fluid, they should be placed anteriorly close to the bicipital groove; and, if necessary, a counter-opening must be made behind, but this will not always be necessary.

Tuberculous disease of the shoulder is less common, too, than that of other joints, but is met with sufficiently often to deserve close attention. It may start either in the synovial membrane or in the bone; and if in the latter, either under the encrusting cartilage of the head or in the growing line of the epiphysis of the great tuberosity, or more rarely in the glenoid cavity.

Symptoms.—The diagnosis between the synovial form and that commencing in the bones is not very difficult if the following points are attended to, and the affection be seen in time. Early effusion and limitation of movement indicate synovial disease. Swelling of the soft parts and thickening of the bones without much effusion and with a good deal of pain at night usually indicate bone disease. But great difficulty is often experienced in diagnosing the various forms of bone disease one from the other. But in these days of aseptic exploration of joints by incision this is not of so much importance as formerly. Still, if a distinction can be made between disease of the great tuberosity which is developing outside the capsule from disease of the head, or glenoid cavity, much is gained.

Tuberculous disease starting in the great tuberosity usually leads to much bony thickening on the outer aspect of the head of the humerus with considerable nocturnal pain, but without limitation of the movement of the joint or effusion. Very soon a soft doughy spot may be felt as the disease spreads and softens the bone, and later a definite caseous abscess on the outer side of the head of the humerus. This abscess will usually work its way forwards or backwards, rarely through the deltoid outwards. But it must not be forgotten that if neglected such disease will almost certainly involve the joint ultimately.

Tuberculous disease commencing in the head of the bone proper, either in the epiphysial line or under the cartilage, causes much general thickening of the bone, and very soon extends to the surfaces and to the synovial membrane.

The diagnosis of tuberculous disease of the shoulder from the other affections is not very difficult, in view of the patient's history and the gradual development of the condition.

The general treatment is the same as that for other joints (page 1049). The **local treatment,** on the other hand, differs in so far as it has perhaps a better chance of success here than in almost any other articulation. The reason for this is probably the ease with which perfect immobilisation can be carried out in the shoulder without interfering with outdoor exercise. In the very earliest stages of the disease, either of the synovial membrane or bone, the arm can be carried in a sling, secured to the side; and unlike the hip, knee, and ankle, the joint has nothing but its own weight to contend against, and hangs in the position of greatest ease naturally. There is no need, therefore, for elaborate appliances to keep it in position, and all the aspects of the joint are left free for the applications of remedies. These consist in blisters and discutients in the early stages, and when these have done their work in reducing the hyperæmia, friction and passive movement can be employed. Whether it is that these can be so easily and efficiently applied in the earliest stages of the disease, that they are followed by speedy relief, or that there are factors in the case of the shoulder differing from other joints, which render the tubercular process in the shoulder more amenable, may be left an open question; but the fact remains that the disease much more rarely requires operative treatment than in the other joints mentioned.

Occasionally, however, the process goes on to destruction of the surfaces and the formation of caseous abscesses, and it becomes necessary to evacuate the latter and remove diseased tissues. This is best done through a long anterior incision over the bicipital groove. When the tendon in the latter is reached it is lifted out of the groove to the outer side, and all damaged tissue can then be excised by gouge and chisel. This is better than the complete removal of the end of the bone or resection of the glenoid hollow by any of the formal resections formerly practised. Indeed, formal excision of the shoulder joint for tuberculous disease may be said to be a rare operation nowadays. Still, it may have to be done occasionally, in neglected cases in which the process has been allowed to run on to destruction of the head of the humerus and of the glenoid cavity. But such an operation should be avoided whenever possible.

Where the disease has started in the great tuberosity it should be early dealt with by free incision over the softened or tender spot, and by thorough gouging out of the morbid material. If left to run on, the risks of infection of the joint are considerable.

Syphilitic disease of the shoulder joint.—This is a rare affection, and only calls for passing notice. When seen it is usually in the course of a syphilitic osteitis or periostitis of the head of the humerus.

The *treatment* is that of tertiary syphilis of other parts.

Osteo-arthritis of the shoulder.—This is a very common and a very troublesome affection. The general pathology, symptoms and treatment have been already discussed (page 1052), and require no further comment here. It only need be remarked that the disease is especially painful when seated in the shoulder, and very soon leads to stiffness and atrophy of all the muscles around. Everything should be done by massage and passive motion to prevent the latter, and the patient may be assured that the more the arm is used the less pain and stiffness there will be. The whole shoulder should also be protected from changes of temperature by warm clothing and special woollen coverings.

Synovial cysts of the shoulder.—Apart from enlargement of the bursa under the deltoid muscle these affections are rare. No special description of them is necessary after what has been already said upon the subject (page 1069).

Loose bodies in the shoulder.—These are far from common, and have the same pathology as those met with elsewhere.

The *treatment* will be removal through an anterior incision along the bicipital groove.

Anchylosis of the shoulder joint.—This condition following upon any form of destructive disease is a very serious one, whether only fibrous or actually bony. (*See* page 1067.) For, although the free play of the scapula on the ribs compensates to some extent for the loss of movement in the joint, the latter, if completely spoiled, leads to a very crippling condition. When we remember that the shoulder joint is designed for freer movement in all directions than any other articulation in the body, this is easily understood. In all cases the joint is fixed in the position of greatest rest, *i.e.* with the arm hanging straight by the side. In this position, if rigid, the limb is very helpless.

Where this limitation of movement is due simply to slight adhesions of the surfaces and thickenings in the capsular structures, the condition may often be much improved by massage, and, if this fails, by forcible passive movement under an anæsthetic. There is always, however, great difficulty in carrying out the latter, owing to the difficulty of fixing the scapula; and if the adhesions are firm, sufficient force cannot be applied to tear them. And even if they were torn, very little good would result; for the raw surfaces would be almost sure to re-unite, the painful condition of the shoulder preventing the patient from active movement to any adequate extent.

A sufferer after such treatment is apt to suppose that he is exerting the joint, when he is really only moving the scapula with the humerus, and consequently the advantages of the forcible manipulation under an anæsthetic are soon lost. In such cases where the patient is very seriously crippled by the fixation of the joint, there is nothing left but a formal excision of the head of the humerus. This operation, if carefully carried out, whether for strong fibrous anchylosis, or for synostosis, is very successful. The new bond of union, which develops between the humerus and the scapula, admits of free motion in all directions, and is nevertheless strong and reliable. The operation itself calls for no detailed description in this place.

AFFECTIONS OF THE ELBOW.

This joint is very subject to all the varieties of disease we have been considering in connection with the other articulations. All forms of synovitis are met with here, from the simple to the tuberculous, and the affections of the bones are no less frequent.

Simple synovitis of the elbow.—This, the result of injury, strain, or exposure, manifests itself in the first place by a sense of stiffness in the joint, soon followed by pain on movement. Swelling soon supervenes upon this, being most marked at either side of the olecranon behind where fluctuation will be felt if there is much effusion. The joint, under these conditions, will be held in the attitude of greatest ease—namely, semi-flexed and semi-pronated—and any attempt to alter its position will cause considerable pain. The changes in the synovial membrane in this affection have been already considered (*see* page 1033), and it remains to deal with the treatment here.

The **treatment** is the same for all acute joint effusions. (*See* page 1034.) In all cases absolute rest for the part above all. This is best carried out on an angular splint placed on the inner aspect of the limb, and adapted to its position of semi-flexion and semi-pronation. Having secured the joint upon this, leeches may be applied in sthenic cases, and when they have done their work and have withdrawn a considerable quantity of blood, hot fomentations should follow. By these means the vascular tension will usually be soon relieved, and the absorption of the effusion within the joint will follow. If there is much delay in the latter, an aspirator may be cautiously used through one of the distended pouches behind, and the fluid may be drawn off. After this, if the joint refill, it may be again aspirated, or, by a succession of blisters applied over the lateral aspects, the fluid may be made to disappear. Later, massage and gentle passive movement may be begun, until all stiffness has passed off, and active movements will follow as soon as the pain subsides.

In the *suppurative forms* of the disease, whether traumatic or pyæmic, the general symptoms will be the same as in the last affection, with the addition of deep redness and œdema round the joint, and high fever. Here no time should be lost in relieving the cavity by free incisions. These should be placed at either side of the olecranon behind, and ought to lay the synovial sac freely open, so that every part of the joint can be washed out thoroughly with one of the antiseptic solutions. Drainage should be provided for in these cases until the inflammatory process has quieted down, and then the drains must be at once removed, and the incisions encouraged to close. During all this time the joint must be kept upon a suitable splint at right angles, or at a somewhat acute angle, so that if anchylosis be the sequel the limb may be in the position of greatest usefulness. But the position should be altered occasionally to the straight line, and then returned to the right angle, so that adhesions forming may be stretched. The older practice of subjecting the joint to constant passive movement during the process of repair is now almost entirely abandoned, as tending to keep up the inflammation and increase the plastic exudation. Only when the separative process is nearly completed should movement be begun. In other words, as soon as the patient begins to feel that the joint can be gently flexed and extended without pain. Massage and passive movement, as soon as all inflammatory action has subsided, will be necessary for long periods, to restore these movements of the joint.

The **simple subacute** and **chronic forms of synovitis** of the elbow have the same general pathology as in the case of other joints (*see* page 1036), and need no special description.

The *treatment* of the subacute form consists in rest in the position of greatest ease, with blisters and rubefacients. In those cases where much effusion remains, aspiration is proper, followed by strapping or elastic bandaging and massage.

In the chronic form the treatment is practically the same, but where a great deal of thickening of the synovial membrane remains with loss of power, the joint should be opened and washed out, and, at the same time, any redundant folds of synovial membrane should be excised. After this the incisions should be carefully closed, and usually without any drainage. In these cases much good often follows alternate hot and cold douches, followed by regular massage.

Syphilitic disease of the elbow has no special features which need occupy us here. Enough has been said at page 1044 to enable the student to recognise and treat it.

Tuberculous synovitis of the elbow in its general characters differs in no way from the same affection in other joints. (*See* page 1046.) It is a common affection, and a particularly serious one, the movements of the joints rarely escaping, unless the treatment be early and thorough.

The **symptoms** are those of chronic synovitis, with pulpy swelling, most marked at either side of the olecranon behind the joint, but the latter, as a rule, is also generally swollen. The family and personal history of the patient, and chronicity of the affection, together with the absence of any other known cause for the persistence of the condition, are our chief guides in diagnosis. In these cases there is often but slight pain in ordinary movement, but there is much stiffness from the beginning, and the limb is kept semi-flexed and semi-pronated.

The **treatment** of this condition is one of the most difficult problems in surgery. Where the affection is seen early in patients of fairly good physique, it may be arrested by the ordinary constitutional and local remedies for tuberculous synovitis combined (*see* page 1049), but where it has been neglected or appears from the first in delicate individuals, it is most intractable. One common experience, however, should guide us, at all events in very young children—namely, that in them the disease may be very severe and run on to extensive abscess and sinuses, and yet in the end quiet down and leave the joint, to all intents and purposes, as good as before. In adults and half-grown young persons the case is quite different, and where abscesses once form the result is usually a stiff joint. With these facts before us, we should exhaust every kind of palliative treatment in young children, including Bier's method (page 1050), before having recourse to excision, while in the older patients excision offers a better prospect than waiting, in many if not in most cases.

The aim of operation in such cases should be to excise as much of the synovial membrane as can be reached without destroying the functions of the joint. To do this the latter may be opened from behind in many ways, and a careful dissection be made in all directions. Perhaps the best means of carrying this out is to make an oval or H-shaped incision over the back of the joint, and then divide the olecranon and turn it up within the upper flap. If the joint be now fully flexed, the synovial pouches are thoroughly exposed, and can be dissected away completely. The olecranon is then wired in its old position, and the wounds are closed. Any secondary foci found in the bone must at the same time be removed with gouge or chisel. When such an operation has been done with perfect asepsis, the result is frequently complete arrest of the disease, and a limb but little impaired in usefulness. But, in very young children, it is better, perhaps, to deal with the foci of softening as they occur, by opening and scraping them out, the limb being kept at a right angle the whole time.

This is a joint which lends itself *par excellence* to Bier's treatment together with the injection of iodoform in suspension (page 1050).

When the bones are the seat of the primary tuberculous disease in children, or are deeply involved in disease secondary to the synovial affection, the same rule of waiting and dealing with the foci as they soften may be followed, but with adults the case is different. In these the joint must be opened as just described, and all tuberculous osseous tissue be removed.

In many cases it may be possible to do this without sacrificing the ends of the bones completely, by dividing the olecranon and using the gouge freely, but often it will be necessary to adopt the old classical excision in one or other of its forms.

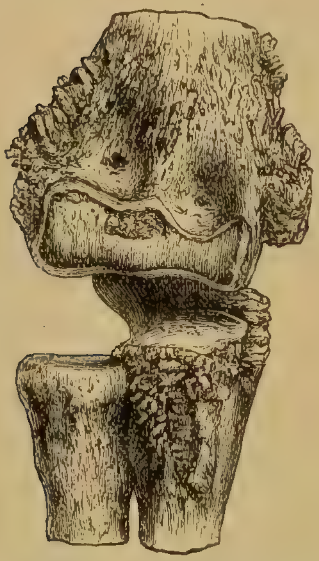

Fig. 461.—Tuberculous Disease of the Elbow, starting in the synovial membrane and extending to the cartilages.

It is very hard to lay down rules for guidance in these cases, but this much may be said, that surgery is becoming every day more conservative in dealing with them. It will be remembered that the osseous form of tuberculous disease in the elbow may start from either of the three bones primarily, either at the epiphysial lines or under the encrusting cartilage, or it may be secondary to disease commencing primarily in the synovial membrane as in Fig. 461. In Fig. 462 the disease is seen distinctly in three situations—(*a*) under the encrusting cartilage; (*b*) in the epiphysis of the olecranon; (*c*) in the synovial pouches.

Position after excision.—When partial or complete excision of the elbow has been performed, the question arises: What position is best for the joint during the process of healing? Formerly, that of flexion to a right angle and semi-pronation was always recommended in view of possible, or, indeed, probable anchylosis. But now, with aseptic wounds, we need not fear the latter so much, and may put up the limb at first quite straight or semi-flexed, with slight pronation; in fact, in the position of greatest ease to the patient.

I am in favour of abandoning all splints in these cases on theoretical and practical grounds, and of trusting for support to abundance of padding of wool round the joint. This gives sufficient support to the joint, and what little movement is permitted by the elasticity of the dressing is rather beneficial than otherwise. As to the question of passive movement in such cases, I am in accord with those who hold that this need not be carried out in the way formerly thought necessary. Indeed, it is not required at all at first. But when the wounds have soundly healed the joint may be gently moved in various directions, so far as this movement gives no pain. But the patient should be encouraged to use the joint as soon as ever he can, which will be so soon as it becomes more or less free from pain. Massage will also be of great use at this period in preventing atrophy of the muscles, and in keeping up the nutrition of the parts during the processes of repair.

Osteo-arthritis of the elbow.—This is not an uncommon affection in any or all its forms, and much limitation of movement may be the result.

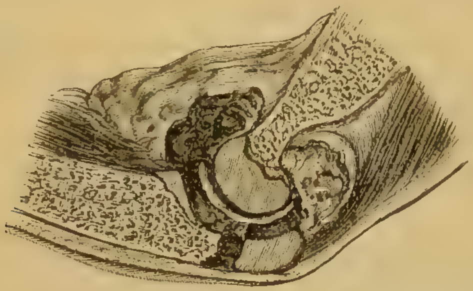

Fig. 462.—Extensive Tuberculous Disease of the Elbow Joint involving Epiphyses, encrusting cartilage and synovial membrane.

The diagnosis is easy, as a rule, in view of the fact that other joints are usually affected at the same time and to a larger extent, and that the joint, being so exposed, will easily show irregularities of shape and movement. The general and local treatment are the same as for other joints (page 1055).

Anchylosis of the elbow.—This condition not infrequently follows upon destructive disease of the joint, and may be a source of great discomfort to the patient. If the anchylosis have taken place at a right angle, the limb may be fairly useful for some purposes, but is much crippled for all ordinary work. If the union of the bones, on the other hand, have taken place in the straight position, though the limb may be useful for some handicrafts, the patient is unable to bring the hand to the head and cannot feed himself. In either of these cases the only course to pursue is to perform a classical excision and remove enough bone to ensure that the bond of union following the operation will be only fibrous. It is remarkable what excellent results follow this operation at the present day, a joint being formed almost equal in strength to the natural one.

In the case of very young children this formal excision should not be done. The operation must be postponed until the limb has developed almost or entirely to its full extent, and can then be undertaken with the best prospects of a useful limb.

Neuralgia of the elbow is not infrequently met with as a sequence of the inflammatory affections of the part. Here it manifests itself in shooting pains, not always depending on movement, and unassociated with any evidence of active disease of the joint. It is most usually met with in debilitated neurotic individuals subject to neuralgic pains of other parts.

The *treatment* consists in tonics and attention to the general health, with blisters or the application of the actual cautery to the painful spots. Later, rubefacients, such as tr. iodi, will be beneficial, and massage. The use of the joint should be encouraged.

Neoplasms about the elbow.—These are the usual new growths of bones and periosteum, and are mostly met with in young adults. Thus we meet with chondromata, osteomata, and the varieties of the sarcoma group affecting either the lower end of the humerus or the ends of the radius and ulna.

Sometimes it is possible to remove them without sacrificing the joint, but, as a rule, the only safe treatment is amputation well above the disease.

Cysts about the elbow are rare, but may be met with either as bursal enlargements, or as distensions of parts of the capsule of the joint.

Their diagnosis is not a matter of much difficulty when their history and slow formation are taken into account.

Their *treatment* consists in careful dissection of the cyst out of its bed and suture of the last portion, if it cannot be reached without sacrificing the joint.

In many cases this will be sufficient here as in the neighbourhood of other joints.

Loose bodies in the elbow joint.—These are not very frequently met with. When present they have the same general pathology, symptoms and treatment as described elsewhere (page 1070). Most frequently they spring from pendulous folds of synovial membrane, the result of chronic inflammation (Fig. 430).

AFFECTIONS OF THE WRIST JOINTS.

Simple synovitis.—In this, as in the other joints, the various forms of acute, subacute, and chronic synovitis, are met with; but for some reason, not easy to determine, are less common than in the larger joints. When met with they are usually the result of infection in the course of general septic intoxication of one kind or another, whether, for instance, it be in the course of a gonorrhœa or a typical pyæmia. As the result of injury and over-use, pure and simple, these forms of synovitis are comparatively rare, probably owing to the fact that the great mobility of the hand enables it to escape the effect of those forces in a way that in a more unyielding part would not be possible.

The **symptoms** of acute synovitis of the wrist, however produced, are similar to those observed in other joints. At first there is stiffness, then pain of a dull aching character follows, and before long swelling most marked on the dorsal aspect. If the affection be very acute, and especially if septic, there will be œdema of the subcutaneous tissue. Fluctuation of fluid in the joint can rarely be felt, the capsule being comparatively small and covered on both aspects by tendons. In uncomplicated cases the latter move freely and without pain in their sheaths, and the fingers can be flexed and extended without difficulty. But in many cases the inflammatory irritation has extended from the joint to the tendon sheaths, and all movements of the tendons within the latter are painful, as is also pressure over the joint.

The **treatment** of simple traumatic cases is easily carried out. It consists in absolute rest on a splint, such as that depicted in Fig. 463 and in the elevated position. General rest in bed will also be desirable at first, to quiet the circulation, and saline aperients will also help in relieving vascular tension.

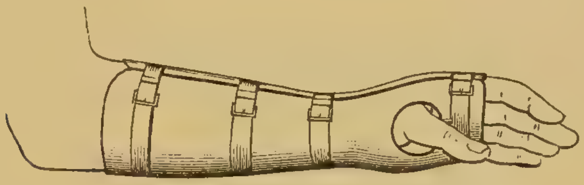

Fig. 463.—Leather Splint for the Wrist.

Locally, ice or evaporating lotions may be applied at the outset, and, later, hot fomentations with belladonna. If seen very early leeches ought to be applied in cases where there is much tension. Under these remedies, simple acute synovitis, as a rule, subsides rapidly in robust individuals, and nothing but stiffness and some swelling is left behind. These effects will often give much trouble, owing to the painful neuralgic conditions left, as the consequence of the bygone inflammation and the consequent disinclination of the patient to use the part. They are best met by rubefacients and massage, followed by regular passive motion. Sometimes it will be necessary to break down adhesions under an anæsthetic, but this must be resorted to with much caution. When all traces of active inflammation have passed off, the patient should be encouraged to use the hand freely in every way.

When this acute inflammation is due to sepsis, early incisions should be made into the joint behind at either side of the extensor tendons, followed by free flushing of the sac, either with sterilised hot water or some mild germicide. The cavity is also to be carefully drained, as long as effusion is abundant. Many such cases recover with wonderfully little permanent injury if this treatment be carefully carried out, but some are left with much stiffness.

The *subacute* and *chronic forms* of these affections differ only in degree, and require the same treatment in less vigorous shape.

Tuberculous disease of the wrist may be primary either in its synovial membrane or its bones, or may be secondary to tuberculous teno-synovitis starting in any of the tendon sheaths round the joint. In any case it is a very grave affection, and most intractable. When synovial at the outset, it as a rule soon attacks the bones, and spreads to the whole carpus in many cases.

The **symptoms** are stiffness, followed by uneasiness and a little pain. As a rule, the latter in the earlier stages of the affection is very moderate, and the tendons may move freely over the joint without suffering or limitation. Presently the swelling becomes very marked, and the swollen, doughy wrist presents a characteristic appearance. The softness and doughiness are most marked behind at either side of the extensor tendons. Then as the bones become more deeply engaged and lose their encrusting cartilage, the pain becomes very severe and continuous, aggravated by the slightest movement. The suffering often tells very much upon the patient's sleep and general health, and he becomes thin and weak.

Treatment.—The general treatment is that for tuberculosis in any part of the body. (*See* page 1049.)

The local treatment consists in perfect rest on a splint, such as that already figured. Blisters may be applied in the early stages or other counter-irritants, and Bier's method (page 1050), with or without the injection of iodoform in suspension, should be given a fair trial. Some of the results of the latter which I have seen have been quite surprising, especially in this joint.

But where these means fail, and the tuberculous material either in the bones or synovial membrane is undergoing caseation and softening, as indicated by thinning of the soft parts and fluctuation, nothing is left but a more or less formal excision of the diseased tissue. In such cases I am in favour of as limited operative interference as possible, incisions being made wherever there is evidence of softened tissue, and through these the removal of the morbid matter, with free gouging and flushing and immediate closure of the wound. These measures will, in my opinion, accomplish all that can be achieved by the most radical excision, and with less risk. But these cases are not very satisfactory, as a rule, and where recurrence is obvious, and any further interference would leave a very much impaired articulation, amputation is required in but too many instances.

The great hope for the future lies here in very early diagnosis and energetic treatment.

Osteo-arthritis of the wrist.—This is a common and most intractable affection. The rules already given for its treatment in other joints apply here, and need not be repeated.

AFFECTIONS OF THE AMPHI-ARTHRODIAL JOINTS.

The most important of these are the articulations of the bodies of the vertebræ, of the pubic bones and of the sacrum on each side, with the ossa innominata.

The first of these, considered in the Article on Diseases of the Spine, need not be further alluded to here.

Disease of the symphysis pubis.—Disease here is not at all common, and when met with is usually the result of septic inflammation in the course of pyæmia or of tuberculous deposit. In the first case the local disease will take quite a secondary place to the general. It is mostly characterised by rapid swelling and effusion over the joint, with redness and œdema. If left to itself the collection will burst, probably through the skin, but may make its way backwards into the pelvis.

The **treatment** is early evacuation and drainage. If the patient survive the general infection, it may be desirable subsequently to scrape out the carious surfaces of the joint; or if this is not adequate, to resect the diseased surfaces, wiring together the clean-cut ends.

In tuberculous disease of this joint the proper treatment will be removal by gouging and scraping of the softened tissue, and perfect rest for long periods on the back. When the patient is well enough to get up, a firm case of plaster-of-Paris closely fitting the whole pelvis, and running as low and as high as possible, will give much support.

In operating upon such disease the aim should be to remove it before it has extensively invaded the surrounding parts, or has burst through the skin. When this is done it may be possible to secure primary union of the wound without any sinus.

Disease of the sacro-iliac joint.—What has just been said of the symphysis pubis applies equally to this joint as regards its general pathology — namely, it may be the seat of secondary septic infection in the course of chronic or acute pyæmia, or it may become infected with tubercle. It is not necessary to speak in detail of the first condition. If abscesses form in and about the joint, they are evacuated and washed out early. If the patient survive the general infection, the condition in the joint will probably heal in time with careful drainage, if not subsequently infected with tubercle. In any case convalescence will be very slow, and the recumbent position must be insisted on for months, bearing in mind the great strain put upon both joints in the erect position, and the probability of this being injurious if the process of healing is incomplete.

Tuberculous disease of this joint is one of the most serious conditions possible, and until within the last few years was considered by all who gave special attention to the subject as invariably fatal in the long run. Of late, however, owing to improvements in the methods of dealing with tuberculous disease generally and locally, and the recognition of its true nature, we have had better results, and it may now be regarded as by no means so deadly as formerly. I have had lately several most encouraging cases where extensive disease of this joint with large abscesses was successfully combated, and brought to a standstill.

Symptoms.—The history of tuberculous sacro-iliac disease is at first, as a rule, most indefinite. It is usually seen among young adults from twenty to thirty, though also met with among children and those in the decline of life. The first thing complained of is, as a rule, fatigue and a sense of weakness in the lower part of the back. This is followed by aching pain, aggravated by exertion, and severe towards the close of the day. But probably before this stage is reached the sense of weakness will have prevented the patient from taking any exercise, and he will remain recumbent most of the day. This is not invariably the case, and I have seen a case terminating rapidly in death from abscess and exhaustion, in which the patient was able, for a while, to walk about and jar the pelvis by stamping with the foot, even while the abscesses were forming, without any marked distress or weakness. Everything depends upon the extent to which the synchondrosis is involved. If only one border is affected, and the rest of the joint is firm, the weight of the body may be well borne, as well as jars and strains. But if the whole surface be implicated, the patients will feel—as they express it—" broken in two," and the ilium may be so loosely attached to the sacrum that the patient may be conscious of the grating. Sometimes, too, lateral pressure on the crests of the ilium may produce pain in the joint, but this is obviously only likely to take place when considerable disease is present. If the disease be seated on the anterior aspect of the joint, pain along the course of the nerves, derived from the lumbar plexus, may be a notable symptom, and cases have been mistaken for ordinary sciatica which turned out to be sacro-iliac disease, associated with abscess, running along the course of the plexus. In such cases, too, the psoas muscle may be irritated and more or less contracted.

One of the early symptoms of this disease is certainly abscess. For very often the ligamentous supports of the joint remain firm enough to guard it against movement and production of any pain, until an abscess is formed and quite apparent. Such collections may present in various situations. Those derived from disease on the posterior aspect of the articulation will usually show best just over the joint; but they may travel outwards under the gluteus into the buttock, and present below the border of the muscle. Those on the front of the synchondrosis may pass along the small rotators of the thigh, through the sciatic notch, and come to the surface behind the great trochanter, or, on the other hand, if the patient preserve the recumbent position, they may run upwards and over the upper border of the joint to present posteriorly. Again, they may burst into the cæcum or rectum. In these cases, if the abscess be allowed to burst spontaneously, the result is almost invariably the same.

After a longer or shorter interval the cavity becomes septic, and the patient is worn out with hectic fever or cut short by one of the acute wound infections.

There appears to be a good deal of evidence that this disease is more likely to begin on the borders of the joint than towards its deeper central portions. And this is fortunate, as regards treatment.

The *treatment* at the present day is far more radical than formerly. We now know that large portions of the articulation, if diseased, may be removed with advantage by operation. In carrying out the latter the incisions should be free, and the aim should be to reach all parts of the diseased area. No definite rules can be laid down for these incisions, but in any case the aim should be to remove by chisel or flushing-gouge every trace of tuberculous material, whether it lie in the bones or soft parts, or in the resulting abscess. The disease should always be thus attacked before any breach of surface and formation of sinuses has taken place, and after removal of the morbid material the wound may be closed without drainage. If by this means union by first intention be secured, the whole process may be arrested without any open sinus. And, even if recurrence take place, a second or even third operation on the same lines may succeed where the first has failed, and the patient be left with a sound bone, and without external opening. At all events, if the latter cannot ultimately be avoided, and a sinus form, it will lead down to a much reduced focus of disease, and, perhaps, only after consolidation has taken place to a large extent, and all extensive abscess formations have ceased.

Working on these lines, I have lately treated a case of extensive sacro-iliac disease on both joints combined, with a huge abscess running from side to side in front of the sacrum. This was opened and flushed out, scraped, and at the same time the bone disease was gouged away. Primary union was obtained after the first and second operations; but after the third, sinuses formed, but led down to a much reduced focus of disease. Ultimately the patient was sent to the country able to walk, but came back with some recurrence. The abscess was again opened, and bone was gouged away, and the whole cavity scraped and flushed. There are still small sinuses, but the patient is daily gaining strength, and there appears every prospect of a favourable termination before long.

When allowed to get up, the pelvis should be supported by a plaster-of-Paris case carefully and closely applied, and carried up as high as the thorax, and as low as the trochanters. Of course, every kind of constitutional treatment suitable to tuberculous disease is carried out by means of tonics and dieting, and prolonged rest for months is enjoined.

Disease of the sacro-coccygeal joint.—This may follow on general pyæmic infection on injury, or tuberculous infection as a sequel to the latter. There is little or no difficulty in diagnosis in either case. The seat of the swelling, pain, and ultimate abscess will soon betray the nature of the affection.

No special rules need be laid down for treatment, but it should be remembered that in many cases the condition is best met by the careful excision of the whole coccyx, which will remove all diseased tissue, and leave the patient with less pain and general suffering, either on sitting down or defæcation, than even if the disease were brought to a standstill by less radical measures. For in those cases in which the active disease has been arrested by the ordinary modes of treatment, a very painful and harassing state of things remains, which has often ultimately to be met by excision.

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